بسم الله الرحمن الرحيم

شبكة المعلومات الجامعية

التوثيق الالكتروني والميكروفيلم

شبكة المعلومات الجامعية

جامعة عين شمس

التوثيق الالكتروني والميكروفيلم

قسم

نقسم بالله العظيم أن المادة التي تم توثيقها وتسجيلها

علي هذه الأفلام قد أعدت دون أية تغيرات

يجب أن

تحفظ هذه الأفلام بعيدا عن الغبار

في درجة حرارة من ١٥-٢٥ مئوية ورطوبة نسبية من ٢٠-٤٠%

To be Kept away from Dust in Dry Cool place of 15-25- c and relative humidity 20-40%

بعض الوثائق الاصلية تالفة

بالرسالة صفحات
لم ترد بالاصل

Anesthetic Management Of The Severely Head Injured Patient. Implications Of The Measurement Of The Arterio-Jugular Venous Oxygen Difference And The Use Of The Free Radical Scavenger Tirilazad Mesylate

Thesis

Submitted to the Faculty of Medicine,
University of Alexandria,

In partial fulfillment of the requirements
Of the degree of

DOCTOR OF ANESTHESIA

By

Mohamed Medhat Khalil

MBBCh. Alex
MSAnesth Alex

Faculty of Medicine
University of Alexandria

2001

SUPERVISORS

Prof. Dr. Abd Elkader Zakaria Abd Elkader

Professor of Anesthesia,

Faculty of Medicine

University of Alexandria.

Prof. Dr. Wafaa Kamel Rady

Professor of Anesthesia,

Faculty of Medicine

University of Alexandria.

Dr. Hamed Mohamed Darwish

Assistant Professor of Anesthesia

Faculty of Medicine

University of Alexandria.

Co- Workers

Dr. Saleh Abd El Aziz Hamouda

Assistant Professor of Anesthesia

Faculty of Medicine

University of Alexandria.

For his experience in anesthetic management of neurotrauma

Dr. Yasser Mahmoud El Banna

Assistant Professor of Neurosurgery

Faculty of Medicine

University of Alexandria.

For his experience in the measurement of intracranial pressure

Acknowledgements

I am deeply obliged and grateful to **Prof. Dr. Abd El Kader Zakaria**, Professor of Anesthesiology for his kind paternal supervision, continuous encouragement, unlimited effort, patience and guidance without which the accomplishment of this work was not possible.

I would like to express my profound gratitude and appreciation to **Prof. Dr. Wafaa Kamel** Professor of Anesthesiology for her endless help, very kind supervision and selfless thoughtfulness.

My sincere gratitude and appreciation to **Dr. Hamed Darwish** Assistant Professor of Anesthesiology who provided me with great help and devoted guidance throughout this work.

I wish to express my deepest thanks to **Dr. Saleh Hamouda**, Assistant Professor of Anesthesiology for his great effort, continuous support and skillful guidance that helped me very much in every aspect.

I am greatly indebted to **Dr. Yasser El Banna** Assistant Professor of Neurosurgery for his encouragement, continuous advice, guidance and his invaluable help during the practical part of this work.

Finally, I would like to express my obligation to the personnel of the emergency operative theatres and the intensive care unit whose help was indispensable to make this work possible.

CONTENTS

INTRODUCTION

INTRODUCTION

Head injury has consistently plagued humans throughout history. Early documentation describing the sequelae of head injury has been reported by the ancient Egyptians as evidenced in the Edwin Smith papyrus which gave a simple protocol for determining the survivability from head trauma received in battle.[1]

Epidemiology

In the UK, head injury accounts for 9 deaths/100 000 population and is one of the commonest causes of death among the young adults.[2] In the USA, approximately two million head injuries occur each year with the rate of 175-200/100 000 population and causing as many as 56 000 deaths per year with staggering economic and emotional toll.[3]

Although men and women of all ages are affected, traumatic brain injury is predominantly a disease of young adult men with the peak incidence is the age range of 15-24 years, while secondary peaks are seen in infants and children and in the elderly.[4]

Pathology and pathophysiology of head injury

Current literature tends to classify brain damage resulting from head injury as focal or diffuse, and meanwhile emphasizing the concept of the existence of primary and secondary injuries.[5,6]

Primary damage (Fig 1, 2)

By definition, primary brain injury occurs at impact and may involve neural or vascular elements of the brain. It may take the form of focal or diffuse lesions.[7]

Focal lesions

Traumatic hematomas

May be epidural (extradural) or intradural with the latter being either subdural or intracerebral. Traumatic hematomas can occur alone or in combination and constitute a common and treatable source of morbidity and mortality resulting from brain shift, swelling, cerebral ischemia and elevated intracranial pressure.[8]

Epidural hematoma

It results from blunt trauma to the skull, fractures, mostly linear, are present in 30-91 percent of patients.[9,10] This hematoma progressively strips away the dura from the skull and widens the perimeter of the hematoma.[10] The pathological effects of epidural hematomas are primarily due to compression of the underlying brain and later due to swelling of this compressed area together with brain distortion and raised intracranial pressure (ICP).[10]

Subdural hematomas

These can be acute or chronic.[11]

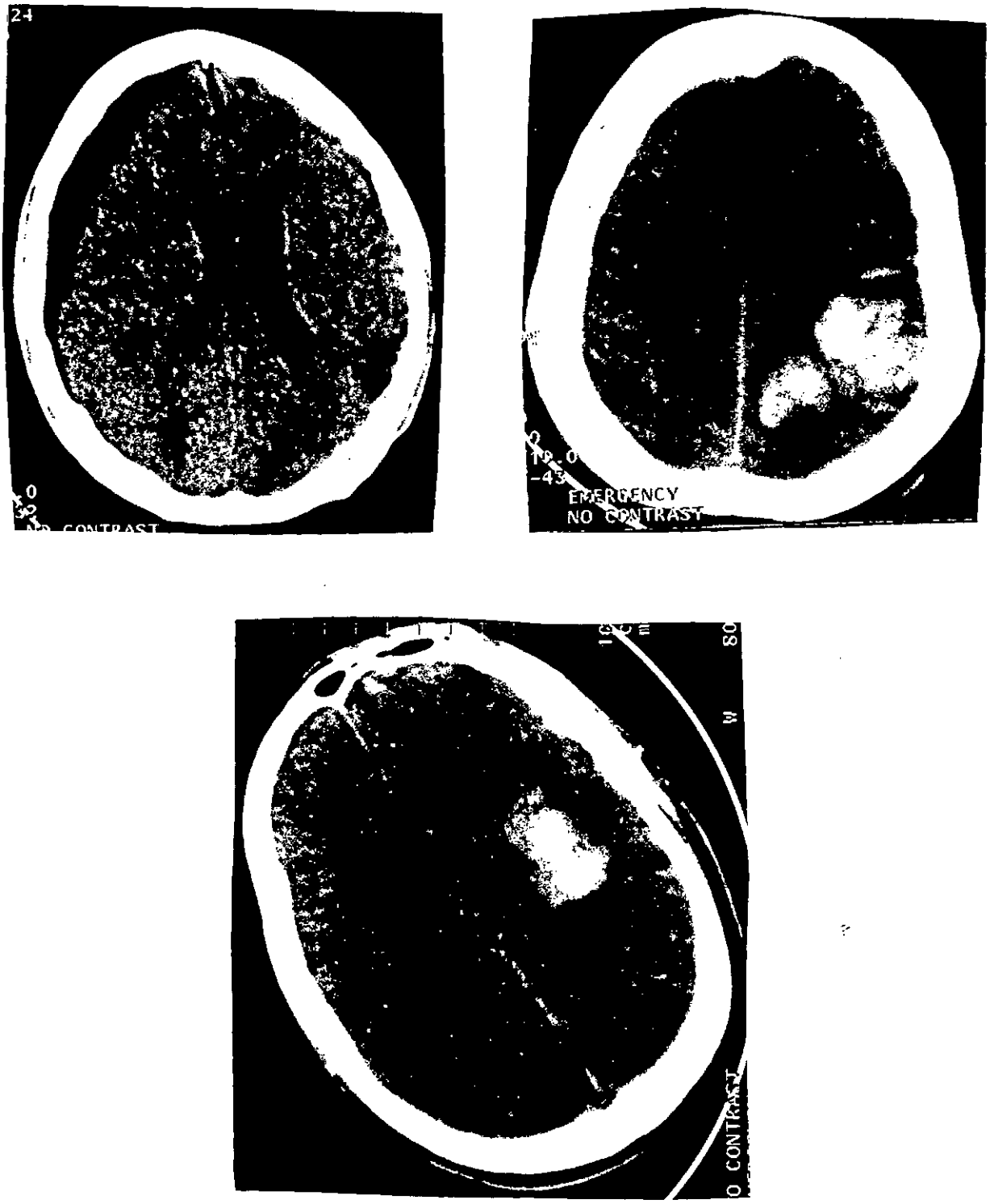

Fig. (1) Focal lesions

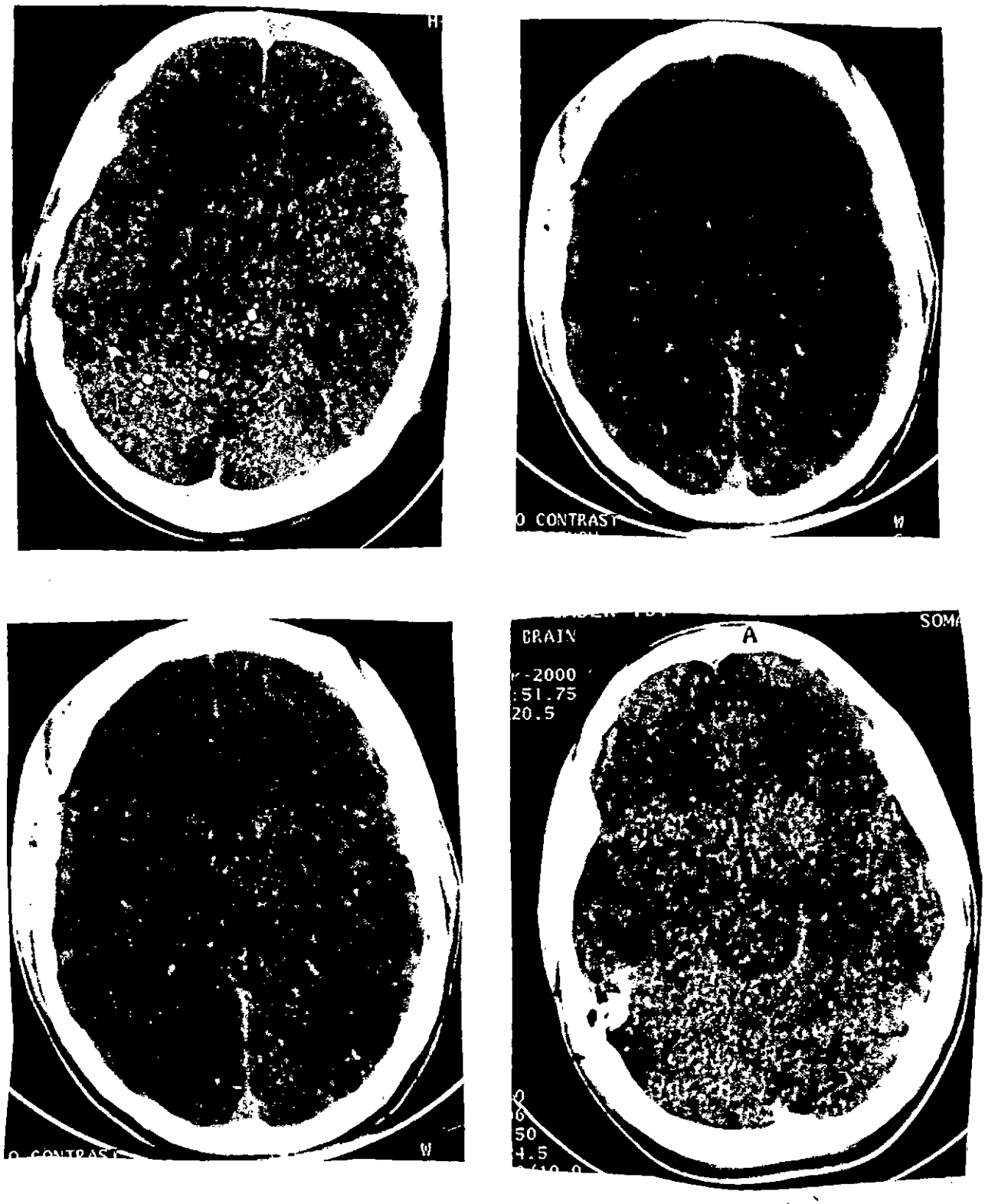

Fig. (2) Diffuse lesions

Acute subdural hematoma

Occurs in association with high rate of acceleration and deceleration of the head at the time of trauma. They probably result from inertial loading with high strain rates causing injury to vascular elements mainly the bridging veins in the subdural space where they lack reinforcement by arachnoid trabeculae.[12]

Acute subdural hematoma is one of the most lethal of all head injuries with reported mortality of 30-90 percent.[12,13] This high mortality and limited recovery may be due to the fact that the impact causing acute subdural hematoma commonly results in associated parenchymal brain injuries which may influence outcome more than the hematoma per se.[8]

Chronic subdural hematoma

They occur at least two weeks after trivial head injury but can persist months or years after such injury and most frequently occur in the elderly and in infants.[14]

Intracerebral hematoma

Intracerebral hematomas are present in about 15 percent of fatal head injuries. They may be single or multiple. They result from shearing and tensile stresses within the brain tissue leading to direct rupture of small vessels within the parenchyma at the moment of impact.[15] Intracerebral hematomas most frequently occur in the white matter of the frontal and temporal regions and are commonly associated with contusions, subdural hematomas and diffuse axonal injury. Deeper intracerebral hematomas occur in the basal ganglia, corpus callosum, centrum ovale and brain stem.[16]