



COMPARISON BETWEEN THE EFFECT OF HYPERBARIC AND NORMOBARIC HYPEROXIA ON THE FUNCTIONAL OUTCOME IN PATIENTS WITH TRAUMATIC BRAIN INJURY

Essay

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بِسْمِ اللَّهِ الرَّحْمَنِ الرَّحِيمِ

قالوا

لَسْبَدَانِكَ لَا نَعْلَمُ لَنَا
إِلَّا مَا عَلَّمْتَنَا إِنَّكَ أَنْتَ
الْعَلِيمُ الْعَظِيمُ

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LIST OF ABBREVIATIONS

AES	:	Apathy evaluation scale
AGE	:	Arterial gas emboli
APP	:	Amyloid precursor protein
ARDS	:	Acute respiratory distress syndrome
ATA	:	Atmospheres absolute
ATLS	:	Advanced Trauma Life Support
AVDO₂	:	Arteriovenous oxygen difference
BBB	:	Blood brain barrier
CaO₂	:	Arterial oxygen content
CBF	:	Cerebral blood flow
CBV	:	Cerebral blood volume
CDI	:	Central diabetes insipidus
CNS	:	Central nervous system
COPD	:	Chronic obstructive pulmonary disease
CPP	:	Cerebral perfusion pressure
CSF	:	Cerebrospinal fluid
CSW	:	Cerebral salt wasting
CT	:	Computed Tomography
DAI	:	Diffuse axonal injury
DALYs	:	Disability adjusted life years
DCS	:	Decompression sickness
DLCO	:	Diffusing capacity for carbon monoxide
DRS	:	Disability rating score
DVT	:	Deep vein thrombosis
EEG	:	Electroencephalography
EGOS	:	Extended Glasgow outcome scale

List of Abbreviations

ETCO₂	:	End-tidal carbon dioxide
FIO₂	:	Fraction of inspired Oxygen
FSW	:	Feet of sea water
GCS	:	Glasgow coma scale
GIT	:	Gastrointestinal tract
H₂	:	Histamine receptor -2
Hb	:	Haemoglobin
HBOT	:	Hyperbaric oxygen therapy
ICP	:	Intracranial pressure
ICU	:	Intensive care unit
IL-10	:	Interleukin-10
IL-4	:	Interleukin-4
IMPACT	:	International mission for prognosis & clinical trial
Lactate (art.)	:	Arterial lactate
Lactate (j.v)	:	Jugular venous lactate
LOI	:	Lactate oxygen index
MD	:	Microdialysis
MRI	:	Magnetic resonance imaging
MSW	:	Meter of seawater
MV	:	Mechanical ventilation
NBH	:	Normobaric hyperoxia
NCSE	:	Nonconvulsive status epilepticus
PaCO₂	:	Arterial carbon dioxide tension
PaO₂	:	Arterial oxygen tension
PCS	:	Postconcussion syndrome
PET	:	Positron emission tomography

List of Abbreviations

PSI	:	Pounds per square inch	PtiO₂
:		Brain tissue oxygen tension	PvO₂
	:	Jugular venous oxygen tension	rCBF
	:	Regional cerebral blood flow	
rCMRG	:	Regional cerebral metabolic rate for glucose	
rCMRO₂	:	Regional cerebral metabolic rate for oxygen	
rOEF	:	Regional oxygen extraction	
RSI	:	Rapid sequence intubation	
RTA	:	Road traffic accidents	
SAH	:	Subarachnoid hemorrhage	
SaO₂	:	Oxygen saturation of the arterial blood	
SIADH	:	Syndrome of inappropriate anti-diuretic hormone secretion	
SjvO₂	:	Jugular venous oxygen saturation	
SvO₂	:	Oxygen saturation of the jugular venous blood	
TAI	:	Traumatic axonal injury	
TBI	:	Traumatic brain injury	
TCD	:	Transcranial doppler	
UPTD	:	Unit pulmonary toxic dose	
VAP	:	Ventilator associated pneumonia	
VF	:	Ventricular fibrillation	
VFP	:	Ventricular fluid pressure	

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INTRODUCTION

Traumatic brain injury (TBI) continues to be a major cause of death and disability in both civilian and military populations throughout the world (*Maas et al., 2007*).

In Egypt, road traffic accidents is the leading cause of TBI, 70% of accidents are due to ignorance and carelessness, and 30% are the result of mechanical problems and poor road conditions. These include insufficient pedestrian crossing facilities and deficient traffic signs at intersections. The situation in Egypt is serious and getting worse year by year, especially exacerbated by the progressing density of traffic, with an annual increase of 80,000 vehicles (*El-Gindi et al., 2001*).

The first stages of cerebral injury after TBI are characterized by direct tissue damage and impaired regulation of cerebral blood flow and metabolism. This ischemic pattern leads to accumulation of lactic acid due to anaerobic glycolysis, increased membrane permeability, consecutive edema formation and this anaerobic metabolism is inadequate to maintain cellular energy states (*Werner and Engelhard, 2007*).

The fact that TBI has been shown to be associated with decreased brain tissue PO_2 and increased brain lactate levels has led to the general hypothesis that increasing brain tissue PO_2 may improve cerebral aerobic metabolism. Clinical studies based on this reasoning have involved such techniques as increasing the fraction of inspired oxygen (FIO_2) or hyperbaric oxygen therapy (HBOT) (*Wilson et al., 2004*).

Hyperbaric oxygen therapy (HBOT) is the therapeutic administration of 100% oxygen at environmental pressures greater than 1 atmosphere absolute (ATA). This involves placing the patient in an airtight vessel, increasing the pressure within that vessel, and administering 100% oxygen for respiration. In this way, it is possible to deliver a greatly increased partial pressure of oxygen to the tissues. Typically, treatments involve pressurization to between 1.5 and 3.0 ATA , for periods between 60 and 120 minutes, one or more times daily (*Bennett et al., 2012*).

Another method of supernormal O_2 delivery is increasing the FiO_2 to 100% at normobaric pressure. Normobaric hyperoxia therapy is a potentially attractive alternative to HBO $_2$ because of its ease of administration. Several studies have shown that as FiO_2 increases, there is a corresponding rise in brain tissue PO_2 . In addition, microdialysate lactate decrease, which likely indicates improvements in tissue hypoxia (*Tolias et al., 2004*).

AIM OF THE ESSAY

Is to discuss the effect of hyperbaric to normobaric hyperoxia on the functional outcome in patients with traumatic brain injury.

CHAPTER 1: EPIDEMIOLOGY AND PATHOPHYSIOLOGY OF TRAUMATIC BRAIN INJURY (TBI)

I) Epidemiology:

TBI accounts for approximately 40% of all deaths from acute injuries in the United States. Annually, 200,000 victims of TBI need hospitalization, and 1.74 million persons sustain mild TBI requiring an office visit or temporary disability for at least 1 day. The financial cost is estimated at approximately 4 billion dollars per year (*Corrigan et al., 2010*).

The TBI incidence rate in developing nations is generally higher than more developed nations and is predicted to surpass many diseases as a main cause of death and disability by the year 2030 (*Tagliaferri et al., 2005*).

Developing countries bear the brunt of the fatalities and disabilities from road traffic crashes, accounting for more than 85% of the world's road fatalities and about 90% of the total disability adjusted life years (DALYs) lost due to road traffic injuries. The problem is increasing in these countries at a fast rate, while it is declining in all industrialized nations (*Farghaly et al., 2005*).

In Egypt, road traffic accidents (RTA) is the leading cause of TBI, 70% of accidents are due to ignorance and carelessness, and 30% are the result of mechanical problems and poor road conditions. These include insufficient pedestrian crossing facilities and deficient traffic signs at intersections. The situation in Egypt is serious and getting worse year by year, especially exacerbated by the progressing density of traffic, with an annual increase of 80,000 vehicles (*El-Gindi et al., 2001*).

The Egyptian Ministry of Internal Affairs in March, 2001 reported that, 6% of accidents were due to roads and environmental conditions, 24% were due to vehicle transportation while the human factors represent about 70% of the causes of accidents (*Farghaly et al., 2005*).