

Electrocardiographic Disturbances in Head Trauma Patients

Thesis

Submitted for Partial Fulfillment of the Master
Degree in General Intensive Care

By

Mohamed Fawzy Ibrahim Aly

M.B.B.Ch, Faculty of Medicine, Tanta University

Under Supervisors

Prof. Dr. Adel Mikhail Fahmy

Professor of Anesthesia, Intensive Care & Pain Management Faculty of Medicine – Ain Shams University

Dr. Mahmoud Ahmed Abd Elhakim

Lecturer of Anesthesia, Intensive Care & Pain Management Faculty of Medicine – Ain Shams University

Dr. Amr Sobhy Abd El Kawy

Lecturer of Anesthesia, Intensive Care & Pain Management Faculty of Medicine – Ain Shams University

Faculty of Medicine
Ain Shams University
2019





First of all, thanks to Allah whose magnificent help was the main factor in completing this work.

I would like to express my profound gratitude and appreciation to Professor Dr. / Adel Mikhail Fahmy, Professor of Anesthesia, intensive Care and pain management Faculty of Medicine – Ain Shams University the orchestrator of this work. I do thank him for valuable advices and suggestions that gave me light in the darkest times. I can hardly express my thanks for all what he has done encouraging me in front of all the obstacles.

I am greatly indebted and grateful to Dr. / Mahmoud Ahmed Abd Elhakim Lecturer of Anesthesia, intensive Care and pain management Faculty of Medicine – Ain Shams University for his valuable supervision, brotherly advices and everlasting guidance. He usually motivated me, advised me and paved the way for me to finally produce this work and I pray a lot for him, asking ALLAH to forgive him.

I wish to thank Dr. / Amr Sobhy Abd El Kawy, Lecturer of Anesthesia, intensive Care and pain management, Faculty of Medicine – Ain Shams University, the dynamo of this research. He kindly carried the burden of revising the scientific writing of the thesis. I would like to express my deepest thanks and great appreciation to his. As without his contribution, this work would not exist this way.

And last and not least, I would like to thank my family who surrounded me with all the love and support I was in great need for, and shared with me the hardest hours throughout the entire work.

Contents

Subjects Pa	
List of abbreviations List of figures List of tables	V
• Introduction	1
Aim of the Work	4
• Review of Literature	
♦ Chapter (1): Head Trauma	5
♦ Chapter (2): Heart Brain Interactions	31
◆ Chapter (3): Clinical Value of ECG in Nor Cardiac Conditions	
Patients and Methods	53
• Results	61
• Discussion	74
• Summary	83
Conclusion and Recommendations	86
• References	88
Arabic Summary	

List of Abbreviations

AF : Atrial fibrillation

CAD : Coronary artery disease

CBF : Cerebral blood flow

CNS : Center nervous system

CO : Carbon monoxide

CSF : Cerebrospinal fluid

ECG : Electroencephalogrphic

LQTS : Long QT Syndrome

MAP : Mean arterial blood pressure

NSM : Neurogenic stunned myocardium

SAH : Subarachnoid haemorrhage

SAH : Subarachnoid hemorrhage

SCD : Sudden cardiac death

TBI : Traumatic brain injury

TCAs : Tricyclic antidepressants

VF : Ventricular fibrillation

VT : Ventricular tachycardia

.

List of Figures

No.	<u>Figure</u>	Page
1	Overview of cranial anatomy	8
2	The Meninges. The meninges cover the brain and consist of three layers: the dura mater, arachnoid, and pia mater.	11
<u>3</u>	Volume-Pressure Curve.	15
<u>4</u>	CT Scans of Intracranial Hematomas.	20
<u>5</u>	Overview of brain autonomic nerves and the heart as an interactive system.	34
<u>6</u>	Demographic data distribution of the study group.	61
<u>7</u>	Risk factors distribution of the study group.	62
<u>8</u>	Clinical data of the study group.	63
9	Types of traumatic brain injury of the study group.	64
<u>10</u>	Electrocardiographic findings in the study group.	66
<u>11</u>	Electrocardiographic findings in the study group.	67
12	Echocardiographic characteristics in the study group.	68

List of Tables

No.	<u>Table</u>	<u>Page</u>
1	Baseline Characteristics and demographic data.	65
<u>2</u>	Distribution of ECG and Echocardiographic changes.	69
<u>3</u>	Basal characteristics of laboratory Parameters in the studied patients.	70
4	Correlation between TBI and ECG changes in the study population.	72
<u>5</u>	Comparison of patients with ECG disturbances and without disturbances.	73

Introduction

Traumatic brain injury (TBI) is a major public health concern (**Vijay et al., 2014**). Complications after TBI known as second insults can worsen neurologic and patient outcomes (**Jeremitsky et al., 2003**).

Acute myocardial damage and heart failure can happen during brain injury in patients without a history of basic cardiovascular pathology (**Polderman et al., 2000**).

Cardiovascular complications are common after brain injury and are associated with increased mortality and morbidity (**Tamsin and Martin, 2012**).

Patients with head trauma may have a variety of electrocardiographic changes (Macmillan et al., 2002).

The neurogenic stunned myocardium (NSM) syndrome is characterized by ECG changes, cardiac arrhythmias, release of biomarkers of cardiac injury, and left ventricular dysfunction (**Tamsin and Martin, 2012**).

These changes may be disturbance of rate, rhythm, P wave, QRS complex which may be in the form of prolonged Q-T interval, ST segment elevation or depression, inversion or peaking of T wave and large positive U wave (Collier et al., 2004).

The association of specific ECG changes with intracranial disease has been recognized for 5 decades. ECG abnormalities occur most often in patients with subarachnoid hemorrhage, but also have been described in cases of ischemic stroke, intracranial hemorrhage, head trauma, neurosurgical procedures, acute meningitis, intracranial space-occupying tumors, and epilepsy (Surawicz, 1995).

Brain injury-related ECG abnormalities are particularly common after SAH where they are reported in 49–100% of cases. The most common findings are ST segment changes, flat or inverted T waves, prominent U waves, and prolongation of the QTc interval (QTc is the QT interval corrected for heart rate) (Grunsfeld et al., 2005).

Cardiac rhythm disturbances, including sinus tachycardia, atrial fibrillation, premature atrial and ventricular contractions, and AV dissociation, are also common after brain injury and usually occur within the first 7 days (Baguley et al., 2006).

The majority of studies suggest that these electrocardiographic abnormalities result from disturbances in the autonomic nervous system promoting a local excess of catecholamines associated with enhanced adrenal

production and activation of the calcium channels leads to an increase in cytosolic and mitochondrial calcium as well as release of free radicals causing contraction band necrosis and reflected as electrocardiogram alterations (**Baguley et al., 2006**).

Traumatic brain injury (TBI) and subarachnoid haemorrhage (SAH) induce a systemic catecholamine 'storm' driven by the central neuroendocrine axis which massively increases sympathetic outflow and activates the adrenal glands. Damage to the insular and hypothalamus also initiates a complex cascade of events, including activation followed by dysfunction of the autonomic nervous systems and an intense inflammatory response, which have major adverse effects on the heart (**Tamsin and Martin, 2012**).

Early ECG may serve as an inexpensive test to screen for cardiac dysfunction prior to ordering more expensive and potentially more invasive testing. So, ECG should be amandatory investigation for all patients with isolated traumatic brain injury (Jeremitsky et al., 2003).

Aim of the Work

If there are ECG changes in head trauma and certain type of brain injuries, a 12-lead ECG may be an important and inexpensive screening tool to evaluate TBI patients for cardiac dysfunction prior to further diagnostic studies or interventions, which will be useful in managing these cases and in selection and adjusting medication for TBI patients.

Chapter (1)

Head Trauma

Head injuries are among the most common types of trauma seen in North American emergency departments, with an estimated 1 million cases annually. Many patients with severe brain injuries die before reaching a hospital, and almost 90% of prehospital trauma-related deaths involve brain injury. About 70% of patients with brain injuries who receive medical attention can be categorized as having minor injuries, 15% as moderate, and 15% as severe. In 2003 there were an estimated 1,565,000 traumatic brain injuries (TBIs) in the United States, including 1,224,000 visits, 290,000 hospitalizations, and 51,000 deaths. Survivors of TBI are often left with neuropsychologic impairments that result in disabilities affecting work and social activity.

Every year up to 90,000 people in the United States suffer long-term disability from brain injury. In an average European country (Denmark), 363 per million inhabitants suffer moderate to severe TBI yearly, with more than one-third of these requiring brain injury rehabilitation. Therefore, even a small reduction in the mortality and

morbidity resulting from brain injury should have a major impact on public health (Faul and Wald et al., 2010)

One study searching for the leading causes of TBI in Egypt in 2010, found Male sex was predominantly affected, 79% of cases. Moderate and severe injuries account for 17.2% of all TBI presented cases (Montaser and Hassan, 2010)

In another one in Egypt found that road traffic accident was the commonest cause of trauma in 73.7% of the patients. 62.1% of the patients had mild head injury, 17.5% of the patients had moderate head injury, while the remaining 20.3% had severe head injury. Brain contusions, Brain edema, and epidural hematoma were the commonest traumatic pathology in 39.1%, 36.5%, 17.6% respectively. 63.6% of the patients had associated injuries rather than head injury. Good recovery was reported in 62.7% of the patients, moderate disability in 12.1% of the patients, while the mortality rate was 14.3% of the patients (**Taha and Barakat, 2016**).

Anatomy:

A review of cranial anatomy includes the scalp, skull, meninges, brain, ventricular system, cerebrospinal fluid, and tentorium (figure 1).

Scalp:

The scalp is made up of five layers of tissue that cover the skull: skin, connective tissue, aponeurosis, loose areolar tissue, and pericranium. Loose areolar tissue separates the aponeurosis from the pericranium and is the site of subgaleal hematomas. Because of the scalp's generous blood supply, scalp lacerations may result in major blood loss, especially in infants and children (**Oishi and Luce**, **1995**).

Skull:

The skull is composed of the cranial vault (calvaria and the base). The calvaria is especially thin in the temporal regions but is cushioned here by the temporalis muscle. The base of the skull is irregular, which may contribute to injury as the brain moves within the skull during acceleration and deceleration.

Chapter (1): Head Trauma

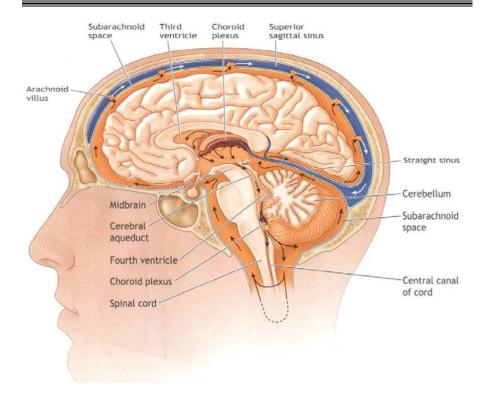


Fig. (1): Overview of cranial anatomy.

The floor of the cranial cavity is divided into three distinct regions: the anterior, middle, and posterior cranial fossae. Simply put, the anterior fossa houses the frontal lobes, the middle fossa the temporal lobes, and the posterior fossa the lower brainstem and the cerebellum (Parent and Carpenter, 1995).

Meninges:

The meninges cover the brain, and consist of three layers: the dura mater, arachnoid, and pia mater (figure 2). The dura mater is a tough, fibrous membrane that adheres