

**BRAIN DAMAGE
and
BRAIN DEATH**

Essay

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Master Degree in ANAESTHESIOLOGY.

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CONTENTS

✧ INTRODUCTION.....	i	-ii
✧ AETIOLOGY AND PATHOPHYSIOLOGY OF BRAIN DAMAGE.....	1	-57
- Definitions.....	1	-2
- Mechanisms of consciousness and unconsciousness.....	2	-3
- Aetiology of brain damage and brain death.....	3	-5
- Physiological aspects of brain oxygenation & circulation....	5	-9
- Categories of brain hypoxia.....	9	-14
- Pathophysiology of brain oedema following brain ischaemia..	15	-23
- Ionic changes in brain ischaemia.....	24	-35
- Induced hypotension and brain ischaemia.....	36	-45
- Head injury and brain ischaemia.....	46	-57
✧ DIAGNOSIS AND PREDICTIVE CRITERIA FOR OUTCOME AFTER BRAIN DAMAGE....	58	-91
- Introduction.....	58	-61
- Diagnosis of brain stem death.....	61	-74
- Pitfalls in diagnosis of brain death.....	74	-77
- Safeguards for the patient.....	77	-78
- Corroborative testing with brain death.....	79	-87
- Predictive criteria for outcome after brain damage.....	87	-91
✧ PREVENTION AND TREATMENT OF BRAIN DAMAGE.....	92	-133
- Prevention of hyperglycaemia.....	92	-95
- Prevention of seizures and status epilepticus.....	95	-96
- Prevention of increased intracranial pressure.....	96	-101
- Prevention of brain damage during induced hypotension.....	101	-107
- Treatment of brain damage.....	108	-133
◦ Calcium entry blockers.....	109	-114
◦ Anticonvulsants and drugs depressing cerebral metabolism.....	114	-128
◦ Drugs promoting microcirculation post-ischaemia.....	128	-129
◦ Prostaglandins and drugs affecting prostaglandin synthesis.....	129	-130
◦ Free radical scavengers.....	130	
◦ Miscellaneous drugs.....	131	-133
✧ SUMMARY.....	134	-138
✧ REFERENCES.....	139	-164
✧ ARABIC SUMMARY.		

2

INTRODUCTION

INTRODUCTION

People have been alarmed for centuries at the prospect of being declared dead when they were still living. There was generalized anxiety about the subject 145 years ago, after Edgar Allan Poe had published various stories, such as *Premature Burial*, in which people had been interred alive. This was confronted with in the past when death was defined as, absence of pulse and respiration which produced prompt death of the brain. (Pallis, 1982)

Nowadays, it is possible to sustain brain function in the absence of spontaneous respiration and cardiac function, so that death can no more be equated with the loss of these two vital functions. On the other hand, a dead brain in a body whose heart is still beating is one of the more macabre products of modern technology. During the past 35 years techniques have developed that can artificially maintain ventilation, circulation, and elimination of waste products of metabolism in the body whose brain has irreversibly ceased to function. (Posner, 1985)

Anaesthesiologists were among the first to confront the dilemma of whether a comatose patient whose brain is

7

dead and who is receiving respiratory and/or circulatory support is alive or dead.

Brain death is a term used to describe a condition in which there is complete cessation of brain function and in which cessation of all organs is imminent and inevitable. (Veith, et al., 1977)

Our essay has the following objectives, first, to emphasize that it is legitimate to equate brain death with death (which is now widely accepted in medical and legal circles throughout the world); second, to suggest that the necessary and sufficient component of brain death is death of the brain stem which can be reliably be diagnosed at the bed side (Pallic, 1982); third, mentioning the causes and pathophysiological events occurring during brain damage and brain death; and lastly, mentioning means of protecting the brain against damage and some drugs that protect and treat brain insult that may lead to brain damage and eventually brain death.

These objectives would lessen human distress, lead to more rational use of our limited intensive care facilities, as ventilating corpses (brain-dead patients) is distressing to relatives, bad for morale of attending doctors and nursing staff, beside the cost-effectiveness equation.

2

**AETIOLOGY
AND PATHOPHYSIOLOGY
OF BRAIN DAMAGE**

AETIOLOGY AND PATHOPHYSIOLOGY OF BRAIN DAMAGE

~DEFINITIONS:-

Consciousness include two independent but separate functions : wakefulness and psychologically recognizable mental activity. (Posner, 1985)

Coma, is a state of complete mental unresponsiveness with eyes closed and no evidence of psychologically or physiologically appropriate responses to stimulation.

Between these antipodes lie a series of abnormal states of mentation and arousal that reflect the effects of different degrees and acuteness of brain dysfunction damage.

Drowsiness describe states of impaired alertness or wakefulness wherein patients continue to respond to verbal stimuli.

Stupor is a state wherein subjects arouse when vigorously stimulated but immediately sink back to unresponsiveness as soon as external stimuli are withdrawn. (Posner, 1985)

The vegetative state describes a usually chronic or semichronic condition wherein patients with severe forms of brain damage, sleep and awaken but have no recognizable psychological functions. Brain stem and autonomic functions are retained. (Posner, 1985)

~MECHANISMS OF CONSCIOUSNESS AND UNCONSCIOUSNESS

The physiologic basis of consciousness depends on close interaction between the intact cerebral hemispheres and activating mechanisms located in the central gray matter of the upper brain stem. The cerebral hemisphere contribute the substrate for most of the specific psychologic components, including language, memory, intellect, and learened responses to sensory stimuli. However, in order for the cerebrum to function and to integrate its component psychologic activities, the hemispheres must be aroused or activated by structures that originate in the thalamus, hypothalamus, mid brain, and tegmentum of upper pons. An important component of this arousal mechanism is located within what Magoun, Morruzzi, and their colleagues called the ascending reticular activating system; other brain stem systems lying along the deep central gray matter core of the brain stem also influence cerebral cortical activity and the state of consciousness. In addition, conscious behavior is

heavily influenced by the activity of intra- and inter-hemispheric interconnecting neural pathways. Lesions of the cerebral cortex alter the integrative aspects of consciousness, and the sudden total loss of cortex or its connections to the deeper nuclei causes several days or weeks of coma even if the brain stem remains intact. (Posner, 1985)

~AETIOLOGY OF BRAIN DAMAGE AND BRAIN DEATH:-

Anaesthetists are involved regularly in management of patients who have suffered brain ischaemia and consequent brain damage and death.

Head injury is the commonest cause of brain damage and death, respiratory failure may occur soon after the patient reaches hospital; alcohol, and hypotension or hypoxia from associated injuries, may be complicating factors. Also, respiratory failure may occur in patients who have been recovering from the impact injury but have developed secondary complications such as intracranial haematoma or brain swelling. (Chou, 1981)

Intracranial haemorrhage is also one of the causes, as there may be respiratory failure soon after the initial

ictus; or be a secondary event following rupture of an aneurysm a second time after recovery from the initial bleed. (Jennet, et al., 1981)

While in subarachnoid haemorrhage, temporary apnoea is common immediately and may recover spontaneously in a minute or so, but in a few patients recovery occurs after one hour or more of apnoea, if artificial ventilation has been maintained during this period.

Brain damage or death may also occur when treatment for other intracranial conditions such as, brain abscess, tumor, encephalitis or meningitis proves fruitless. (Prior, 1980)

Other causes which are encountered within operative theatres, are cardiac arrest or profound systemic hypotension and in whom heart action and circulation have been restored, but too late to save the brain. According to the duration and intensity of hypoxia these patients may be brain dead, or may be left in the vegetative state.

The other causes are the anaesthetic use of induced hypotension, deliberate circulatory arrest in cardiac surgery and local cerebral circulatory arrest in neurosurgery, all

of which may produce brain insults up to the extent of brain damage and brain death. (Pallic, 1982)

~PHYSIOLOGICAL ASPECTS OF BRAIN OXYGENATION & CIRCULATION:-

Whatever the circumstances, irreversible hypoxic brain damage can occur whenever there is insufficient oxygen in blood or a reduced blood flow to the brain, or both. Brain oxygenation is dependent on respiration and the circulation, certain aspects of which should be considered before the pathology of hypoxic brain damage is described. (Graham, 1985)

*Respiration :-

The oxygen content of blood decreases from $19.6 \text{ ml}^{-1} \text{ dl}^{-1}$ to $12.9 \text{ ml}^{-1} \text{ dl}^{-1}$ while passing through the brain and carbon dioxide content increases from $48.2 \text{ ml}^{-1} \text{ dl}^{-1}$ in arterial blood to $54.8 \text{ ml}^{-1} \text{ dl}^{-1}$ in the internal jugular vein. Thus, the respiratory quotient to the brain is almost unity, implying that glucose is the most likely single source of energy by oxidation. In normal adult resting subjects, the cerebral metabolic rate for oxygen is $3.2-3.8 \text{ ml O}_2 \text{ min}^{-1} / 100 \text{ g}$. (Graham, 1985)

Brierley and colleagues, (1980); found that the arterial PO₂ could be reduced to 4.7 KPa without affecting either brain metabolism or function. It was not until arterial PO₂ had been reduced to 2.8-3.2 KPa for at least 8 min that the EEG became isoelectric and irreversible brain damage ensued.

***Circulation :-**

The brain of the normal adult receives approximately 15 % of the cardiac output representing a mean cerebral flow of approximately 50 ml min⁻¹/100 g. The important relationship between perfusion pressure and cerebral blood flow is governed primarily by the phenomenon of autoregulation, which may be defined as the intrinsic tendency of the brain to maintain a relatively constant blood flow in response to moderate variations in perfusion pressure. In man, cerebral blood flow remains constant within the pressure range of 65-140 mmHg. The autoregulatory mechanism fails (the lower limit) below 65 mmHg, when the vessels are dilated and flow then decreases with decreasing pressure. The brain compensates by increasing its extraction of oxygen so that the symptoms of cerebral ischaemia do not necessarily develop immediately.

At high values of arterial pressure a point is reached where cerebral vasoconstriction can not be sustained as cerebral blood increases; this is termed the upper limit of autoregulation. Factors that alter ability of the cerebral vessels to constrict or dilate interfere with autoregulation. (Graham, 1985)

Cerebral blood flow depends upon two factors, namely the pressure difference between its arteries and veins (cerebral perfusion pressure) and the resistance of the intervening vessels (cerebrovascular resistance). The cerebral perfusion pressure is commonly regarded as the difference between systemic arterial pressure and intracranial pressure. The critical point is reached when intracranial pressure increases to approximately 45 mmHg of systemic arterial pressure, following which cerebral blood flow decreases and the increasing intracranial pressure leads to a decrease in cerebral perfusion pressure where flow to the brain becomes patchy. (Seisjö, 1981)

***Energy :-**

It is produced in the brain almost entirely by oxidative metabolism of glucose.