

Letters to the editor

THERAPEUTIC INTERVENTION SCORING SYSTEM IN MEDICAL INTENSIVE CARE

I read with interest an article published in your journal (Med. J. Malaysia Vol. 44, no. 2, June 1989) entitled "Therapeutic Intervention Scoring System in Medical Intensive Care" (pp. 134-139).

In this article on p. 139 under "Death Definition" it is stated ".....a clinical neurological assessment followed by an EEG is carried out to identify brain death...." I wish to point out that "Brain Death" is accepted today as having occurred when there is "irreversible cessation of brain function including brain stem function" (I am quoting from decisions taken at the recently completed 5th World Congress on Intensive and Critical Care Medicine (W.C.I.C.C.) held in Kyoto, Japan, 3-8.9.89 where I was invited to speak on "Brain Death - Problems in a Developing Country").

At this Congress the World Body has concluded that to certify "Brain Death" the requisites are:-

1. Demonstration of the absence of brain stem reflexes by clinical tests - absolute requirement.
2. Corroborative Tests (not absolutely required).
 - a. Absence of cerebral blood flow using radio-isotopes (Xenon or Technitium pertechnetate.)
 - b. cerebral angiography
 - c. computerized tomography scanning.
 - d. evoked potentials.
 - e. echoencephalography.
 - f. EEG

The recommended corroborative test is (a) absence of cerebral blood flow because of its convenience and relative accuracy of information provided.

EEG is not recommended today (even in USA most centres have excluded the EEG as an absolute requirement). Even as a corroborative test the EEG must be repeated within 6-24 hours to have any meaning.

This letter is written to make clear the current up-to-date status of Brain Death (5th W.C.I.C.C. 3-8.9.89) and to clarify that EEG is not an accepted absolute criterion of Brain Death.

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We wish to thank Dr. Delilkan for the interest shown in the study which was reported. We would like to stress that clinical assessment formed the prime criteria for death definition, not EEG or other tests.

Corroborative tests mentioned by Dr. Delilkan included both radio-isotope study to demonstration cerebral blood flow and EEG. The list of the corroborative tests is 'not absolutely required'

yet isotope study is 'recommended'. Though the World Body may prefer the isotope study and the medical profession in U.S.A. has given up EEG, uniformity of procedure to define brain death has yet to be reached. If the medical profession in Malaysia decides to accept what was said in Kyoto in 1989, we would definitely apply those criteria to define brain death, at least to be on the right side of legal issue.

Till then we would like to assure Dr. Delilkan that clinical assessment will be used to define brain death and include isotope study as one of the confirmation studies if need arises.

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FULMINANT HEPATITIS DUE TO INFECTIOUS MONONUCLEOSIS

We report an unusual and fatal complication of infectious mononucleosis.

A five year old Chinese boy presented with a three day history of fever, anorexia and vomiting. His younger brother was hospitalised at that time with the diagnosis of infectious mononucleosis.

Clinical examination revealed an alert child with a tinge of jaundice. An erythematous rash was noted over the anterior abdominal wall. Multiple firm, mobile, discrete and non-tender lymph nodes, about 1 cm in diameter, were palpable in the cervical and inguinal regions. The throat was normal. The liver was enlarged 4 cm below the right costal margin in the mid-clavicular line and was soft and non-tender. The spleen was enlarged 4 cm below the left costal margin. A clinical diagnosis of infectious mononucleosis with hepatitis was made.

The relevant investigations revealed raised liver enzymes-aspartate transferase of 370 IU/L and alanine transferase of 270 IU/L. Blood monospot test was negative. Serology for hepatitis B surface antigen, cytomegalovirus, toxoplasmosis, rubella, herpes simplex and syphilis was negative. IgM antibody for Epstein Barr virus was positive. The general condition worsened gradually. Terminally he developed hypokalemia, hyponatremia, melena, haemetemesis, septicemia due to *E. coli* and died four weeks after admission. Post mortem liver biopsy revealed hepatic necrosis mainly in the perivenular area and minimal mononuclear cell infiltrate in the portal tracts.

Fulminant hepatitis is a rare, but recognised complication of infectious mononucleosis.¹ This possibility must be kept in mind in a patient with acute progressive jaundice.

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Reference

1. Chang M.Y., Campbell W.G. Fatal infectious mononucleosis associated with liver necrosis and herpes-like virus particles. *Arch. Pathol* 1975; 99: 185-91.