DEPARTMENT OF MEDICINE

MUHIMBILI MEDICAL CENTRE

Incorporating

THE FACULTY OF MEDICINE

THE UNIVERSITY OF DAR ES SALAAM.

ACUTE BRAIN SYNDROME IN THE MEDICAL WARDS OF MUHIMBILI MEDICAL CENTRE DAR ES SALAAM.

A Dissertation for the Degree of Master of Medicine (M.Med)

By:

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DECLARATION

I, IDDI IBRAHIM MWANGA, declare that this dissertation has not been presented elsewhere in any University for the award of Master of Medicine (M.Med) Degree.

ACKNOWLEDGEMENTS.

The preparation for the Master of Medicine (M.Med) degree (The University of Dar es Salaam) promotes not only clinical experience but also interest in research and scientific work.

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SUMMARY:

Acute psychiatric symptoms may be associated with a wide variety of physical illnesses (acute brain syndrome). These symptoms may arise as a result of direct involvement of the brain as in meningitis or cerebral malaria, or they may arise as a result of altered brain function associated with diseases primarily involving systems other than the central nervous system, such as lungs and kidneys.

It is important that doctors recognise those psychiatric symptoms which may indicate underlying physical disease since failure to do so may lead to the patient's death.

This study was undertaken to identify the causes, clinical presentation and prognosis of the acute brain syndrome in general medical practice in Dar es Salaam, since there have been no previous studies on this topic in Tanzania.

During a six week period from July 13 until August 31 1981, 21 patients were admitted to the medical wards of Muhimbili Medical Centre with evidence of acute brain syndrome, either before or at the time of admission. The mean age of the patients was 25.9 years (range 8 - 48 years). The male: female sex ratio was 1.3:1.

Malria was the disease most commonly associated with acute psychiatric symptoms in the present study, (14 (66.6%) patients). Five (23.8%) of the 14 patients had other diseases such as anaemia, uraemia, hypertension, hepatitis and epilepsy.

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Malria was the disease most commonly associated with acute psychiatric symptoms in the present study, (14 (66.6%) patients). Five (23.8%) of the 14 patients had other diseases such as anaemia, uraemia, hypertension, hepatitis and epilepsy.

In the remaining 7 patients the disease associated with the acute brain syndrome were: chronic renal failure (2 patients), meningitis (1 patient), meningoencephalitis (1 patient), subdural haematoma (1 patient), rickettsial disease (1 patient) and anaemia (1 patient).

Fourteen (66.7%) patients had clouded consciousness on admission and 7 (33.3%) patients were either semiconscious or unconscious by the time of their admission.

Of the 14 patients with clouded consciousness 10 (47.6%) had agitated delirium and 4 (19.1%) had quiet delirium. Of the 7 patients who were semiconscious or unconscious at the time of admission 4 (19%0 had agitated delirium and 3 (14.3%) had quiet delirium during the recovery phase.

Thought disturbances were observed in 14 (66.7%) patients and speech disturbances in 16 (76.2%) patients. All patients at sometime during the course of their illness showed evidence of disturbance in orientation, insight, memory and intelligence.

The mean duration of symptoms prior to admission was 3.8 days (range 1 - 14 days). The main factor leading to delay in reporting to hospital was the belief by relatives that the patients problems were related to supernatural causes. Delay was also related to the patient's age, the older the patient the longer the delay in referral.

Duration of stay in hospital was also longer in older patients and in those who had other diseases apart from those which were

responsible for admission. There was also a relationship between duration of stay in hospital and the severity of psychiatric symptoms.

Seventeen (81%) of the 21 patients recovered but 3 patients required later referral to the Psychiatric Unit.

Four (19%) patients died; one from cerebral malaria, two from uraemia and one as a result of subdural haematoma. The diagnosis of subdural haematoma was made at post-mortem. This patient's history highlights the need for all medical practitioners faced with a patient with acute psychiatric symptoms, to make every effort to exclude underlying physical illness.

INTRODUCTION.

The history of medicine carried the belief that there are two main categories of mental disorder, those due to medical or organic causes and those due to supernatural or moral causes. For many centuries medicine had very little responsibility for the mentally ill. However the development of medicine, particularly pathology led in Europe to the intensive study of the mentally ill from the point of view of the natural sciences.

The interrelationship between somatic medicine and psychiatry is well established in Africa. Giel, (1975) observed that it is quite dangerous to detach altogether the practice of psychiatry from that of somatic medicine. Failure to diagnose an organic cause of a mental disorder has adverse effects on both the patient's health and the doctor's reputation. This is more so in countries where the load of infectious diseases is still great. Lambo (1956) noted that where infectious diseases are endemic the role of infection in the aetiology of mental illness can be substantial. German (1972) stated that the psychiatrist researcher in Africa, besides considering the cultural and social factors in mental disorder, must constantly be prepared to assess the down-to-earth but massive contributions made to any mental illness by organic deprivation.

Aetiologically acute confusional states may result from acute physical illness, either primarily because of direct involvement of the brain as in meningitis or secondarily in association with diseases

such as pneumonia, metabolic disorders, toxic, vascular insufficiency or endocrine disturbances.

Reviewing the observations of several workers on the prevalence of a wide range of organic illnesses which can precipitate acute confusional state, in all parts of Africa, Jilek - Aall (1970a) list the following conditions in the order of their relative importance as aetiological agents - Meningoencephalitis, trypanosomiasis, cerebral malaria, syphilis, pulmonary infections, amoebic dysentery and bilharziasis, general malnutrition, especially protein deficiency, hormonal imbalance, B-avitaminosis and drug addiction, helminth infestation, climatic effects, heat stroke and rapid dehydration. Jilek himself added deliberate poisoning with local herbs which have active pharmacological substances. German (1972) put ethyl and methyl alcohol in the top list. He added typhoid, paratyphoid, tuberculosis and tropical ulcer in the list.

Organic illnesses which secondarily affect the brain to cause acute confusional state do so by exerting a powerful background conditioning effect which facilitates the appearance of subsequent acute mental state in response to minimal precipitations. With such preconditioning, added stresses of organic or psychological origin can acquire an unexpected aetiological power and produce a range of psychotic reactions.

At one time psychiatrists believed in a characteristic mental picture for every physical disease. But Bonhoeffer in 1910 demonstrated that the same signs of mental dysfunction may be associated with widely

differing illnesses. For example impaired consciousness may result from a number of toxic processes acting on the brain.

In Tanzania no work has yet been published about acute brain disease. The purpose of this study is to identify the causes of acute brain disease in the medical wards or Muhimbili Medical Centre Dar es Salaam and to assess prognosis. In addition it is intended to study the factors which may have led to delay in diagnosis, for example local attitudes or misdiagnosis in hospital.

DEFINITION:

According to the Diagnostic and Statistical Manual of the American Psychiatric Association, the organic brain syndrome is a basic mental condition characteristically resulting from diffuse impairment of brain tissue function from whatever cause, either directly for example meningitis or indirectly in association with diseases such as pneumonia.

Acute brain disease or syndrome, toxic psychosis, confusional state and clounding of consciousness are among the many terms used to describe either the whole syndrome or one of its clinical manifestations. Because the mental changes result from metabolic insult to the brain, the same syndrome is also known as metabolic encephalopathy.

Its symptoms are impairment of orientation, memory, judgement, intellectural function, lability and shallowness of mood.

For descriptive purposes the term acute confusional state will be adopted to refer to the broad range of these related conditions.

From the psychiatric point of view these patients present emotionally into two groups. Some patients are quiet, apathetic withdrawn and have no hallucinations. This state is sometimes called simple confusion or quiet delirium. Other patients are sweaty tremulous, anxious and hyperactive. This is called agitated delirium. A few patients may experience both poles of emotional response during an acute confusional state.

Pathogenetically this syndrome is associated with and sometimes caused by brain tissue dysfunction. This impairment in brain tissue activity is caused either by a failure of metabolic or biochemical processes associated with a general lowering of organ function or by a loss through death of cellular units. The mechanism is yet unknown whereby toxic factors whether chemical or microbial in origin, interfere with normal cerebral metabolism. The insufficiency may be temporary and reversible with full recovery when the underlying cause is treated. This is usually the case with acute brain disease. On the other hand the insufficiency may be permanent and irreversible and full recovery is not anticipated after treatment of the causative factor. This is chronic brain disease. Acute brain disease can progress into chronic brain disease.

The main derangement in all confusional states is a disorder of attention. The patient fails to focus accurately on incoming stimuli to fix them clearly in his mind. He therefore lacks concentration and is easily distracted. For example in the course of a medical examination, he may abruptly stop his participation and respond to an irrelevant

conversation by other patients near him. Because of faulty attention this conversation may be misinterpreted as a musical song and the patient may start dancing until the other patients are kindly asked to keep quiet. When the patient talks, his conversation is fragmentary and often disjointed. From inability to attend to his surroundings he may be disorientated in time, place and personality. He will fail to remember any information after an interval. Hence his memory for recent events is lost.

Alteration of consciousness is variable. Some patients remain fully alert. Many show some reduction in the level of consciousness varying from unusual quietness and tendency to sleep to states of increasing lethargy, stupor and even coma. Hallucinations are usually visual or tactile. Symptoms tend to worsen at night. This is because the sensory stimulation is reduced resulting in frightening and threatening voices or objects.

AIMS OF THE STUDY.

1. To investigate the causes, clinical features and prognosis of acute brain disease in the medical wards of Muhimbili Medical Centre, Dar es Salaam.
2. To identify causes of delay in the diagnosis of acute brain disease and to suggest ways in which management and prognosis may be improved.
3. To compare my findings with those described in other countries.

PATIENTS AND METHODS.

All male and female patients above the age of seven years admitted in Muhimbili Medical wards, between 14th July 1981 and 31st August 1981 who had evidence of acute brain disease as pointed out in the definition were included in the study. The age, sex, address and date of admission of the patients were recorded. Educational level and occupation were included. The length of stay in hospital and the outcome of treatment were noted for each patient.

Every day's admissions was reviewed in the morning, evening and at night to ensure no patient was missed. Every patient was interviewed either directly or through a relative who accompanied him. In case a relative was not available, every effort was made to contact him or her on the following day. Interview was on the presenting illness, social life and the past medical and psychiatric illnesses of the patient. The traditional belief on the nature of the disease was sought.

Both physical and mental evaluation was carried out on admission and later until the patient either recovered or died to evaluate response to treatment. Physical examination included a general assessment of the patient to note whether or not the patient was febrile, anaemic, dehydrated or jaundiced. Evidence of wasting, peripheral tremors or oedema were also considered. Systemic examination included examination of the cardiovascular, respiratory, gastrointestinal, genito urinary and central nervous systems.

The clinical features of patients with malaria alone is shown in Table 3. Fever was common to all except one. Mental confusion was common to all patients. Those unconscious on admission were also mentally confused after they had gradually regained consciousness. Other symptoms included headache, weakeness diarrhoea, vomiting and fits.

Temperature ranged from 37 - 39°C. Those patients with equivocal neck stiffness were subjected to lumbar punctures. They all showed normal cerebral spinal fluid.

Table 4 shows patients with malaria and other associated illness. These showed symptoms and signs of malaria and those of the associated illnesses. Malaria lone, the average stay in hospital was 3.2 days. When malaria was present with other illnesses the average stay in hospital was 5.2 days.

Other diseases shown in Table 2 included chronic renal failure, meningitis, meningoencephalitis, subdural haematoma, louse-borne typhus and anaemia. Although fewer patients carried these diagnoses than those with malaria associated with other illnesses, these diseases were responsible for three out of four deaths (Table 7) in this series of patients. Furthermore three patients who were referred for psychiatric management after the treatment of their physical conditions belonged to this group of diagnoses. Only one patient of cerebral malaria with dehydration died. All the rest were managed and discharged home from the general medical wards.

Table 5 shows alcohol drinking habits among the patients in this series. Thirteen patients (62%) drank alcohol against eight patients

(38%) who did not drink alcohol. Age range of those who drank varied from 20 - 48 years. Of these there were eight males with age range of 36 - 48 years. All of them were either government employees or self employed. Five females with age range 20 - 22 years drank alcohol along with their husbands except one who was divorced and a barmaid. The eight patients who did not drink alcohol were all below 19 years old. They were either school pupils or students who were dependent on their parents.

Table 6 shows duration of symptoms before admission to hospital. Delay was marked in those patients in the fifth decade followed by those in fourth and third decades. Least delay was observed in teenagers.

Table 7 shows distribution of patients according to their state of consciousness and psychiatric conditions on admission and after improvement of their unconscious state. Fourteen patients (66.7%) were admitted in clouded consciousness and seven patients (33.3%) were admitted in either a semiconscious or unconscious state. Of those admitted in clouded consciousness ten (47.6%) had agitated delirium and four (19.1%) had quiet delirium. Those who were admitted unconscious were followed up while under treatment until their consciousness started to clear. This ranged between one to three days of treatment. Four patients (19.0%) had agitated delirium and three patients (14.3%) had quiet delirium.

Table 8 and 9 shows the prognosis in respect of age group and psychiatric condition prevailing on admission. Both tables show that younger patients have better prognosis than older patients. Table 8 further shows that fifteen patients (approx. 75%) were distributed in

the age group below 40 years. Overall seventeen patients (81%) were treated and discharged against four patients (19%) who died.

Deaths Two patients were diagnosed as chronic renal failure. The mortality rate of patients with chronic renal failure at a Medical Centre without dialysis services is high. Nothing much could have been done to save their lives. Two other patients who died could have been saved. One had been diagnosed as cerebral malaria. Although antimalarials were given, enough fluids were not given. Only one litre had actually been given by the second day when the condition had worsened. Possibly he died of dehydration. The other patient was misdiagnosed as alcoholic intoxication and hypoglycaemia until he died on the same day. There was no sign of external injury. Postmortem diagnosis was extensive subdural haematoma. Since it is only police cases where postmortems are done at the centre, no one knows how many patients of subdural haematomas die undiagnosed. To improve the prognosis of this group of patients would be to entertain the diagnosis of subdural haematoma in any patient with history of trauma as well as consulting a neurosurgeon who may make a burr hole to explore and evacuate the haematoma.

Table 10 shows distribution of patients according to their thinking disturbance. Of the fourteen patients (66.7%) with clouded consciousness, eight patients, (38.1%) had slowed thinking. Other less frequent disturbances included persecutory delusions, perseverance and wandering thoughts. Only one patient had no thinking disturbance.

Seven patients (33.3%) with semiconscious or unconscious state on admission but later evaluated showed gradual improvement of their mental state. Six patients (28.5%) had slowed thinking. The overall thinking disturbance was slowed thinking in fourteen patients (66.6%) followed by persecutory delusions in three patients (14.3%).

Speech disturbance is shown in Table 11. Overall disturbance was slowed speech in sixteen patients (76.2%) followed by irelevant speech in three patients (14.3%).

Orientation, memory, insight and intelligence disturbance were uniformely found in all patients.

The commonest disturbance of perception was visual and tactile hallucinations. The majority of patients, twelve (57%) were normal.

Commonest mood changes included depression and anxiety with irritability.

Attitude of relatives towards acute confusional state.

Cases Nos. 2, 6, 8, 9 and 10 showed a marked attitude of relatives towards witchcraft and traditional healers. Case No. 2 consulted a traditional healer for her menstrual problems. On admission with acute confusional state secondary to malaria and uraemia, she was found to have black point in the mouth which was said to be due to local medicine given by a traditional healer. Case No. 6 started with a traditional healer. On finding no improvement the relative sent her to several dispensaries, before she was admitted to hospital. The diagnosis was louse-borne typhus with acute confusional state. Case No. 8 had epilepsy diagnosed in 1979. Before diagnosis his mother took him to traditional

healers without improvement. Successful treatment was carried out in hospital. The mother of the patient developed so much confidence in hospital services that she rushed her son to hospital within an hour of present illness. Case No. 9 first attended hospital but the relatives requested early discharge only to take the patient to traditional healer. Case No. 10 was straight forward malaria. Her husband entertained a belief of witchcraft and had called for his parents to come for a consultation on this other side of the picture. I encouraged him to remain calm until his wife was cured. Injections are feared lest they worsen the witchcraft or evil spirt influences.

Cases of delay in diagnosing acute confusional states.

Traditional beliefs of witchcraft and fear of injections may keep the patient home for traditional treatment, thus reporting to hospital late. The belief that orthodox doctors are not capable of treating certain diseases like mental confusion brings the patient to hospital as a last resort.

In hospital delay in diagnosis may result from late referral from peripheral dispensaries and health centres either due to poor transport or failure to make an early diagnosis of acute confusional state.

In the Medical Wards quick diagnosis of psychogenic confusional state and quick referral to a psychiatrist at the expense of carrying out investigations can delay the diagnosis of acute confusional state.

The tendency that clouding of consciousness fluctuates from day to day and even over the course of the day may delay diagnosis if a single examination is carried out at the time the patient has mildest

degrees of clouding of consciousness.

As shown in Table 6 older patients delay to report for medical treatment.

Prognosis of Acute Confusional State.

Prognosis can be improved by educating the public to value the use of hospitals until they are advised otherwise by the doctors. This will enourage early reporting to hospital for an early diagnosis and treatment.

An easily treatable underlying disease carries a better prognosis than one in which the complete treatment is a difficult one. A single disease has a better prognosis than multiple underlying disease.

Younger age group has better prognosis than older age group.

The severity of psychiatric condition worsens the prognosis.

Rate of Resolution of mental symptoms.

Widely differing illnesses produce the same mental symptoms. The rate of resolution of mental symptoms depends on early diagnosis and treatment. In addition to specific treatment, dexamethasone to reduce cerebral oedema hastens the rate of resolution of mental symptoms. It was also observed in this series of patients that, those on whom lumbar puncture was done also improved very fast. Younger patients resolve faster than older patients.

Diagnosis and treatment.

This has been explained in detail in disease descriptions; Cases Nos. 1, 2, 3, 4, 5, 6 and 7.

RESULTS DISTRIBUTION OF PATIENTS BY AGE AND SEX

TABLE 1:

AGE GROUP (YEARS)	MALE	FEMALE	TOTAL NUMBER OF PATIENTS	PERCENTAGE
0-19	1	-	1	4.8%
10-19	3	4	7	33.3%
20-29	-	5	5	23.8%
30-39	2	-	2	9.5%
40+	6	-	6	28.6%
TOTAL	12	9	21	100.0%

TABLE 3: CLINICAL FEATURES OF PATIENTS WITH MALARIA ONLY

SER. NO.	NAME	AGE	SEX	DURATION OF SYMPTOMS (DAYS)	PRESENTING SYMPTOMS	PRESENTING SIGNS	PROGNOSIS
1.	D.J.	11	M	ONE	Weakness. Fever Fits. Loss of Consciousness.	Pallor. Temp. 39°C. Unconscious. Pulse 100/min. BP 100/60 mm. Hg. Puils equal. Normal reaction to light.	Treated and discharged after 4 days stay in hospital.
2.	R.M.	36	M	ONE	Weakness and chills, Diarrhoea, vomiting Confusion.	Dehydration. Temp. 37.5°C. Clouded consciousness. Pulse 80/min. BP 120/80 mm. Hg.	Discharged after 4 days treatment.
3.	Z.S.	16	F	FIVE	Fever. Headache. Fits. Vomiting Confusion	Temp. 38°C. Drowsy. Confused. Neck stiffness equivocal. Pulse 100/min. BP 120/80 mm. Hg.	Discharged 5 days treatment
4.	B.M.	19	F	HALF	Confusion. Picked from Police Station where she wanted to write a statement of death to her father	Confusion. Temp. 37°C. Pulse 80/min. BP 100/80 mm. Hg.	Discharged after 2 days treatment
5.	S.A.	18	M	TWO	Fever. Fainting Photophobia Confusion	T. 38°C. Photophobia. Agitation. Confusion. Pulse 96/min. BP 120/80 mm. Hg.	Discharged after 5 days treatment

CONT..... TABLE 3:

SER. NO.	NAME	AGE	SEX	DURATION OF SYMPTOMS (DAYS)	PRESENTING SYMPTOMS	PRESENTING SIGNS	PROGNOSIS
6.	E.K.	40	M	TEN	On and off fevers. Vomiting. Loss of consciousness.	T. 38°C. Confusion	Discharged after 1 day treatment.
7.	A.A.	20	F	ONE	Fever. Sudden fall.	T. 38°C. stuporous. Pulse 100/min. BP 120/80 mm. Hg. Pupils equal. Normal reaction to light	Discharged after 5 days treatment
8.	A.K.	12	F	SEVEN	Intermittent Fever. Headache. Confusion	T. 38°C. Splenomegaly 6 cm. below costal margin.	Discharged after 2 days treatment.
9.	M.N.	45	M	THREE	Joint pains. Fever. Loss of appetite. Weakness. Loss of consciousness	T. 39°C. Dehydrated. Pulse 114/min. BP 100/60 mm. Hg.	Died after one day stay in hospital

TABLE 4: CLINICAL FEATURES OF PATIENTS WITH MALARIA AND OTHER ASSOCIATED ILLNESSES

SER. NO.	NAME	AGE	SEX	DURATION OF SYMPTOMS (DAYS)	PRESENTING SYMPTOMS	PRESENTING SIGNS	PROGNOSIS
1.	J.M.	20	F	SEVEN	Fever. Loss of Appetite. Fits. Confusion. Incontinence of urine	T. 37.3°C. stuporous BP 170/100 mm. Hg. Range 190/140 to 110/80 mm. Hg. at death	Uraemia + malaria. Patient died after stay in hospital for 14 days.
2.	Z.E.	8	M	ONE	Fits. Fever. Headache. Confusion. Known epileptic.	T. 39°C. Left sided twitching. Agitated.	Left temporo-occipital epilepsy with malaria. Discharged after 5 days treatme in hospital.
3.	A.K.	13	M	THREE	Epistaxis. Fever Headache.	Pallor. Temp. 37.5°C. Pulse 120/min. BP 100/70 mm. Hg.	Malaria + anaemia Discharged after tre ment for 5 days.
4.	M.A.	20	F	THIRTY	Weakness. Headache. Fever. Confusion. Hyperactive at night.	T. 38°C. Agitated. Pulse 100/min. BP 160/100, 180/130 170/130 mm. Hg.	Hypertension + malar Treated and discharg on request before complete cure after 3 days treatment.
5.	M.P.L.	45	M	SEVEN	Intermittent fevers. Confusion. Pain in right hypochondrium	T. 38.6°C. Tender palpable liver, 4 cm. below costal margin. Stuporous. Later developed jaundice.	Hepatitis + malaria. Treated and discharg after 12 days stay in hospital.

DIAGNOSIS AND TREATMENT.

1. MALARIA.

All patients suspected of malaria were diagnosed by demonstrating malaria parasites in the blood after staining their blood slides with Giemsa stain. Lumbar puncture was done to those patients who were either admitted unconscious or had fits to exclude other diagnoses like meningitis and encephalitis. All patients had normal cerebral spinal fluids.

Treatment was both specific and symptomatic. Specific treatment ranged from 140mg. - 200mg of chloroquine according to the age of the patient. A start dose was given intramuscularly. Eight-hourly doses followed for the next 24 hours. This was followed by chloroquine tablets 2 daily for three days. Symptomatic treatment included dexamethasone 5mg once or twice daily. It was given until the patients' mental state cleared. Parenteral fluids were given when dehydration was demonstrated.

II. Other diseases were diagnosed and treate as:

(i) Uraemia: Case No. 1 and 2

(ii) Meningitis: Case No. 3

(iii) Meningoencephalitis: Case No. 4

(iv) Subdural haematoma (misdiagnosed): Case No. 5

(v) Louse borne typhus: Case No. 6

(vi) Anaemia: Case No. 7

Case No. 1: URAEMIA.

A.R. was a 48 year old male admitted in Muhimbili Medical Centre on 6th August 1981. A relative said that the patient was well until eleven days prior to admission when he complained of fever. Three days before admission he complained of abdominal pain, vomiting followed after every meal. There was no diarrhoea but stool was black. Anorexia and weakness were prominent. The reason given for not reporting to the hospital early was that the patient was being treated by a traditional healer. They decided to come to hospital because there was no improvement. No history of previous medical illness.

On physical examination, the patient was wasted, weak, confused dehydrated but not jaundiced or anaemic. Pulse rate 100/min. regular and moderately thin. Blood pressure 120/70mm.Hg. No lymphadenopathy. The neck was soft and Kernig's sign negative. Chest was clear on both percussion and auscultation. Heart was not enlarged and murmurs were not detected by auscultation. After two weeks the pressure was noted to have risen to 140/100 but started to drop three days before he died.

Mentally he was confused, disorientated, unco-operative and memory for recent events was absent. He showed perseverance and his speech was slowed. He was generally depressed and his insight was partial.

Investigations showed a blood area of 194mg% four days before he died. Serum potassium was 4.1mEq/Litre.

Treatment was based on a diagnosis of chronic renal failure. Restriction of protein diet and regulation of fluid input depending on fluid output was carefully carried out. Mild tranquillisation with valium 10mg. intramuscularly was administered when confusion and

sleeplessness had become marked. He died of uraemia 21 days after admission.

Case No. 2. URAEMIA.

J.M. was a 20 year old barmaid admitted in Muhimbili Medical Centre on 14th July 1981. Relatives said that the patient was married and divorced 4 years ago due to illness said to have been of renal origin. She has one child 4 years old. She had complained of irregular menstrual periods for the previous three months. Hence she consulted a traditional healer who used to give her local medicines. One week before admission she had fever and vomiting. While at Ukonga dispensary she vomited and convulsed. She urinated during the episode. Socially she drank alcohol but did not smoke.

On physical examination she was afebrile with a temperature of $37^{o}C$. The mouth had a black paint said to be due to local medicine given by a traditional healer. There was no sign of head injury. Pupils were equal and reacting well to light. No sign of meningeal irritation. Deep tendon reflexes were normal including a Babinski flexor response. Pulse rate was 88/min. regular and in good full volume. Blood pressure was 190/140mm.Hg. Chest full of transmitted sounds - possibly due to aspiration of vomitus. Heart and abdomen were both normal. Vaginal examination showed normal cervix and retroverted uterus which was free and non-tender.

Five hours following admission she had a clouded consciousness and confused. She was completely disorientated in place, time and person. At nitht she was agitated and overactive. She talked her vernacular and she did not want to cover herself. Her speech was

slowed, thinking was fragmented and slowed. Mood was of general depression unless she was delirious at night. Insight and memory were completely lost. She requested a bottle of beer many times.

Investigations showed protein 500mg/dl in urine. Blood sugar 44mg/dl. Malaria parasites positive ++. Blood urea varied from 275 - 400mg/dl. at death. Serum potassium 5.0mg/Litre and creatinine of 11mg/dl. few days before death.

She died of uraemia after two weeks stay in hospital. Treatment was essentially symptomatic with protein restriction and careful balancing of fluid administration plus high caloric diet.

Case No. 3. MENINGITIS.

I.H. a 48 year old Traffic Officer was admitted in Muhimbili Medical Centre on 7th August 1981. Relatives reported a history of acute onset of fever, semiconsciousness, confusion and restlessness of four days duration. He denied a history of trauma, excessive intake of alcohol or any history of epilepsy.

On physical examination the patient was febrile with a temperature of 38°C. Pulse rate was 100/min. regular and good volume. Blood pressure was 170/100mm.Hg. The neck was stiff and Kernig's sign was positive with right sided hemiparesis. Pupils were symmetrical and equally reacting to light. Chest revealed bilateral coarse crepitations by auscultation. Abdomen was scaphoid without organomegally. Heart was not enlarged.

On the second day of admission he was conscious but confused. Orientation with time, place and personality was poor. It remained

so until 16 days after admission. He had a decreased motor activity, irrelevant speech and thinking which was fragmented and slowed. He manifested persecutory delusions, thinking that his disease was due to his relationship with a certain old man. Insight was poor. Mood was labile, sometimes he was depressed and another time he appeared normal. Memory for recent events was affected for he did not know how he entered hospital. Intelligence was reduced. A standard VIII man could not know the answer of 100 - 7 = 96 - 7 = 100.

On admission a diagnosis of meningitis, was made clinically. Urgent lumbar puncture with cerebrospinal fluid analysis showed pandy test positive. Protein was more than 100mg%. No pus cells were noted. Microscopy did not reveal any organisms. Cerebrospinal fluid sugar was reduced to 28mg%. Malaria parasites were positive.

The patient was started on crystalline penicillin 5 mega unit 6 hourly - intravenously for 14 days. Dexamethasone 5mg. intramuscularly 8 hourly for one week was administered. Aprinox 5mg. daily to reduce the raised blood pressure. Patient was afebrile by third day of starting treatment. Chest signs cleared by twelfth day. Neck stiffness and Kernig's sign disappeared by fifteenth day. Mental signs were not noted by eighteenth day. He was put on physiotherapy and was discharged on 12th September 1981.

Case No. 4: MENINGOENCEPHALITIS.

Name: B.K. Age: 30 years Sex: Male Occupation: Sisal cutter.

Admitted: 21/7/81

Symptoms: Fever, headache, convulsions, confusion and bed sores for 14 days.

Signs: Temperature 38°C. Dehydrated. Confusion. Neck stiffness was equivocal. Kernig's sign negative. Bed sores on buttocks. Other systems were normal. Mentally he was very confused disorientated in time, place and person. He had no memory for recent events.

Investigations: Lumbar puncture urgently done was blood tinged, which cleared after a short time. Results included Pandy test - strongly positive. RBC 100/cu.mm. Total protein 45mgs%. Cell count 0/cu.mm. There was no organism seen on Gram stain or on Nelson stain. Culture did not reveal any bacterial growth. Cerebral spinal fluid sugar was initially 45mg/dl. and two weeks later it was 77mg/dl. Blood sugar was not known. Blood slide for malaria parasites was negative.

Diagnosis: Meningoencephalitis

Treatment: Crystalline penicillin 5 mega unit 6 hourly for two weeks. Dressing of would with Eusol. Largactil 50mg. twice daily.

Transfer: He was transferred to psychiatric unit for the management of psychiatric symptoms.

Case No. 5: SUBDURAL HAEMATOMA:

A.T. was a 40 year old male admitted as a Police Case in Muhimbili Medical Centre on 21st August 1981. The accompanying policeman reported that they picked him up after he had fallen down on getting off a bus. No relative could be obtained to give a full history of the patient. However when the patient gained consciousness he said he had gone to drink local brew and he was walking to his

home. He did not know he was now a patient in hospital.

On physical examination he was semiconscious smelling of alcohol , responding to his name but could not obey commands. Pupils were symmetrical, reacting well to light. They were not dilated. The head was normal, no sign of would or fracture. He was afebrile with a temperature of 37°C. Pulse rate was 60/min. regular and full volume. Blood pressure was 120/80. Respiration rate was 24/min. He was moderately dehydrated. Abdomen was scaphoid and no organomegally on palpation. Chest was clear. Deep tendon reflexes were within normal range. Babinski was flexor in response. Neck was soft and Kernig's sign negative.

Five hours following admission, the patient was conscious but confused and clouded. He was completely disorientated to time, place and personality. Motor activity was slowed, but cooperative. Speech was slowed and confabulated that he does not drink more than a bottle of alcoholic drink. Insight was absent. A flat mood predominated. Memory and intelligence were reduced.

A diagnosis of alcoholic intoxication and hypoglycaemia was entertained until the patient died on the same day. Postmortem diagnosis was a left sided big subdural haematoma and small one on the right side. No external injuries or fracture.

Case No. 6: LOUSE BORNE TYPHUS.

R.A. a 13 year old girl was admitted in Muhimbili Medical Centre, female ward on 28th August 1981. History was taken from a relative who said that she had been brought to Dar es Salaam from Bukoba to employ

her as a "Yaya" (Baby Sitter). She faired well until four days before admission when she changed her behaviour and attitude towards her employer. She became stubborn and unco-operative. She was careless to the baby. When assigned to do anything she told the employer to do it herself. On the following day she bursted into crying, singing and laughing inappropriately. This was intolerable. The employer took her to a traditional healer who diagnosed "Shetani" (devil) in her. They spent a night at the witch doctor's house. At night the episode recurred and she was sent to Mwananyamala and Juwata dispensaries before admission at Muhimbili. Relative denied history of fever, fits or mistreatment to the patient.

On physical examination, she was afebrile with temperature 37°C. Pulse rate 90/min. regular and good volume. Blood pressure 100/70mm.Hg. Conscious, no palor. Neck was soft and Kernig's sign negative. Deep tendon reflexes were normal. The chest was clear on percussion and auscultation. The heart was not enlarged and no murmurs were heard on auscultation. In the abdomen there were no palpable masses. Extremities were normal but scalp revealed lice eggs and scars at hair-neck lining. No live lice was seen. Examination of other members of the house showed live lice in the baby's hair.

Mental evaluation showed a conscious but confused young girl, reciting short chapters from the Quran in memory. Moving from one end of the ward to the other. She was disorientated in time, place and person. Hallucinations were present. She knelt obediently asking her father (not present) to forgive her although she did not know her faults.

She showed perseverance by saying Rukia, Rukia etc. Her speech was slowed and irrelevant. When asked to mention her name she said "Bukoba". Mood was inappropriate, sometimes smiling and at other times she remained quiet and withdrawn. Memory and insight were poor.

A diagnosis of acute confusional state with organic cause was entertained.

Investigations included a lumbar puncture under sterile conditions. Results showed protein content of less than 20mg%. Pandy test was negative, cells - zero. Gram stain and Ziehl Nelson stain did not show organisms. Widal test was negative. S. typh, "O" less then 1/20 S. typh,"H" - reagents out of stock.

Proteins OX 19 - 1/5120, OX 2 1/80, XX 1/20.

Full blood picture: Haemoglobin 10.6g% WBC 19,200/cu.mm.

Differential: Neutrophils 28% Lymphocytes 68%, Stab cells 4%

Film: normochromic, normocytic.

Final Diagnosis: Louse-borne typhus and acute confusional state.

Treatment included chloramphenicol 500mg. 6 hourly for 10 days. Tranquillizers included 10mg. Valium intramusculary at night to prevent delirious outbursts. Patient is still in the ward at the time of writing.

Case No. 7: ANAEMIA:

Name: A.J. Age: 22 years. Sex: Female. Occupation: Housewife.

Admitted: 26/7/81 Discharged: 12/8/1981

Symptoms: Admitted with history of fever, weakness palpitations and swelling of limbs for two months. A week before admission she changed

her behaviour, by being frightened, forgetful and blank staring. She gave a history of child birth six months previously.

Signs: On examination, she was anaemic, mild oedema of legs. She was neither febrile nor dehydrated. She was not jaundiced either. Her pulse rate was 120/min. regular and good volume. Blood pressure was 120/60mm.Hg. Jugular venous pressure was not elevated. Heart was not enlarged on palpation and both first and second heart sounds were heard and murmurs were not detected on auscultation. In the abdomen the liver was enlarged about 6cm. below the costal margin. The spleen was not palpable. Chest was clear. Apart from fine tremors of fingers in outstretched hands the central nervous system was normal.

Mental evaluation showed a confused patient with clouded consciousness. She remained quiet during the day but restless and agitated at night. Her speech was slowed with occasional muteness. She showed wandering thoughts and visual hallucinations by seeing people ordering her to sleep. Her memory for recent events was lost.

Investigations: Full blood picture - haemoglobin 4.8gm/dl.
WBC 19,000/cu.mm.

Differential: Neutrophils 24%. Stab cells 24%. Metamyelocyte 4%. Myelocyte 6%. Lymphocytes 42%. Nucleated RBC/100 EBC. 5 MCV 115.

Film: Normochromic, Anisocytosis ++. Macrocytosis. Polychromasia ++ Leucocytosis left shift. No malaria parasites was seen in blood. A bone marrow showed evidence of mild iron defiency.

Diagnosis: Anaemia. Iron deficiency. Two units of blood transfusion elevated haemoglobin to 9.5g/dl. Total dose Imferon infusion was given. A chloroquine course was also given. Patient was discharged through psychiatric clinic for follow up after treatment in the general ward for eighteen days.

Case No.8 CEREBRAL MALARIA AND EPILEPSY (TRADITIONAL HEALER CONSULTED)

Z.E. was a 8 year old boy who was admitted in the male ward of Muhimbili Medical Centre on 11th August 1981. History taken from his mother showed that the patient had fever and fits involving the left side of the body one hour prior to admission. He had had recurrent convulsions since childhood. He was first diagnosed as a problem of epilepsy in May 1979. Since then he has been a regular attendant of epilepsy clinic. He has never defaulted to anticonvulsive drugs. Before the above diagnosis was made the patient used to be sent to traditional healers without much improvement. The last fitting was a year ago. So mother believes that the hospital drugs are superior to traditional herbs. Family history revealed epilepsy in the patient's uncle's son. There was no history of trauma and the patient was a full term baby with a spontanous vaginal delivery.

On examination, he was in good nutritional status. No scars on face. Sedated from casuality. No tongue bite and the pants were dry. He was febrile with a temperature of 39°C. Dehydration, anaemia and jaundice were not present. Pulse 100/min. and blood pressure 110/70 mm.Hg. Heart was not enlarged and no murmurs were detected. The viscera in abdomen were not palpably enlarged. Neurological examination was normal.

Mental evaluation was done on the second day when the patient was out of sedation. Patient was noted to have a clouded consciousness with confusion, remaining quiet during the day but agitated, crying with delusions and hallucinations at night. He said he saw some boys hiding under hospital beds with sticks ready to beat him. His mood varied from depressive and flat during the day to irritable and anxious at night. His memory for recent events as well as insight were reduced. Intelligence could not be assessed because he has never been to school. He was also unco-operative.

Investigations included a blood slide for malaria parasites, the result of which was recorded as ++++ positive. An electroencephalogram done in 1979 had shown bilateral epileptiform like changes. The left temporo-occipital region appeared to be more affected.

The patient was on anticonvulsive therapy. At casualty he was given 10mg. of valium intramuscularly to control convulsions. He was put on chloroquine 150mg. intramuscularly 8 hourly for 24 hours followed by two tablets daily for three days. On the following day he was afebrile with temperature of 37°C. Since he became delirious at night he also received Largactil 100mg. and dexamethasone 5mg. intravenously to tranquillise him and to reduce the intracranial pressure respectively. On the next day after this treatment, the patient was quiet both during the day and night. He was discharged on the fourth day after admission to be followed up in the medical outpatient clinics.

Case No. 9: CEREBRAL MALARIA + HYPERTENSION (TRADITIONAL HEALER CONTACTED.

M.A. a 20 year old girl was admitted in Muhimbili Medical Centre on 11th August 1981. She was admitted confused and her mother reported that the daughter complained of weakness, excessive sleep, arthralgia and fever during the evenings for a duration of one month. She thought it could be malaria and she insisted on her to take chloroquine tablets without success. One day before admission, mental changes started at night. She started by being afraid. She insisted to have her mother's company and she feared darkness. As a result lights were left on throughout the night. She complained that she was seeing snakes on the wall. This raised the suspicion that she was being possessed by devils. Her husband was on safari, so she decided to take her daughter to a private clinic in town. At the clinic the grandmother refused the patient to be given any injection because in case it is Shetani" (evil spirit) the disease would worsen. So the doctor referred the patient to Muhimbili.

Past medical history did not reveal any illness. Socially she is the only female child in a family of three children. She drinks alcohol and loves dancing greatly. She does not smoke. She has a fiance.

On examination she was in good nutritional state. She was afebrile on admission. Pulse was 80/min. regular and good volume. Blood pressure taken in three readings for 24 hours were 140/90, 160/100, 170/130mm.Hg. The heart was not enlarged and on auscultation no murmurs were detected. Abdominal examination did not reveal any

palpable masses. Chest was clear on auscultation. Apart from fine tremors of hands the central nervous system was normal.

Mental evaluation revealed an agitated young girl who was restrained after she had run away shouting widly. She was overactive and aggressive. She appeared confused and clouded in consciousness. She was quiet disorientated in time, place and personality. Speech was slowed and thinking of her mother and grandmother all the time. Mood was labile, sometimes anxious and euphoric and at other times depressed and anxious. At night she was irritable and suspicious. Hallucinations consisted of seeing snakes on the wall at night. Memory and intelligence were markedly reduced.

Investigations revealed malaria parasites +2. Other investigations failed because relatives did not want injections and furthermore if it was an evil spirit which suck patient's blood withdrawing more blood would injure the patient more. Grandmother was the centre of these myths.

The patient was started on chloroquine 200mg. intramuscularly stat, then followed by 100mg. 6 hours later and then chloroquine tablets 2 daily for three days. Valium 20mg. and Largactil 200mg. were given intravenously and slowly during delirious moments. On the second day she was talking clearly although she still looked suspicious and disorientated in time. On the third day she was much better, co-operative and going to toilet alone. Her mother requested for a discharge. Permission was granted. She was discharged on chloroquine, aprinox and tranquillizers.

At night, the same day of discharge, the patient became delirious. Grandmother directed the mother of the patient to go to Bagamoyo for a traditional healer. Four days later I visited the home when the patient was already back from Bagamoyo. In fact she had not changed. She was alert but very suspicious. She clung to her father and was suspecting me of having come with something harmful. Her mother told me that the diagnosis made by traditional healer was "mashetani" (evil spirits). When I suggested some injections the father rejected forthwith and I could learn from their faces that my suggestion was criminal. I left in peace.

A week later I returned. The father was not present. The grandmother was present. She did not even bother to greet me. Mother was fairly welcoming. However she refused me to see the patient by asserting that she was asleep. On hearing my voice the patient peeped with the door slightly opened. She murmured at me and threw her hand at me. I learnt that I was generally unwanted. She showed she was not cured since she was still hidden from public. Mother told me if she developed fever she would be brought back to hospital. Otherwise traditional healers would attend her. I stopped following up the patient.

Case No. 10: CEREBRAL MALARIA (WITCHCRAFT ENTERTAINED).

A.A. a 20 year old married female with one child was admitted in Muhimbili Medical Centre on 10th August 1981. The husband reported that the patient fell suddenly from bed and lost consciousness. He denied history of drug ingestion or trauma. She did not show abnormal behaviour before the acute illness. Fever was present but no history ho vomiting or diarrhoea.

She drinks about one to two bottles of beer at a sitting. She does not smoke. He denied a history of epilepsy in the family. Husband thought of witchcraft to be the cause of the disease.

On examination she was febrile with a temperature of 39°C. She was neither dehydrated nor anaemic. The pulse rate was 100/min. regular and good volume. Blood pressure was 120/80mm.Hg. Heart was not enlarged and no murmurs were detected. Systems were quite normal including pupils which were equal and well reacting to light.

Mental evaluation was not possible in the first to third day because the patient was unconcious, only reacting to deep pain.

On 4th day the patient was of clouded consciousness and confused. She was disorientated in time, place and personality. She had variable motor activity, being restless and agitated mainly at night and decreased activity at day time. Speech was slowed and at times stopped responding. Thinking was slowed and fragmented. Insight and intelligence was markedly poor. Mood varied from anxious and irritable at night to depressed and flat at day time. She had visual and tactile hallucinations seeing people standing by her, touching her buttocks and giving injections, hence screaming at night. She lost memory for recent events as she could not know how she was not with her baby who was left at home.

Investigations included a lumbar puncture under sterile condition. A clear fluid was obtained. Protein was less than 10mg.%. Pandy test was negative. No cells. Microscopy did not reveal any organisms. Cerebral spinal fluid sugar was 58mg/dl. where blood sugar was 88mg/dl.

Urea was 20mg/dl. Electrolytes were not done because machine was out of order. Blood slide for malaria parasites was positive +2.

The diagnosis of cerebral malaria was made. Chloroquine course and dexamethasone were given. Valium was given to control the mental symptoms. By 5th day the patient was well. She was discharged on the sixth day to continue with treatment at home.

TABLE 5: DISTRIBUTION OF PATIENTS ACCORDING TO DRINKING HABITS

AGE GROUP	DRINK ALCOHOL	DO NOT DRINK ALCOHOL
0-19	NIL	8
20-39	7	NIL
40+	6	NIL
TOTAL	13 (62%)	8 (38%)

TABLE 6: DISTRIBUTION OF PATIENTS ACCORDING TO DURATION OF SYMPTOMS BEFORE ADMISSION

Age Group (years)	Average Duration (Days)
0-19	2.6
20-39	4.8
40+	7.7

TABLE 7: DISTRIBUTION OF PATIENTS ACCORDING TO STATE OF CONSICIOUSNESS ON ADMISSION AND PSYCHIATRIC CONDITION ON ADMISSION AND AFTER THE IMPROVEMENT OF THE UNCONSCIOUS STATE

STATE OF CONSCIOUSNESS ON ADMISSION	CLOUDED AND CONFUSED	SEMICONSCIOUS AND UNCONSCIOUS
Number of Patients on Admission	14 (66.7%)	7 (33.3%)
Agitated delirium	On admission	After 1-3 days of treat
	10 (47.6%)	4 (19.0%)
Quiet delirium	On admission	After 1-3 days of treat
	4 (19.1%)	3 (14.3%)

TABLE 8: DISTRIBUTION OF PATIENTS ACCORDING TO THEIR AGE GROUP AND THEIR PERIOD OF STAY IN HOSPITAL AND PROGNOSIS

AGE GROUP (YEARS)	NUMBER OF PATIENTS	AVERAGE STAY IN HOSPITAL (DAYS)	NUMBER OF PATIENTS DISCHARGED	NUMBER OF PATIENTS DIED
0-19	8	4.6	8	-
20-39	7	7.2	6	1
40+	6	1.2	3	3
TOTAL	21	23.8	17 (81%)	4 (19%)

TABLE 9: PSYCHIATRIC CONDITIONS AND AGE GROUP OF PATIENTS AND THEIR PROGNOSIS

PSYCHIATRIC CONDITION	CLOUDED AND AGITATED DELIRIUM	UNCONSCIOUS AND LATER AGITATED DELIRIUM	CLOUDED AND QUIET DELIRIUM	UNCONSCIOUS AND QUIET DELIRIUM
AVERAGE STAY IN HOSPITAL (DAYS)	5.2	9	10.8	13.6
AVERAGE AGE OF EACH GROUP (YEARS)	18.5	25.2	38.5	46.5

TABLE 10: DISTRIBUTION OF PATIENTS ACCORDING TO THEIR THINKING DISTURBANCE

THINKING DISTURBANCE	CLOUDED AND CONFUSED PATIENTS ON ADMISSION	SEMICONSCIOUS AND UNCONSCIOUS PATIENTS ON IMPROVEMENT
Slowed	8 (38.1%)	6 (28.5%)
Persecutory delusions	2 (9.5%)	1 (4.8%)
Perseverance	2 (9.5%)	-
Normal	1 (4.8%)	-
Wandering	1 (4.8%)	-
TOTAL	14 (66.7%)	7 (33.3%)

TABLE 11: DISTRIBUTION OF PATIENTS ACCORDING TO THEIR SPEECH DISTURBANCE

SPEECH DISTURBANCE	CLOUDED AND CONFUSED PATIENTS ON ADMISSION	SEMICONSCIOUS AND UNCONSCIOUS PATIENTS ON IMPROVEMENT
Slowed	10 (47.7%)	6 (28.5%)
Excessive	2 (9.5%)	-
Irrelevant	2 (9.5%)	1 (4.8%)
TOTAL	14 (66.7%)	7 (33.3%)

DISCUSSION.

The present study is merely descriptive and no attempt was made to calculate incidence or prevalence rates. Twenty one patients seem to be a small sample, but taking into account that these were collected in forty five days, then the relative frequency of admissions of this group of patients in the medical wards of Muhimbili Medical Centre is one patient of acute confusional state for every two days. This is consistent with the observation by German (1972), that organic mental illness are extremely common in East Africa. In Butabika Mental Health, Uganda, Cox (1978) found 21 male patients (22.3%) of organic confusional state being the second frequent diagnosis to number one diagnosis of schizophenia - 35 male patients (37.2%). His total number of patients was 94. The wide prevalence of acute confusional state in particular and organic psychoses in general and the frequency with which it is associated with general hospital admissions would suggest that a thorough training in psychiatry is essential for a physician in Tanzania.

Most patients, twelve (57.1%), were in their second and third decade of life. This compares with Nadeem and Younis (1977) in Sudan. The low percentage in the first decade life does not mean that this age group is not vulnerable, but indicates that below the age of seven the patients present to paediatricians. While fewer patients were noted in the fourth decade in this study the number started to rise with age, six patients (28.6%) in the fifth decade. The explanation for this phenomenon could be that in the second and third decade

of life the brain cells are still immature and that in the fifth decade onwards the cells are decaying. Both types of cells are more vulnerable to CNS disturbance.

The pattern of alcohol drinking is shown in Table 5. Thirteen patients (62%) drank against eight (38%) who did not. The latter were below the age of 20 years while the former ranged from 20 - 48 years. In the drinking group there were eight males and five females. Although these are small figures, still they show that men are overrepresenting the drinking group. This is similar to previous reports in West Africa, (Asuni (1974), Odejide (1975) and Odejide and Olatawura 1977). Our results show that four females were married and their husbands also drank alcohol, thus allowing their wives to drink along with them. This attitude exists in Zambia (Tongue 1976). The other female was a barmaid. For her drinking was an occupational hazard.

The age range of the drinking group shows that increase in alcohol consumption increases with advancing age and that all occupational and educational categories are represented. These views are also shared in West Africa by Odejide (1979) and Odejide and Olatawura (1979).

Although females have been over represented Claver (1974) observed female alcoholism developing rapidly and seemingly for the same reasons as male alcoholism.

Excessive alcohol consumption in Tanzania is evident. While addressing students at the Kivukoni College on May 26, 1976, President Nyerere said: "We cannot call ourselves revolutionaries if our leadership consists of people whose main preoccupation is to empty bottles of beer -

Drunkenness and serious nation building do not go together"..... he further said, "The problem of drunkenness is not confined to the leadership only but is also rampant among the people, some of whom have taken to drinking "Moshi" at the expense of nation building ..... a quotation by Tongue (1976).

The relevance of alcohol as an independent cause of acute mental state in our series of patients is not entertained because there was no case of delirium tremens. However thirteen patients (62%) in our series lay in the same age range (20-48 years) as that of the drinking group, as shown in Tables 8 and 5. Does alcohol play a role? This answer can be answered correctly if a control study is carried out. However in developed countries where alcoholism has been rampant for many years, at present 10% presenting to alcoholic units will have a clinically obvious organic brain syndrome. For the remaining 90%, over half of them can be shown to have shrinkage of brain although they are clinically intact. Mental assessment show some degree of cognitive disturbance, problem solving ability is reduced, abstract thinking is defective, memory and psychomotor speech are impaired (Leishman 1981). German (1972) observed that alcohol psychosis was extremely common in East Africa. Wood (1968) found that 13% of Butabika Mental Hospital patients, alcohol was the important cause of acute mental disorder.

Alcohol intoxication and its associated effect of hypoglycaemia joins other organic factors such as birth trauma, brain damaging illnesses of infancy and subnutrition as possible causes of sub-clinical

cerebral dysfunction. In view of this Castle (1970) found excessive high frequency of posterior slow activity in the EEG which he said was a physiological response to disease on deprivation nutritionally in early stages of brain development. It is therefore believed that there is a large reservoir of subclinical cerebral dysfunction in Africa which facilitates the appearance of acute brain disease in response to physical and psychic stresses.

The diagnostic criteria for organic brain syndrome in psychiatric research was proposed by Freighner et al. (1972). By this criteria two conditions must be met. Firstly, there has to be impairment of orientation, memory and deterioration of other intellectual functions. Secondly, one disturbance in the first condition plus a known probable physical cause of organic brain syndrome.

The commonest physical illness in our series of patients was malaria. This is quite understandable in a country where malaria is endemic. This is consistent with the findings of Nadeem and Younis (1977) in Sudan.

Cerebral malaria and other malignant forms of falciparum malaria do affect people of all age groups and of different socio-economic strata. In the present state of knowledge about malaria immunity, we are not able to explain the exact nature of this phenomenon. However Adams and Magraeth (1971) think that there are certain individual differences in the ability to develop immunity against malaria right from early childhood to adult life due to yet unidentified genetic and environmental factors. Cook and Lewis (1975) found high values of

immunoglobulins in healthy Zambian Africans. But Alamin (1981) found that all the adult Zambian patients with cerebral malaria had demonstrated low values of immunoglobulins in serum and cerebral spinal fluids. This probably suggests immunoimpotence in these patients.

Pathologically there is marked swelling of the brain. Microscopically the picture is more of encephalitis than meningitis. There is considerable cerebral oedema with perivascular infiltration, mainly with lymphocytes monocytes and plasma cells. Plasmodium falciparum is found in small vessels. Leptomeninges show mild inflammation and congestion.

In our series of patients, two were diagnosed as cases of uraemia. Both of them manifested mental symptoms. The syndrome of uraemia both in acute and chronic renal failures is accompanied by disordered mental functioning. Stenback and Haapanens (1967) had a large consecutive series of patients in their renal unit. Mental manifestations occured in 60 per cent, rising to 75 per cent when the blood urea exceeded 250mg/dl. Mental changes were as common in the acute as in the chronic uraemic patients. So it is advisable to consider uraemia in the differential diagnosis of delirium of uncertain aetiology

Sometimes, probably depending on premorbid personality the picture may resemble functional mental illness. Baker and Knutson (1946) reported a patient initially diagnosed as suffering from catatonic schizophrenia, but with rapid flunctuations between psychotic and normal periods. Close examination showed organic mental symptoms. So obvious impairment of physical healthy with weakness, wight loss and anorexia, should alert one to the true diagnosis of organic mental disease as opposed to the

functional mental disease alone.

Although mental changes become commoner with increasing elevation of blood urea, there is no linear relationship (Stenback and Haapanens, 1967). Therefore other abnormalities must play a direct role with elevation of blood urea serving mainly as an indicator of the severity of overall metabolic disturbance. Other protein derivatives such as uric acid or other unidentified toxins and electrolyte disturbances are thought to be responsible for the mental symptoms along with elevated blood urea.

Gradual improvement usually follows dialysis, with return of mental clarity a short while after chemical normality has been achieved. However too rapid dialysis worsens the clinical condition. Headache, muscle twitching, fits and confusion may develop, sometimes progressing to coma with signs of brain stem compression (Kennedy et al. 1962; Peterson and Swanson, 1964; Mawdsley, 1972). This is commoner when the initial level of blood urea has been very high and usually occurs towards the end of dialysis. The exact cause is unknown.

Since the Muhimbili Medical Centre has no dialysis services it was difficult to observe the beneficial and harmful effects of dialysis discussed above. However in future if the dialysis services will be provided, the points discussed on dialysis will be applied for the benefit of such patients.

One patient in the series of patients studied had pyogenic meningitis. Mental disturbance in pyogenic meningitis takes the form of an acute confusional state, with drowsiness extending to coma. Sometimes they have hallucinations, excitement and other symptoms of delirium.

In tuberculous meningitis mental symptoms of acute confusional state can even precede the overt signs of meningeal infection. Williams and Smith (1954) noted clouded consciousness as the first definite sign, the patient lying quietly and without apprehension. In contrast to pyogenic meningitis, the patient with tuberculous meningitis with delirium sleeps when alone only to be disturbed when aroused.

With present day management, a full physical and mental recovery can usually be obtained. Williams and Smith (1954) found that the majority of patients returned gradually to their former efficiency. Lorber (1961) followed up 100 patients and found that seventy-seven had made complete recovery, including some who had very severe neurological abnormalities during the active phase of the illness. It should be emphasized that early diagnosis and treatment is very essential to good prognosis.

One patient in the series carried the diagnosis of meningoencephalitis. After treatment with antibiotics for two weeks in the medical ward he was referred to psychiatrists because he was very psychotic and nothing much could have been done to help him in the medical ward. Hollander, et al. (1965) described one case in which neurologists and psychiatrists debated over the diagnosis for a number of days before encephalitis became clearly manifest. Even with a detailed family history, personal history with adequate physical and laboratory examination it is often not possible to make a diagnosis. In such cases the patient must be cared for by an inter-disciplinary team to give him maximum medical and psychiatric support.

The reported mortality of untreated cases has varied from 50-70%. survivors have frequently suffered neurological impairment that times resulted in institutionalisation. Hence differential diagnosis become more important to begin treatment early.

To differentiate psychiatric from neurological disorders, Weinstain, l. (1953) and Weinstain et al. (1954) used amylobarbitone (Amytal) rview test. In catatonia amytal will usually result in the patients nning to talk and psychotic speech or delusions will be obvious. ncephalitis or other brain disease the neurological and communication cits will usually become more prominent.

To add to the problem of differential diagnosis of encephalitis is frequent occurence of psychological or social stress factors in the ent's life. These will appear as precipitating cause of functional hosis. Some recent work by Solomon et al. (1974) has strongly linked ss emotions and immunity. The immunosuppressive qualities of nocortical hormones, whose levels are responsible to stress may be rtant in the pathogenesis of infectious illness.

To avoid mistakes in diagnosing schizophrenia, Freighner et al.(1972) suggested that the clinician must consider the presenting picture, functioning and family history before making a diagnosis of zophrenia. Too quick reliance on a diagnosis of schizophrenia prevents her investigation of the aetiology of the psychotic state.

To investigate encephalitis, erythrocyte sedimentation rate, serology, n biopsy and culture, pharyngeal and rectal cultures, brain scan and phalogram may be helpful.

Treatment is good nursing care, reduction of cerebral oedema with corticosteroids and antibiotics to prevent bacterial infection. Anticonvulsants are helpful if epileptic fits occur.

One patient was diagnosed as louse-borne typhus after observing scalp lice eggs and a Weil-Felix reaction, Proteus OX-19 $\frac{1}{5120}$. She also had leucocytosis of 19,200/cu mm with lymphocytosis of 68 per cent. The film was normochronic normocytic. The extreme agitation improved after a course of parenteral chloramphenicol 500mg. 6 hourly for ten days. She was then referred to psychiatric ward where psychogenic factors were also found. She has recently been discharged home after a good improvement. This is a good example of physician and psychiatrist co-operation for the benefit of the patient with various aetiological factors which cannot be tackled by either alone.

Maguire and McNally (1972) found in Britain that one-third of the members of the family are affected by scalp lice. In the family of the patient under study, the baby she cared was found affected by lice but not typhus. They also noted that re-infection rate was 17.4% in girls and 13.8% in boys. Hence our patient will need regular follow up and treatment if necessary. Education and co-operation of mother of the family is most important in the success of treatment.

One of the patients who died in the series was diagnosed at post-mortem as having had left sided big subdural haematoma and a small one on the right side. Until he died, the diagnosis of alcoholic intoxication and hypoglycaemia was entertained. Absence of external head injury coupled with undetailed history of how he fell off a bus, and still

further absence of lateralising signs intensified the darkness to the provisional diagnosis of subdural baematoma. The lucid interval was celebrated as clinical improvement and the sudden deterioration of consciousness later during the day to death was like a mystery.

The problems in the clinical diagnosis have been noted to stem from several factors. Aetiology as trauma may be so mild and insignificant that the patient or relative may ignore it. To overcome this problem, trauma as aetiology should weigh greatly. Different authors like Kissock, et al. (1960) and Fogel et al. (1975) have estimated trauma to be as relevant as 50 - 71% of cases. So most neurosurgeons have learnt a lesson that if subdural haematoma is whispered, then no matter how unlikely it may be, they have no choice but to pursue it; if necessary to the stage of exploratory burr boles. In a recent review of 114 cases, Cameron (1978) found rewarding results with surgical procedures. Other problems include pathophysiology, the evolution of the haematoma and the wide variation in reaction of the hematoma, and the wide variation in reaction of the often aging brain to the presence of the haematoma, and the consequent lateral and vertical distortion and displacement of the cerebral hemispheres and midbrain. In addition, these factors vary considerably, not only from one patient to another, but at different stages and times in the same patient.

The frequent presenting symptoms are intellectual deterioration and change in personality. In contrast with other space-occupying lesions, symptoms and signs of raised intracranial pressure i.e. headache

and papilloedema are not present. This adds to the problems of diagnosis. The classical flunctuation of symptoms especially the level of consciousness and intellectual state occurs in one-third of patients. The recent finding of high output of tissue plasminogen activators from the outer membrane of subdural haematoma Ito et al. (1978), supports the view that fibrinolysis plays an important part in their persistence and progressive enlargement.

The essentials of investigation included good skull X-ray to study the pineal calcification which increases with age. If lateral and anter°-posterior views show it is central, this excludes the presence of clinically important haematoma since balanced bilateral lessions are rare. Our patient's left hematoma was larger than that of the right side.

Computerised exial tomography (CAT) if available has a high degree of occurancy in the diagnosis since it clearly shows the displacement of the hemisphere even when the actual haematoma may be "isodence" i.e. same density with brain tissue and therefore not visible. (Scott et al. 1977). The importance of exploratory burr holes has been mentioned above.

One patient in the series of patients was diagnosed as acute confusional state and anaemia. With a haemoglobin of 4.8g/dl, MCV 115 and left shifted leucocytosis and a bone marrow aspiration which showed only evidence of mild iron deficiency; the patient was transfused with two units of blood which elevated the haemoglobin to 9.5g/dl. Total dose imferon was given but because the mental symptoms had not cleared well

she was discharged through the psychiatric &clinic.

The point of discussion here is to point out the role of avitaminosis B_{12} as a cause of acute confusional state in the absence of characteristic changes in the blood and bone marrow. Mc Alpine (1929) confirmed that the cerebral manifestations of avitaminosis B_{12} sometimes preceded that of chacteristic changes in the blood by many months. Strachan and Henderson (1965) had one of their patients who had normal bone marrow with mild iron deficiency but serum B_{12} was abnormaly low 50uug/ml. There was no clinical evidence of spinal cord involvement. Serum B_{12} was not estimated in our patient . So it is difficult to exclude early B_{12} deficiency in view of macrocytosis in peripheral blood. So in the fields of psychiatry, general medicine and neurology it is clear that normal peripheral blood and marrow aspiration or biopsy appearances should not be regarded as excluding vitamin B_{12} deficiency.

The widely differing physical illnesses discussed above produced same symptoms and signs of mental confusional state.

Clouding of consciousness is described as the cardinal feature of acute confusional state in Europe. In this series of cases clouding of consciousness was observed in fourteen patients (66.7%). However seven patients (33.3%) were admitted in a semiconscious or unconscious ,state. None was admitted in clear consciousness. But Nadeem and Younis (1977) found that 19 of their 39 cases were admitted in clear consciousness.

Clouding of consciousness denoted the mildest stage of impairment of consciousness. It can deepen to stupor and coma. The main psychopathology is disturbance of attention. Incoming stimuli are not clearly focused in the mind. From poor concentration he develops distractibility.

German (1972) states that the patient is not in touch with his environment; he is frequently disorientated in time and place, may not recognise relatives and friends. Lipowski (1967a) points out that clouding of consciousness of a patient can be compared to that of a man absorbed in the reading of a book or in watching a film or in listening to music, only by an effort can he come to himself, an effort which is beyond the powers of a patient with clouded consciousness.

It is difficult or impossible to recognise from a single examination the mildest degrees of clouding of consciousness. The patient may show only slowness in responding or a tendency to forget the topic of discussion on the day before. There is also flunctuation of clouding from day to day and even over the course of the day. It is easily noted at the end of the day or when patient is tired.

From the foregoing views it is not difficult to understand why other workers may come across patients in clear consciousness. This depends on at what stage of the disease the patient presents to the expert psychiatrist or physician. It is advisable to reexamine the patient at different times of the day to detect the mildest degrees of clouded consciousness.

It was observed in this series of cases that those who presented in stuporous and unconscious stage, stayed in hospital longer than those admitted in clouded consciousness. This denotes the degree of cerebral injury during admission. Mild injury of brain cells requiring shorter time to heal than moderate to severe brain cell damage do. The younger patients recovered faster than older patients.

The majority of patients had agitated delirium. These included ten patients (47.6%) of those admitted in clouded consciousness and four patients (19.0%) of those admitted in either semiconscious or unconscious state and were followed up closely until their mental alertness started to improve. The total number of patients with agitated delirium was fourteen (66.6%).

In agitated delirium the patients consciousness is so reduced that the patient is disorientated for time and place. The attention field is narrowed. Visual or tactile hallucinations are mainly present. Patient moves about superficially, showing general restlessness, trying to leave his bed, to run away from his imaginary enemies or to fight them. Wolff and Curran (1935) found that the clinical picture was largely dependent on the previous personality of the patient, but these individual differences diminished as the degree of mental disorder increased in severity.

Four patients (19.1%) of those admitted in clouded consciousness had quiet delirium as well as three patients (14.3%) of those who were admitted in semiconscious or unconscious state. The total number of patients with quiet delirium was seven (33.4%). In quiet delirium the patient is mostly quiet. Symptoms like illusions and hallucination do not perplex him.

Mood variation was mainly anxiety with irritability and depression.

Irritability is noted in all organic psychoses, epilepsy, trauma and in advancing age. It is also obvious in Huntington's chorea.

The physiological basis was explained by Cannon (1928). He

emphasized the reflex character of the outburst of anger to render the organism more efficient in the struggle. Heart rate is increased, blood is redistributed, red blood cell mass is also increased. Bronchioles are dilated. Sugar is liberated from the liver. Andrenaline secretion is increased. All these adaptive physiological changes, are mediated through the autonomic nervous system. A part of brain near the thalamus is responsible. It is under the influence of higher centres. Irritability is likely to appear when function of these higher centres gets impaired by structural damage, toxins or physiological changes like hypoglycaemia. Like wise a tired and sleepy man is liable to irritability.

Irritability is also common in early alcoholic intoxication. People from high altitude are said to be irritable because of cerebral anoxaemia. Calcium metabolism or parathyroid gland may be involved since subjects with tetany sometimes suffer from irritability during or between attacks.

Organic states are very commonly the prime cause of depression. It may be the presenting symptom in general paralysis. Acute physical illness may precipatate an attack of depression.

Other causes of depression are septic illnesses, toxamias and influenza. Of the functional psychoses, schizophrenia and depressive psychoses are principle causes.

The patients in this series showed variability in thinking disturbance. Eight patients (38.1%) of the fourteen (66.7%) with clouded consciousness had slowed thinking. Six patients (28.5%) of the

group had slower thinking later. Overall with slowed thinking was fourteen patients (66.6%). The rest showed persecutory delusions perseverance, wandering and fragmentation of thoughts.

Thought processes show characteristic changes when consciousness is impaired. Consequently, thinking is disturbed frequently in delirium and other types of cerebral disease. The central disturbance is incoherence, meaning that the thought is disrupted with fragmentation, repetition and perseverance. Speech disturbance was mainly slowed speech in sixteen patients (76.2%). This was followed by irrelevant speech in three patients (14.3%).

In acute confusional state speech is slow and sparse. This is in agreement with our finding. Difficult questions are usually ignored. Slurring and perseverance occur. In severe cases there may be no more than incoherent murmuring. Still some patients show restlessness hyperactivity and noisily disturbing behaviour.

Orientation, memory, insight and interlligence were uniformly disturbed in all patients.

Memory disturbance in acute confusional state is the result of impairement of consciousness. There is disturbance of registration, retention and recall. Defective retention leads to difficulty with new learning; which is a sensitive clinical indicator in mild stages of acute confusional states. Consequently recent memories prove to be faulty while long term memories are left intact. Defective appreciation of flow of time leads to disorientation in time which is sometimes

regarded as the hallmark of acute confusional states. Disorientation in place and later still in person may follow with worsening of perceptual and cognitive disorganisation. Zangwill and Pertterson (1944) observed the attitude of patients who would maintain to be in hospital in one time but at the same time interpreting his surroundings and behaving as though he were at home in another part of the country. This attitude was shown by our patient, case no. 3. who answered a question put to him as to where he was; he said he was at home in Dar es Salaam and at the same time he claimed he was in Mawenzi hospital in Moshi. He was in fact lying puzzled in a hospital bed in Muhimbili Medical Centre - male medical ward.

The main disturbance of perception was visual and tactile hallucinations.

Hallucinations are perceptions in the absence of external stimuli. Illusions are misinterpretations of external stimuli. They are a result of sensory deprivation and mild brain cell degeneration and is more frequent when hearing is also impaired (Flynn 1962). Past experiences and psychodynamic factors influence the content of organic hallucinations (Penfiled and Perot 1963).

Many visual hallucinations result from cerebral dysfunction as in acute confusional states, dementias, hallucinogenic drugs and in dissociation of the reticular activating system, as in disturbance, of sleep or consciousness (Brain, 1958).

The visual hallucinations may be seen as light, geometrical patterns or colours. Fully formed hallucinations of scenes, people

and animals are experienced. Sometimes the hallucinated material may be grossly distorted as for example with Lilliputian hallucinations where objects and people appear to be minute in size. The patient fully accepts this reality and may react with fear and alarm but sometimes with interest or even amusement.

The duration of symptoms before admissions into hospital revealed marked delay in those in the fifth decade. It also followed that the same age group stayed longer in hospital than the other age groups who delayed less. The aged brain cells exposed to prolonged suffering must automatically take longer time to recover than the young less afflicted brains.

Early diagnosis, which can reduce the morbidity and mortality rates of acute confusional states, has been emphasized by Reding et al. (1964) for the reasons that it will allow reversible syndromes to be treated early. It will enable the physicians and nurses to remain alert to the possibility of unexpected behaviour on the part of the patient. It is also a valuable indicator of the course of the disease for which the patient is being treated. In particular early manifestations of an organic brain reaction have been shown to be one of the first indications of the impending death of the patient.

Seventeen patients (81%) were perfectly cured and discharged from hospital. Four patients (19%) died. Of those who died 75% were at the age above 40 years. Only 25% were in their second and third decades of life.

The death of two patients, could have been prevented by adequate fluid therapy and by early diagnosis of subdural haematoma with burr hole exploration to evacuate the haematoma respectively. Therefore 50% of mortality could have been prevented. This means the overall mortality of 19% in this series would have been reduced to 9.5%.

The Attitude of relatives towards acute confusional states.

& Case Report Nos. 2, 6, 8, 9 and 10. Case No. 2 is reported to have had a history of renal disease. She used to get local herbs from a herbalist due to her irregular menstrual periods for three months perior to admission in hospital with fever, vomiting and convulsion. She died of uraemia in hospital. Case No. 6 started with a traditional healer. On finding no improvement the relative sent her to several dispensaries before she was admitted in hospital. The diagnosis made was "Shetani" (evil spirit) in her. Her relative had another diagnosis of "cursed by her parents" because the patient knelt obediently asking her father for unknown faults. The hospital diagnosis was louse-borne typhus.

Case No. 8. The mother of the epileptic was attending traditional healers until she found no improvement. On getting treatment in hospital her son improved. She developed so much confidence in hospital services that she rushed her son to hospital within one hour of present illness. Hospital diagnosis was severe cerebral malaria. Case No. 9 had started with symptoms which suggested early malaria infection but did not go either to hospital or to a traditional healer. The severe attack started with fear, agitation and hallucinating by seeing snakes. When

she was brought to hospital relatives seemed unhappy when patient was given parenteral treatment - to learn that injections are feared for aggravating the illness if it is due to withchraft, evil spirits etc. Relatives hastened to request a discharge which was given on the following day. They took the patient to a traditional healer. My presence at this other period of outside hospital treatment was not entertained. The request made to mix both orthodox treatment and traditional treatment was turned down forthwith. Permission to see the patient was refused intelligently. Having stopped following up the patient, the mother of the patient sent for me. She had health problems herself. I could read from her face that she was happy. She called for my previous patient. She appeared with aabroad smile. She was happy too. She had shaved her head clean. Her upper arms were both tied separately with black piece of cloth. Local tatoos were found scattered on exposed surface of the body. Her mother thanked me for having dealth with cerebral malaria well. She said the orthodox doctor is not blamed for not curing the abnormal behaviour because it is due to supernatural forces outside his syllabus. Well and good; the patient was happy and settled despite of who treated what. The traditional healer and myself seemed to have worked as a team without mutual consultation!

Case No. 10 was a straight forward cerebral malaria. The husband entertained a belief of witchcraft and had called for his parents to come for a consultation on this other side of the picture. I encouraged him until his wife was cured. She was discharged on the sixth day after admission.

The above case examples show how belief in disease causation among the local population is so different from that of an orthodox doctor. They also show the big role a traditional healer has in the deliverance of health services especially in the rural areas where orthodox doctors and drugs are in short supply or non-existent. While discussing mental health programming in a developing country Swift (1972) emphasized the importance of understanding the community attitude towards the nature of mental illness and its implications in health education. The example he gave from Wazaramo, the coastal tribe of Tanzania, illustrated the social conflicts associated with the belief in witchcraft which has to be taken into consideration for the proper management of disturbed patients and the restoration of inter-personal relationships.

Smart, (1956) in Mirembe Mental Hospital in Dodoma, Tanzania observed that from infancy to the grave the African's life is crowded by supersititious customs and ritual. These superstitious beliefs become so ingrained that it is not uncommon to find even an educated African resorting to his tribal beliefs in moments of stress or illness. Insanity is often believed to be caused by witchcraft or possession by spirits. More recent views about African attitudes of disease have been expressed by Baasher (1975) when he observed that physical and mental disease may be attributed to a variety of causes including breach of a taboo or custom, disturbance in social relations, hostile ancestor, spirits, spirit possession, evil machination and intrusion of objects, evil eye, witchcraft, natural causes and affliction by God or

gods. This attitude does not exclude any socio-economic group. Oyebola, (1980), while studying some aspects of Yoruba traditional healers and their practice noted that people from all walks of life, students, traders, top professionals, nurses, doctors and clergymen consult traditional healers for diverse reasons. 80% of the herbalists were willing to co-operate with western trained doctors if this is suggested. But a few of them believed that orthodox doctors, know nothing about treating problems like jaundice, mental problems, convulsions, trouble with witches or wizards and severe headache; and that patients who went to hospital with such problems never got better or that such patients would be given injections which are contraindicated for such problems. This is consistent with the finding in Case No. 9 of this study.

The traditional healers are many, accessible and culturaly acceptable in every locality (Ademuwagun 1969) and their importance in the delivery of health care in Nigeria has been stressed by Maclean (1965); Lambo, (1969); Ademuwagun, (1969) and Harrison (1974). The Nigerian situation holds for the whole of Africa (WHO 1976). To meet its objective of "achieving health care coverage for all people", the WHO (1976) emphasized the need to develop African traditional medicine so that the truly beneficial activities are retained and the harmful and dangerous practices discarded. To respond to the WHO emphasis Tanzania opened its Research Centre for Traditional Medicine, on February 11, 1981. However, at present, there is insufficient information on various aspects of traditional medicine and its practitioners. So until the new Research Centre just started sheds more light

on the usefulness of local herbs, the public should be encouraged through mass media, to send their patients to hospital first before sending them to the crude herbalists. This will probably improve the prognosis of many diseases especially the acute confusional state where the traditional healer may concentrate on the mental aspect not knowing the supposed "shetani" or evil spirit is actually malaria which requires chloroquine course rather than drumming and jumping about around the patient.

Management of acutely disturbed patient in the General Ward.

Disturbed behaviour can be defined as behaviour that is disturbing to others, particularly the patients, doctors and nurses. This behaviour does not only affect the treatment of the underlying physical illness but also, by jeopardizing the smooth running of the ward, interferes with the care of the other patients. Management must therefore be rapid and effective, and involve the patient, the staff and the treatment setting.

The patient should be nursed in a side room by the same nurse or nurses if possible in order to limit the number of unfamiliar people he is faced by. He should be handled as gently as possible to avoid increasing any paranoid delusion he may have. During lucid intervals when the patient is most approachable, time should be spent talking to him to help relieve the considerable anxiety that confusionsl states carry.

Sedatives should only be given when necessary because they may mask important physical and mental signs. When they are required doses must be adequate and reduced to the individual clinical state with higher

doses being given when he is most likely to be disturbed for example at night. Oral route of administration is preferred to injections because the latter are difficult to give in an agitated paranoid patient. Furthermore they may add to his delusions of persecution and disturb the co-operative attitude of relatives believing in witchcraft or possession by evil spirits, where injections are thought to worsen the disease. However refusal to take oral drugs or when rapid action is desired, parenteral drugs should be given. If patient resists this then sufficient members of staff should be present to avoid any injury to patients or staff. Sometimes mere presence may induce willingness to accepting injections.

The drugs of choice are phenothiazines for example Largactil 100mg. orally in less disturbed patients or 100 - 200mg. intramuscularly or 50mg. intravenously 4 hourly gives control within 24 hours (Gammer, et al. 1978) or haloperidol 2.5mg. - 10mg. intramuscularly at intervals of 30 - 60 minutes where patients usually settle on 1 - 4 injectious (Donlon et al. 1979).

When clinical improvement occurs as indicated by the patient's behaviour and examination of the mental state, oral drugs should be reduced. In acute confusional states this occurs after a shorter period of time usually not more than 24 hours. Sedation can be stopped within 2 - 3 days.

On top of sedation each patient should get specific and symptomatic treatment depending on the underlying physical illness. To reduce cerebral oedema dexamethasone 5mg. intramuscularly twice daily was very useful in hastening the improvement of impaired consciousness. The same result was observed when a diagnostic lumbar puncture was performed.

DATE SHEET

ACUTE BRAIN DISEASE IN THE MEDICAL WARDS OF MUHIMBILI MEDICAL CENTRE

NAME SEX AGE

DATE OF ADMISSION: WARD: CASE NO:

ADDRESS: EDUCATIONAL LEVEL

OCCUPATION:

HISTORY OF ILLNESS (From Relatives Including their attitudes to the disease)

SOCIAL HISTORY

PAST MEDICAL HISTORY

Diabetes Mellitus: Cardiac failure

Epilepsy: Respiratory failure

Renal failure: Hepatic failure

EXAMINATION.

Drowsiness Delirium: Stupor

Coma Eyes Confusion:

Pupils Temperature: Fundi

EXAMINATION C.T.:

CVS: Pulse BP JVP

Murmurs

RESPIRATORY:

HEPATIC DISEASE.

RENAL DISEASE (Including Incontinence)

OTHER (Nutritional Status, Skin etc.)

MENTAL ST. CNS CT

SPEECH	TREMOR
NYSTAGMUS	DIPLOPIA
MINIGNISM	APRAXIA

NEUROLOGICAL EXAMINATION INCLUDE PLANTARS & REFLEXES.

INVESTIGATIONS

1. FBP & ESR
2. B/S
3. Urinalysis Sugar (Albumin) etc.
4. Stool Ova Parasites 5 Skull X-ray
   Bacteria
5. C X R
6. E.E.G.
7. Serum electrolytes
8. Serum Proteins
9. Lumbar Puncture
10. VDRL
11. Serology/Salmonella etc.
12. Serum drug level (alcohol etc.)
13. LFT

NOTES ON DIAGNOSIS + PROGRESS (e.g. time to recover etc.) OR DEATH.

PSYCHIATRIC EVALVATION.

NAME: .....

FILE NO: ..... DATE:.....

(Tick to the left of all relevant items)

State of Consciousness:

---- Clouded
---- Confused
---- Stuporous
---- Unconscious
---- Clear (Normal)

Orientation:

---- Lack of orientation
---- Time ---- Place ---- Person
---- Normal

Motor Disturbances (From obercation)

---- Overactivity
---- Aggressive
---- Destructive
---- Assaultive
---- Localized overactivity (Tics, tremors, stereotypy)
Specify ----------
---- Purposeless
---- Decreased activity
---- Extreme underactivity (Catatonia, marked withdrawal)
Specify ----------
---- Unco operative
---- Negativism
---- Automatic obedience
---- Compulsive movements
---- Echopraxia
---- Normal activity.

MENTAL HEALTH AND DISEASE IN AFRICA.

Mood or Affect:

——— Euphoric
——— Depressive
——— Inappropriate
——— Flat, Unresponsive
——— Anxious
    ——— Generalized
    ——— Phobic
——— Irritable
——— Lubile
——— Normal

Perception:

——— Illusions — Describe Hallucinations .....
——— Hallucinations — .....
    ——— Auditory — .....
    ——— Visual — .....
    ——— Tactile — .....
    ——— Olfactory — .....
    ——— Gustatory — .....
——— Normal — .....

Memory:

——— Amnesia
    ——— Recent
    ——— Remote
    ——— Selective
    ——— Confabulation
——— Déjàvu
——— Hyperamnesia
——— Can't evaluate
——— Normal

Intelligence: (Estimate)

——— Can't evaluate
——— Impaired due to psychiatric disturbance
——— Subnormal
——— Normal

Speech:

---- Excessive
---- Irrelevant
---- Incoherent
---- Slowed (in general)
---- No speech
--- Mute
--- Aphonia

--- Articulator disorder
---- Stammering
---- Lisping
---- Slurred
--- Neologism
--- Echolalia
--- Infantile speech
--- Normal

Thinking:

(a) Progression of Thought:

---- Flight of ideas
---- Fragmented
---- Slowed
---- Can't evaluate due to Mutism
---- Perseverative
---- Wandering
---- Normal

(b) Content of Thought:

---- Delusions Describe Delusions .....
---- -- Persecutory .....
---- -- Grandiose .....
-- Somatic .....
-- Nihilistic .....
Ideas of Reference .....
Depressive .....
-- Suicidal .....
---- Obsessive
---- Ideas of Ureality/Depersonalization
---- Phobias
---- Normal

Insight:

---- Normal ---- Absent ---- Partial
---- Can't evalute

Summary of Above Findings: .....

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Diagnostic Formulation: .....

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Admission Diagnosis:

(Possibilities) 1. .....

2. ....

3. .....

EXAMINING DOCTOR ----------

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