T I T L E

THE BRAIN DEATH SYNDROME IN THE INTENSIVE CARE UNIT, KENYATTA NATIONAL HOSPITAL

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D E C L A R A T I O N

THIS DISSERTATION IS MY ORIGINAL WORK AND HAS NOT BEEN PRESENTED FOR ANY DEGREE IN ANY OTHER UNIVERSITY

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SUMMARY

A retrospective study was carried out on thirty brain dead patients admitted to the intensive care unit (I.C.U.) at the Kenyatta National Hospital (K.N.H.) from January 1976 to December 1985.

There were 20 males (66.7%) and 10 females (33.3%), therefore a male:female ratio of 2:1.

The average age of these patients was 24.4 years and ranged from three to sixty years, with 50% of the patients below 20 years of age.

The thirty patients were admitted for a total of 155 days in the intensive care unit, with an average of 5.16 $\pm$ 0.25 days in the unit. One patient had a diagnosis of brain death with eclampsia on admission, and the diagnosis was confirmed before the heart stopped only after a few hours in the unit. One patient was in the unit for a total of 15 days.

The interval between the diagnosis of brain death to an isoelectric electrocardiogram (ECG) ranged from a few minutes to eight days after admission. About 50% of the patients got an isoelectric ECG

less than one day after brain death was diagnosed clinically. Of the remaining fifteen patients 50% had isoelectric ECGs in less than three days after clinical diagnosis.

Thirteen of the patients (43.4%) had head injury as a cause of brain death, four patients (13.4%) had brain tumours, three (10%) had cerebral anoxia following cardiac arrest; two patients (6.7%) had cerebral vascular accident, two had eclampsia, while one each had, aneurysm, encephalitis, asprin poisoning, tuberculous meningitis, craniovertebral anomaly, and one was admitted with coma of unknown origin which was later diagnosed as meningitis.

On admission, two patients, both with head injuries were intoxicated with alcohol, one patient was on phenobarbitone, and one had diazepam on admission as a stat dose to stop convulsions.

All patients required ventillatory support. Twenty six (86.7%) were put on the ventillator due to total apnoea, four (13.3%) due to inadequate ventillation, and no patient was on muscle relaxants.

On diagnosis of brain death all patients had non-reacting pupils and no motor response in the distribution of the cranial nerves. Vestibulo-occular reflex (caloric) was tested and found negative in twelve patients (40%). Oro-pharyngeal reflex (gag) was tested and found absent in eight patients (26.6%). Corneal reflex and atropine test each done and negative in four patients (13.3%). Dolls eyes were tested and found absent in six patients (20%). Apnoea test was not tested in any of the patients.

Confirmatory tests of brain death were carried out in fifteen patients (50%). The electroencephalogram (EEG) was done and found to be isoelectric in fourteen patients (46.7%), and cerebral blood flow was tested in one patient by carotid angiography and found to be absent.

The fourteen patients who had EEG done, six had it done twice, while in the other eight, it was done once.

In twenty four patients (80%) the diagnosis was made by the consultant, and in four, the diagnosis was made by the registrar. In two patients no information was available.

Eleven patients (36.7%) had at least one cardiac arrest in the unit before the clinical diagnosis of brain death was made. Three of these had a cardiac arrest twice, and each time successfully resuscitated. Where the duration of cardiac arrest was indicated it ranged from a few seconds to fifteen minutes.

In fifteen patients (50%) the treatment was unchanged after diagnosing brain death. Eleven of the remaining patients had all drugs withdrawn with only intravenous fluids remaining, and four had the number of drugs reduced, mainly antibiotics and ionotropic agents.

INTRODUCTION AND LITERATURE REVIEW

Brain death, its definition, diagnosis and management has generated a lot of interest and debate among those involved in the care of the critically ill patient, as well as the legal community and the general public, over the last two or three decades.

With the technological advancement in medicine it is now possible to maintain the circulatory, renal and respiratory functions of the human being thus making it difficult to define death in the classical manner - where a patient, who stops breathing and whose heart has stopped, is dead. It is therefore now not uncommon to find a deeply comatose, unarousable, unresponsive patient with the heart beating only because the patient's respiration is being supported, (1 - 5) what one author calls ventillating a corpse (6).

Brain death syndrome was formally described in 1959 by two french physicians - Mollaret and Goulon, who described coma depasse which means, beyond coma (6 - 8). In 1968 the report of the ad hoc committee of the Havard Medical School to examine the defination of brain death published its report (9) and gave the characteristics of irreversable coma.

In 1974 the Chief Medical Officer of Health and Social Security asked the Royal Colleges of the United Kingdom to consider the definition of brain death and its diagnosis (10),and a statement was issued by the honorary secretary in 1967 (11). A further report was issued in 1979 (12).

In Finland the National Board of Health published its diagnostic criteria for establishing brain death in 1971 (13).

In the United States the developments have been rapid since the first report in 1968 (9). A summary statement of the collaborative study was published in 1977 (14) a presidential commission was appointed and gave it's guidelines for the determination of death in 1981 (5).

The different criteria on the diagnosis of brain death all agree on:-

(a) a comatose patient on a ventillator with a known diagnosis likely to cause irremediable structural brain damage.

(b) Exclusion of reversible causes of coma.

(c) Absent brain stem reflexes.

What they differ on is the confirmatory tests. The British criteria insists that all that is needed is a thorough clinical diagnosis and a further laboratory testing is not necessary where diagnosis causing the brain damage is known (10,11,12). The Americans on the other hand recommend a confirmatory test. The electroencephalogram (EEG) was recommended by the Havard criteria (9) as well as the collaborative study (4) and is supported by the presidential commission (5) which also recommends documentation of cerebral circulatory failure. Pallis (16) is of the opinion that the arguement about the EEG is the concept, not the technicality, and the Americans practice it because they live in a "Litigous society with jurors who have a touchingly naive faith in the supremacy of machines". Sweet(17) an American agrees that, theirs is a litigous society. Several cases have arisen in the courts in U.S.A. relating to brain death (15, 18-20). The British are yet to go to court and their criteria has therefore not been tested legally, although the programme on television - panorama, in 1980 (21) showed the British public's reaction. Tomlin thinks this was due to misinformation (22) but may be the jury would equally be misinformed.

The ad hoc committee of the American electro-encephalographic society on EEG criteria for the determination of cerebral death (7), agreed that the EEG alone cannot be used to diagnose brain death. In their study of 1,665 cases, only in one third was EEG an important factor in the decision to discontinue resuscitation. The report emphasises on the technical problem encountered with the EEG. They recommend that, care must be taken in the definition of an isoelectric EEG. This they define as a linear EEG with no evidence of brain activity over 2 μV between two electrodes ten centimeters apart or more. Bolton argues in favour of the EEG and gives a case report with a clinical diagnosis of brain death while the EEG was not isoelectric. The patient recovered after ten days (23). He is however critisized by Jorgensen as having described a case where the clinical neurological examination was inadequate (24).

EEGs in drug induced comas are a hazard to the certification of brain death (25), and all criteria are in agreement that brain death is not to be diagnosed if intoxication has not been ruled out (3-5,7,9,14,16, 17,24-33). It is now widely accepted that EEG is not necessary for diagnosing brain death (11).

Those who argue favourably about EEG have in the wake of all these criticisms advocated other tests for confirmation of brain death. The American neurological association demands a cerebral blood flow technique in their criteria (4), as well as the presidential commission (5). The Swedish criteria is more technical demanding apart from apnoea and coma, an isoelectric EEG and non-filling of cerebral vasculature after two aortocranial injections of contrast 25 minutes apart (13).

Over the years several alternatives to EEG have been tried. Isotone angiography (9, 35), radionuclide cerebral imaging (36), and evoked potentials on EEG (1, 37, 38). Even cerebral spinal fluid cytology has been suggested (39). One author suggest the use of continous EEG monitoring (40). This (EEG) may be of use in the prediction of the outcome of fatal anoxic brain damage after cardiac resuscitation (41).

The world medical assembly meeting in Australia in 1968, agreed among other things that.."No single technological criterion is entirely satisfactory in the present state of medicine, nor can any one technological procedure be substituted for the overall judgement of the physician (7).

In Kenya death is still defined on the basis of cardiac arrest, and although brain death is diagnosed in the country's I.C.U.s the patients are still ventillated to asystole (42).

With the acceptance of brain death as a definition of death, one field that stands to gain is transplant surgery. Pallis rightly points out that when brain death was first described in 1959, the only means of immunosuppression that was available was total body irradiation, and therefore brain death was not created for the sake of organ transplant (6). Even if alternatives to organ transplants were to be found, brain dead patients would still be diagnosed in our intensive care units. Even by continued ventillation in these patients, the heart stops beating in about fourteen days if not earlier (8). Therefore if the diagnosis of brain death is made earlier, the organs will benefit a patient in need, before the organs deteriorate further by waiting, as will happen in patients ventillated to asystole.

The panorama programme on B.B.C. dwelled on this subject, arguing that brain death in Britain is made with such haste as to make it possible to remove organs from patients who are not "dead" (21).

This programme eroded the trust that the public had in transplant doctors, although Jennett et al thinks differently. They suggest that with the uneven reduction in donor supply after panorama, with some places increasing the numbers provided, strongly suggest that the main cause was increased reluctance of doctors to raise the issue of donation rather than that relatives were more often withholding consent (8).

Trauma is responsible for more deaths in all age groups under the age of forty five , and head injury is the single most important cause of deaths due to trauma (43). Head injuries accounted for half the renal donors in Britain, while intracranial haemorrhage accounted for one-third (8, 30). Head injuries accounted for one-third of brain-dead patients in Kenya (42).

The question of organ donation should never be raised before the diagnosis of brain death has been made (22, 44). The diagnosis should never be made in a hurry. None of the criteria is intended to cover emergency situations, all are formulated to deal with some period of continued cardiac activity with no apparent brain acivity. Therefore, time is

the safeguard against erroneous diagnosis (4,8,28,30). When the original diagnosis is known, and some urgency is needed, say in cases of organ donation, then a confirmatory test would be necessary (28). The Minnesota death criteria recommends clinical evidence of brain death for at least 12 hours (20).

Acute intoxication is the commonest cause of deep coma of fairly rapid onset still defying diagnosis twelve hours after admission to hospital. Alcohol is the commonest but only temporary cause of diagnostic problems. The neurological effects of acute intoxication lasts no more than six to eight hours (8,31,45) Alcohol complicates about one-third of cases of severe head injury when first admitted (28). Depression of the level of consciousness occurs at alcohol blood levels of around 200 mg per 100 mls. There is therefore a definite relationship between alcohol and head injury (45).

However, the interval between tests, once diagnosis has been made, is a matter of medical judgement and repetition time must be related to signs of improvement, stability or deterioration that present themselves (11).

One case of near drowning is presented, where the patient was submerged in water for approximately five minutes and received a slight head injury, before cardiopulmonary resuscitation was commenced. On admission to I.C.U. three hours later, an immediate EEG was "flat" in all leads, and neurological evaluation at the time strongly suggested cerebral herniation with brain death. It is interesting to note this as a diagnosis made in haste (three hours), in a patient who had submersion hypothermia and had earlier been on pancuronium, thiopentone and phenobarbitone (46).

In cases of "surviving" brain deaths, the initial diagnosis is never clear or certain exclusions are never made as to the cause of the coma. The British criteria has been described as too severe making it possible to continue ventillating a "dead" patient (22, 29, 47).

One should be careful in reaching this diagnosis, more so where a persistent vegetative state is concerned (6, 48,49). In some patients it may not be possible to elicit some of the brain stem reflexes, in which case the diagnosis of brain death should not be made (32, 50, 51).

Brain death syndrome has raised a lot of interest in the legal circles, mostly in the United States of America. New statutes have been formulated in different states, trying to incorporate brain death as death legally. (15, 17, 18, 19, 44). Veatch et al argues favourably for the need of statutory recognition of brain death (19, 52), but Byrne et al disagrees (53). Pallis suggests that, determination of death, should be a medical decision and there is no need for a statute (6). This may be true in Britain, but the Americans are in a dilemma. The Saikewicz case in U.S.A. set a very dangerous preceedent. The court decided that when patients are terminally ill beyond cure, and are incapable of consent, all decisions about the institution or termination of life prolonging measures, must be made by the court (20).

AIMS AND OBJECTIVES

This study was undertaken to:-

1. Evaluate the diagnosis of brain death at the Kenyatta National Hospital, intensive care unit.

2. Evaluate the management of brain dead patients in Kenyatta National Hospital Intensive care unit.

3. Make recommendations on new developments in this field.

MATERIALS AND METHODS

A retrospective study of 30 patients with a final diagnosis of brain death, in the intensive care unit at the Kenyatta National Hospital was studied.

The particulars of all those patients who died in the unit was obtained from the admission book in the intensive care unit and the files sought from the Records Department, where available, between the period January 1976 to December 1985.

The files obtained were then subjected to scrutiny and the information obtained from those who died with a final diagnosis of brain death recorded in a data collection form (see appendix). Only in those cases where brain death was mentioned in the files was such data collected, and no attempt was made in making the diagnosis otherwise.

The data collection form was prepared with both the British and American criteria of diagnosis of brain death in mind, and it sought to find out in each patient.

1. Existence of Apnoea

2. Coma

3. Findings on testing the brain stem reflexes

4. Exclusion of reversible causes.

5. Confirmatory tests where available.

The management of these patients as regards their original diagnosis as well as after diagnosis of brain death was reviewed.

RESULTS

The results of the study were as follows:-

The ages of the patients ranged from 3 to 60 years with an average of 24.4 years, 50% of the patients were under 20 years of age (TABLE 1 and FIGURE 1)

All 30 patients were admitted into the intensive care unit for a total of 155 days, with a range of few hours to 15 days, with an average of 5.16 ± 0.25 SEM days. This was the average from the time the patient was admitted to until the patients had isoelectric ECGs. None of the patients had the ventillator switched off. (TABLE 2)

One patient stayed a total of 15 days. This was a patient with primary diagnosis of cranio-vertebral anomaly admitted post-operatively. One patient was in the unit for less than 24 hours. Seven patients stayed for a total of three days, while four patients were admitted for six days and five patients for four days each. (TABLE 2, FIGURE 2).

The time interval from the diagnosis of brain death to the time when the patient got an isoelectric ECG ranged from a few minutes, to two patients "dying" after 8 days.

50% of the patients had isoelectric ECGs one day and less from diagnosis. None of the patients survived longer than eight days. (TABLE 3).

The commonest cause of brain death was head injury (43.4%), brain tumours (13.4%), post cardiac arrest (10%) and eclampsia (6.7%). One patient each had;communicating artery aneurysm, encephalitis,asprin poisoning, tuberculous meningitis, cranio-vertebral anomaly, and one patient was admitted with coma of unknown cause, later diagnosed as meningitis. Therefore more than 56% of the patients were diagnosed as having head injury and brain tumour (TABLE 4).

The drug history was reviewed in each patient as regards central nervous depressants. One patient had diazepam as a stat dose due to convulsions, and one 60 year old patient was on phenobarbitone to rest the brain which was later discontinued before diagnosis of brain death was made.

Two of the head injured patients were smelling of alcohol on admission. None of the four patients had blood taken for analysis of drug levels in blood (TABLE 5).

Twenty patients (66.7%) were males and ten (33.3%) were females, giving a male:female ratio of 2:1 (TABLE 6).

All patients were on ventillator on diagnosis of brain death. Twenty six patients (86.7%) were put on the ventillator because they were totally apnoeic. Four (13.3%) patients had inadequate spontaneous respiration. None of the patients were on muscle relaxants (TABLE 7).

In diagnosing brain death, all patients (30) had none-reacting, dilated pupils. None of the patients had any motor response along the cranial nerves. The caloric (Vestibulo-occular) reflex was tested in twelve patients (40%). Doll's eyes were tested in six patients (20%) and reported as negative. The oropharyngeal (gag) reflex was tested in eight patients (26.6%) and was negative. Four patients (13.3%) had the atropine test done, and was negative. The apnoea test was not carried out in any of the patients (Table 8)

After clinical diagnosis of brain death, no confirmatory

test was carried out in fifteen patients (50%). The electroencephalogram was carried out in fourteen patients (46.7%) and was isoelectric. In one patient carotid angiography done showed no cerebral blood flow. In most of the patients without an EEG recording, the test was requested but never done (TABLE 9).

The diagnosis was made in twenty four patients (80%) by a consultant in the unit, and by the anaesthetic registrar in four patients (13.3%). No information was available for two patients (TABLE 10).

Eleven patients suffered at least one cardiac arrest in the unit, before brain death was diagnosed, and were successfully resuscitated. Of the eleven, three had cardiac arrest twice and successfully resuscitated each time. The duration of cardiac arrest where available, ranged from a few seconds to fifteen minutes (TABLE 11).

After diagnosis ventillation was continued in all patients until the heart stopped beating. In fifteen patients (50%), medication was continued unchanged. In eleven patients (36.7%) all medication was stopped, leaving intravenous fluids. In four patients (13.3%), the number of drugs were reduced. No patient

had an increase in medication, either in number or dosage after the diagnosis.

None of the thirty patients ever recovered.

DISCUSSION

The patients presented in this study were relatively young, with 50% aged less than 20 years (TABLE 1, FIGURE 1). This is probably because our patient population is young.

The commonest diagnosis causing brain death is head injury (TABLE 3), accounting for about half of all cases. This compares well with observations by other authors (8, 30, 42).

Once brain death is diagnosed in any patient, organ transplantation may be considered. Ayim and Clark in 1979 (42), recommended the use of these patients for renal transplantation, since the surgeons in Kenya were pioneering into this field. To date this has not been done, although the number of kidney transplantation has gone up. Donors are usually relatives.

Before brain death is diagnosed, the condition causing it should be a definite diagnosis, known to cause irremediable structural brain damage. This means that, no treatment may be reasonably expected to change the condition (5,11,31,32). All the patients in

the study, except one fell into this category. This one patient was admitted into the unit with a diagnosis of coma of unknown origin. All the clinical features of brain death were present. It was only after a definite diagnosis of meningitis was made, was the patient declared brain dead.

The thirty patients were admitted into the unit, an average of 5.16 $\pm$ 0.25 days (TABLE 2, FIGURE 2). In 50% of the patients the heart stopped in less than one day after brain death was diagnosed. One patient died only a few hours after admission. This was a patient admitted with a diagnosis of eclampsia, query brain death, at twenty four weeks gestation. Two patients were in the unit for eight days each. None of the patients "lived" longer than eight days from diagnosis of brain death to when the heart stopped beating (TABLE 3). After diagnosis, all the patients had ventillatory support continued. This meant that, a bed, ventillator and staff, were unnecesarily occupied. In an eleven bed I.C.U., in a large hospital like ours, this is a luxury we can ill afford.

In these thirty patients the treatment was unchanged in 50% of the cases as far as medication was concerned.

In the remaining 50% of patients, antibiotics and ionotropic agents were stopped, while ventillation and intravenous fluids were continued (TABLE 12).

Once the diagnosis is made, the ventillator should be disconnected. The dilemma of when to switch off the ventillator, has been the subject of much public interest (11). By denying the patient - if assumed to be alive, either medication or ventillation, one is guilty of an error of omission, but since brain dead patients are truly dead, then apart from withdrawing medication, the ventillator should also be disconnected.

The confidence to consider withdrawing ventillation in Britain, was gained only gradually. There are still doctors who prefer to wait for spontaneous cardiac arrest and their views should be respected, as observed by Jennett et al in 1981 (28).

Marrku Kaste et al had twelve patients with the treatment unchanged after brain death was diagnosed, and in all patients the heart stopped beating after an average of twenty five hours (13). The American Neurological Association guaranteed cardiac arrest

twelve weeks after diagnosis of brain death (4). Jennett et al observed that there was abundant evidence that once brain death has been confirmed the heart always stops beating within fourteen days (8). The main reason for this is still unclear presumably it indicates a relationship between the cardiovascular system control and the brain stem that has yet to be clarified (4).

In diagnosing brain death in the thirty patients studied, not all brain stem reflexes were tested. For the diagnosis to be made these patients were:-

1. Having a definite diagnosis known to cause irremediable brain damage.
2. Comatose
3. Apnoeic
4. At least two brain stem reflexes absent

The apnoea test was not carried out in any of the patients, may be because ventillation was continued until the heart stopped. Testing for brain stem death should not be undertaken unless the cause of the coma has been established beyond doubt (32).

Corneal reflex testing requires firmer pressure than normally used in other patients (32).

While performing the caloric test (vestibulo-occular) the external auditory meatus is examined to exclude perforation of the eardrum, or impediments to the flow of water, such as wax. The external auditory meatus is irrigated with 20 - 50 mls of ice cold water. Normal response consists of deviation of both eyes to the irrigated side followed by nystagmus away from that side. If there is a large perforation and the patient still has a functioning brain stem, a fall in blood pressure and bradycardia may occur (32, 50).

Apnoea testing is the ultimate test of brain function. This involves showing no respiratory movements occuring during disconnection from the ventillator, after the partial pressure of carbon dioxide in arterial blood has risen to a level where stimulation of the respiratory control occurs. Testing involves delivering oxygen through a catheter down the trachea at 6 liters per minute after disconnection from ventillator, of ten minutes.

The partial pressure of arterial carbon dioxide should be at least 50 mmHg. Alternatively one may give 5% carbon dioxide in 95% oxygen for 5 minutes after disconnection. Care should be exercised in patients suffering from chronic obstructive airway disease. It is difficult to assess their respiratory function satisfactorily (5, 10, 11, 14, 32, 52,54)

Testing for motor functions should not present any problems, unless the patient is on muscle relaxants.

Where the diagnosis is in doubt, then there is no need for an EEG, though may be demonstration of lack of cerebral blood flow may be used. Alternative confirmatory tests include, isotope passage or clearance, cerebral arterial - venous oxygen difference, and auditory, visual and somatosensory evoked potentials. CSF cytology as well as enzymatic activity and lactic acid content of CSF (1,5,14,28,34 - 40).

In the 30 patients studied, EEG was done in fourteen patients (46.7%) and was isoelectric even after it was repeated in six patients once. In the other eight patients, (TABLE 9), EEG was not repeated. The Ad hoc committee of the Havard Medical School recommends

a repeat of all tests, including EEG, rain death
24 hours (9). Isotope angiography usin entials.
gamma camera can be used, especially when eclare
underlying disease is obscure, and when trai is
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circulatory deficit, thus confirming brain death (34,35
36).

Auditory brain stem responses have also been used.
This is a measure of the functioning of one of the
sensory pathways, traversing the brain stem, Auditory
brain stem responses are normal in drug induced coma (1).

None-filling of cerebral vasculature is one of the
requirements in the Swedish criteria (15).

a repeat of all tests, including EEG, at least after 24 hours (9). Isotope angiography using a bedside gamma camera can be used, especially when the underlying disease is obscure, and when transplantation is contemplated. This method is harmless, safe, convenient, and easily understood, but expensive. The inability to show intracranial blood flow by angiography, in a patient with appropriate clinical criteria is an accepted method of confirming the diagnosis of brain death. Radio-isotope bolus techniques using technetium 99 m pertechnetate, has been used, and recording time activity curves over the cranium and femoral artery simultaneously is an easy, safe and portable test that would detect cerebral circulatory deficit, thus confirming brain death (34,35 36).

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One author (37), describes a case of brain death clinically, with intact visual evoked potentials. He is of the opinion that, one should not declare such a patient brain dead even if the prognosis is hopeless. There may be interference during recording, from extracranial sources, such as the muscles (33, 38).

These second generation attempts at instrumental diagnosis of brain death, are quite technical and expensive (33). In a developing country like Kenya a simple, thorough clinical criteria is adequate. Even a simple EEG at present cannot be done due to some problems.

Five cases of brain dead patients has been described, where cerebro spinal fluid obtained by lumbar puncture was studied, and was found to contain , purkinje cells, polymorphonuclear leucocytes, massive pleocytosis and macrophages (39). Could this be a histological way of diagnosing brain death?

Apart from EEG and carotid angiography, none of the other techniques have been used in our hospital (TABLE 9).

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Apart from EEG and carotid angiography, none of the other techniques have been used in our hospital (TABLE 9).

In 80% of the patients in this study, the diagnosis was made by the consultant, usually the anaesthetist. In four patients it was done by the registrar, while in two patients there was no indication as to who made the diagnosis (TABLE 10). Several criteria suggest that, two doctors make the diagnosis, and where organ transplantation is contemplated, none of the transplant doctors should be involved in making the diagnosis. The ad hoc committee in 1968 suggested that, the physician in charge of the patient consult with one or more other physicians, directly involved in the case before the patient is declared brain dead (9). The British criteria suggests a consultant in charge of the case, and one other doctor, or, in the absence of a consultant, his deputy who should have been registered for five years or more, and who should have had adequate previous experience in the care of such patients, and one more doctor(10,11). The statement issued by the honorary secretary of the conference of medical Royal Colleges and their faculties, in 1976, suggests that experienced clinicans in I.C.U.s, acute medical wards and accidents and emergency departments should not normally require specialist advice. Only when the original diagnosis is in doubt

is it necessary to consult with a neurologist or neurosurgeon (11). This last suggestion should be quite adequate for a developing country, where staffing by specialists is only limited to some centres.

Eleven patients, suffered at least one cardiac arrest of variable duration before the diagnosis of brain death was made. This could have been in itself responsible for irremedial structural brain damage. (TABLE 11). The picture is more confusing in head injuries. Cerebral anoxia results in cerebral oedema, which in turn increases intracranial pressure, thus compromising the cerebral blood flow due to vasomotor paralysis with consequent loss of autoregulation. If intracranial pressure rises to the level of systemic pressure, cerebral death takes place (43). The EEG can predict death in serious brain damage (26).

CONCLUSIONS AND SUGGESTIONS

Patients with brain death syndrome can be maintained on artificial support in our I.C.U for a long time, just as in other countries. The longest one in this study, was in the unit for fifteen days. They are expensive to manage and in many cases, their families are also anxious about the outcome.

A developing country like ours cannot afford the colossal sum of money needed to manage them. Some of them may be suitable organ donors for transplants, which have already been started here.

It is therefore, suggested that a uniform policy on their diagnosis and management be adopted in this country. The British criteria, as outlined by the two Royal Colleges was adopted in this study and there were no flaws. What is therefore probably required is legal and administrative backing in the termination of life supporting mechanisms in future.

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TABLE I

AGE DISTRIBUTION

AGE IN YEARS	NUMBER OF PATIENTS
1 - 5	5
6 - 10	4
11 - 15	1
16 - 20	5
21 - 25	2
26 - 30	4
31 - 35	2
36 - 40	1
41 - 45	2
46 - 50	1
51 - 55	2
56 - 60	1
60+	0
TOTAL	30

TABLE 2

TOTAL STAY IN THE UNIT

DAYS	NUMBER OF PATIENTS	TOTAL DAYS
< 1	1	1
1	3	3
2	0	0
3	7	21
4	5	20
5	2	10
6	4	24
7	2	14
8	2	16
9	2	18
10	0	0
11	0	0
12	0	0
13	1	13
14	0	0
15	1	15
>15	0	0
TOTAL	30	155

TABLE 3

INTERVAL BETWEEN DIAGNOSIS OF BRAIN DEATH AND ISOELECTRIC ECG

INTERVAL	NUMBER OF PATIENTS
ON ADMISSION	1
20 MINUTES	1
2 HOURS	1
12 HOURS	1
1 DAY	11
2 DAYS	2
3 DAYS	6
4 DAYS	3
5 DAYS	1
6 DAYS	1
7 DAYS	0
8 DAYS	2
8 DAYS	0
TOTAL	30

TABLE 4

DIAGNOSIS ON ADMISSION

CAUSES OF BRAIN DEATH

	CAUSE	NUMBER OF PATIENTS	PERCENTAGE
1.	HEAD INJURY	13	43.4
2.	BRAIN TUMOUR	4	13.4
3.	CARDIAC ARREST	3	10.0
4.	CEREBRAL VASCULAR ACCIDENT (CVA)	2	6.7
5.	ECLAMPSIA	2	6.7
6.	POSTERIOR COMMUNICATING ANEURYSM (POST OPERATIVE)	1	3.3
7.	ENCEPHALITIS	1	3.3
8.	ASPRIN POISONING	1	3.3
9.	TUBERCULOUS MENINGITIS	1	3.3
10.	CRANIOVETERBRAL ANOMALY	1	3.3
11.	PYOGENIC MENINGITIS	1	3.3
	TOTAL	30	100%

TABLE 5

PATIENTS ON CENTRAL NERVOUS DEPRESSANTS ON ADMISSION

	ALCOHOL	NUMBER OF PATIENTS
1.	ALCOHOL	2
2.	PHENOBARBITONE	1
3.	DIAZEPAM	1
4.	NARCOTICS	0
5.	HYPNOTICS	0
	TOTAL	4

TABLE 16

SEX DISTRIBUTION

SEX	NUMBER OF PATIENTS	PERCENTAGE
MALES	20	66.7
FEMALES	10	33.3
TOTAL	30	100.0

TABLE 16

SEX DISTRIBUTION

SEX	NUMBER OF PATIENTS	PERCENTAGE
MALES	20	66.7
FEMALES	10	33.3
TOTAL	30	100.0

TABLE 7

VENTILLATOR USE

REASON	NUMBER OF PATIENTS	PERCENTAGE
TOTAL APNOEA	26	86.7
INADEQUATE VENTILLATION	4	13.3
MUSCLE RELAXANTS USED	0	0
TOTAL	30	100.0

TABLE 8

BRAIN STEM REFLEXES TESTED

	REFLEX	NUMBER OF PATIENTS	PERCENTAGE
1.	UNREACTING PUPILS	30	100
2.	CORNEAL REFLEX	4	13.3
3.	VESTIBULO-OCCULAR REFLEX (CALORIC)	12	40
4.	ORO-PHARYNGEAL REFLEX (GAG)	8	26.6
5.	MOTOR RESPONSE	30	100
6.	APNOEA TEST	0	0
7.	DOLL'S EYES	6	20
8.	ATROPINE TEST	4	13.3

TABLE 9

CONFIRMATORY TESTS DONE

	TEST	NUMBER OF PATIENTS	PERCENTAGE
1.	NONE DONE	15	50
2.	ISOELECTRIC EEG	14	46.7
3.	CEREBRAL BLOOD FLOW	1	3.3
	TOTAL	30	100.0

TABLE 10

CADRE DIAGNOSING BRAIN DEATH

CADRE	NUMBER DIAGNOSED	PERCENTAGE
CONSULTANT	24	80%
REGISTRAR	4	13.3%
NOT INDICATED	2	6.7%
TOTAL	30	100.0%

TABLE 11

11 PATIENTS WHO SUFFERED CARDIAC ARREST BEFORE DIAGNOSIS OF BRAIN DEATH, AND DURATION OF ARREST

PATIENT NO.	NUMBER OF ARRESTS	DURATION
1	1	?
2	1	4 minutes
3	1	5 minutes
4	2	?/5 minutes
5	1	15 minutes
6	1	?
7	1	2 minutes
8	1	Seconds in theatre
9	2	?/?
10	1	5 minutes
11	2	10/10 minutes

TABLE 12

TREATMENT AFTER DIAGNOSIS OF BRAIN DEATH

TREATMENT	NUMBER OF PATIENTS	PERCENTAGE
A. VENTILLATION		
1. CONTINUED	30	100%
2. STOPPED	0	0
TOTAL	30	100%
B. DRUGS		
1. UNCHANGED	15	50%
2. INTRAVENOUS FLUIDS ONLY	11	36.7%
3. REDUCED	4	13.3%
4. INCREASED	0	0
TOTAL	30	100%

FIGURE I

AGE DISTRIBUTION

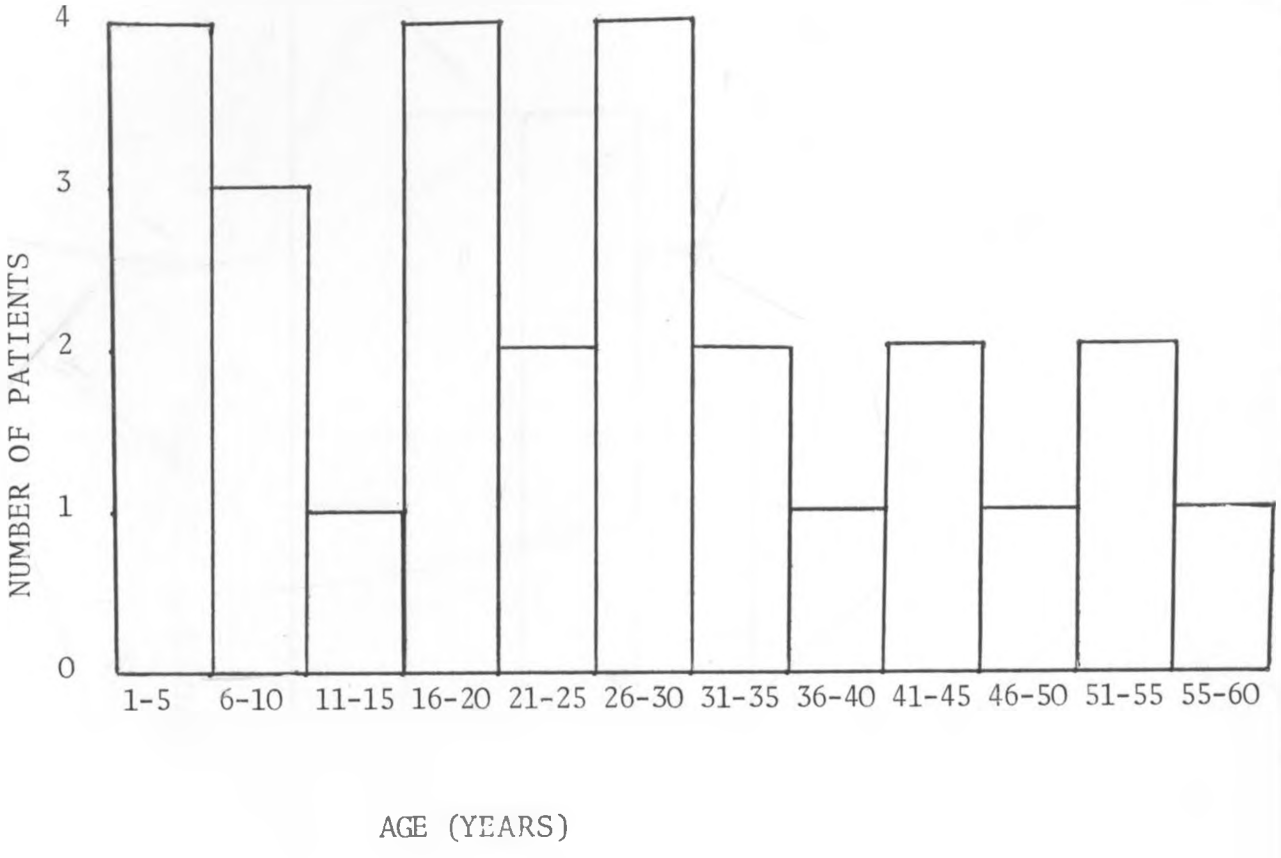
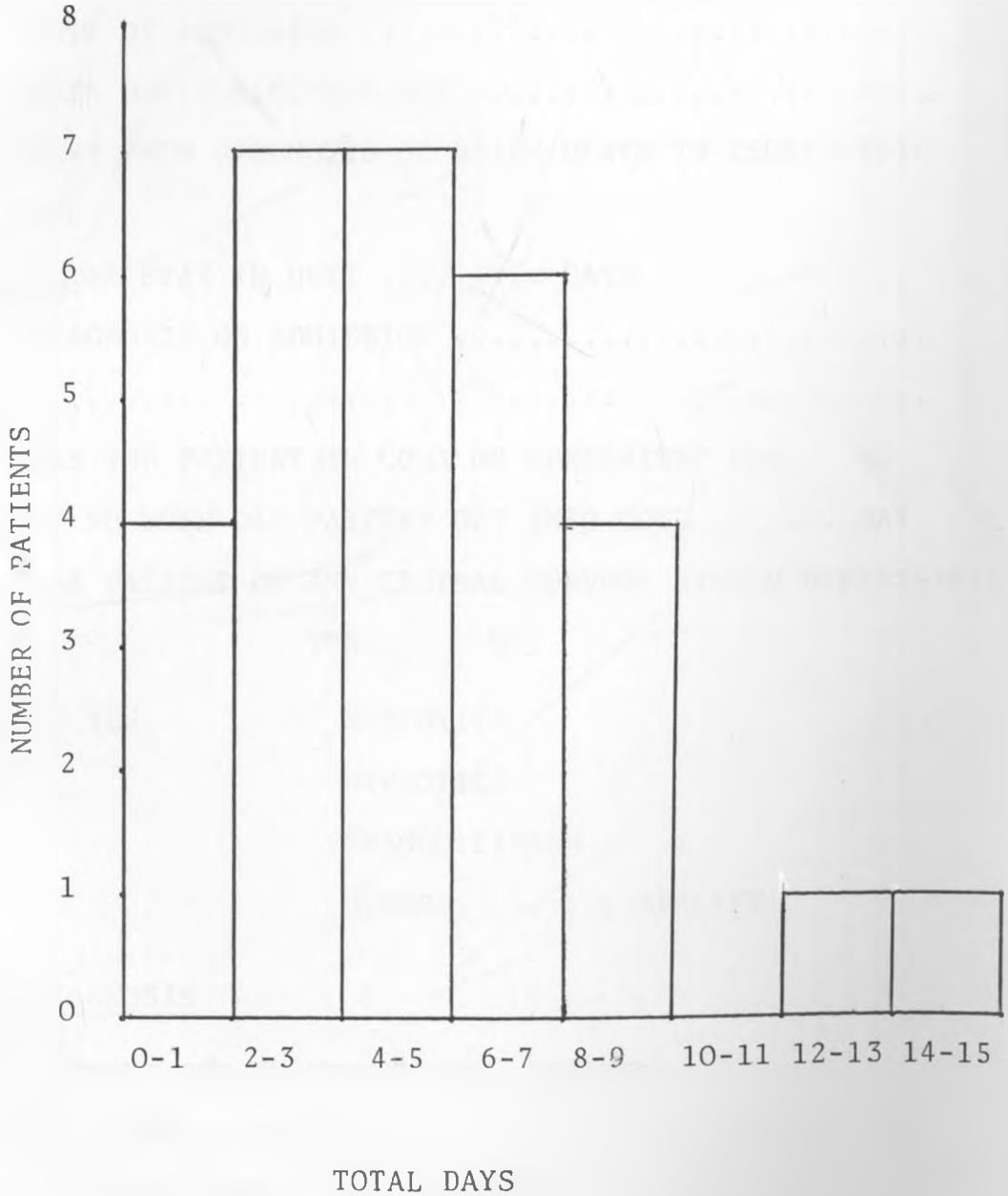


FIGURE 2

STAY IN I.C.U.



APPENDIX I

NAME ..... AGE .....

SEX .....

OCCUPATION .....

DATE OF ADMISSION .....

DATE OF ISOELECTRIC ECG .....

DAYS FROM DIAGNOSIS OF BRAIN DEATH TO ISOELECTRIC ECG

TOTAL STAY IN UNIT ..... DAYS.

DIAGNOSIS ON ADMISSION .....

.....

WAS THE PATIENT IN COMA ON ADMISSION? YES NO

IF NO WHEN DID PATIENT GET INTO COMA .....DAY

WAS PATIENT ON ANY CENTRAL NERVOUS SYSTEM DEPRESSANTS

YES NO.

IF YES: NARCOTICS

HYPNOTICS

TRANQUILISERS

OTHERS.....SPECIFY

DIAGNOSIS

PATIENT PUT ON VENTILLATOR BECAUSE:-

a) TOTAL APNOEA

b) INADEQUATE VENTILLATION

c) MUSCLE RELAXANTS USED.

APPENDIX I

NAME ..... AGE .....

SEX .....

OCCUPATION .....

DATE OF ADMISSION .....

DATE OF ISOELECTRIC ECG .....

DAYS FROM DIAGNOSIS OF BRAIN DEATH TO ISOELECTRIC ECG

TOTAL STAY IN UNIT ..... DAYS.

DIAGNOSIS ON ADMISSION .....

.....

WAS THE PATIENT IN COMA ON ADMISSION? YES NO

IF NO WHEN DID PATIENT GET INTO COMA .....DAY

WAS PATIENT ON ANY CENTRAL NERVOUS SYSTEM DEPRESSANTS

YES NO.

IF YES: NARCOTICS

HYPNOTICS

TRANQUILISERS

OTHERS.....SPECIFY

DIAGNOSIS

PATIENT PUT ON VENTILLATOR BECAUSE:-

a) TOTAL APNOEA

b) INADEQUATE VENTILLATION

c) MUSCLE RELAXANTS USED.

APNOEA TESTING YES NO

IF YES: WITH OXYGEN OR OXYGEN + CO_2

DURATION .....MINUTES

BRAIN STEM REFLEXES

a) PUPILS FIXED IN DIAMETER AND UNREACTING

b) CORNEAL REFLEX

c) VESTIBULO - OCCULAR REFLEX

d) OROPHARYNGEAL REFLEX

e) MOTOR RESPONSE

f) EEG

KEY N/A INFORMATION NOT AVAILABLE

FINAL DIAGNOSIS MADE BY a) CONSULTANT

b) REGISTRAR

c) OTHER (SPECIFY)

HAS PATIENT HAD ANY CARDIAC ARREST IN UNIT BEFORE DIAGNOSIS OF BRAIN DEATH: YES NO

IF YES, HOW MANY AND DURATION

1. .....

2. ..... DURATION

3. .....

MEDICATION AT THE TIME OF DIAGNOSIS OF BRAIN DEATH

.....

MEDICATION AFTER DIAGNOSIS .....

.....