International Journal for Multidisciplinary Research (IJFMR)
E-ISSN: 2582-2160 ● Website: www.ijfmr.com ● Email: editor@ijfmr.com

Epidemiology of Head Trauma in Emergency Surgical Intensive Care Units Retrospective Study in 302 Patients

Dr. Mohamed Amine Haidar[1], Dr. Soufiane Hanssal[2], Dr. Amine Schahrakane[3], Prof. Dr. Afak Nsiri[4], Prof. Dr. Rachid Alharrar[5]

[1,2,3]Anesthésiste Réanimateur, Emergency Surgical Icu (P33), Univesity Hospital Ibn Rochd Of Casablanca
[4]PES En Anesthésie Réanimation, Emergency Surgical ICU (P33), Univesity Hospital Ibn Rochd Of CASABLANCA
[5]PES Chef De Service P33 Anesthésie Réa, Emergency Surgical ICU (P33), Univesity Hospital Ibn Rochd Of CASABLANCA

Abstract

Head injuries are a major public health and socio-economic problem, especially in developing countries, because they affect young adults in particular, who make up the working population.

This is a retrospective study from 1 January 2023 to 31 December 2024. It will include all patients with severe head trauma (GCS ≤ 8) with isolated head trauma or as part of a polytrauma hospitalised in the surgical intensive care unit at Casablanca University Hospital ibn Rochd.

The aim of this study was to evaluate the epidemiological data and the various therapeutic aspects of severe head trauma. During this period, 65 head trauma patients, including 22 severe head trauma, were treated in the intensive care unit. The majority of patients (80%) was under 40 years of age and (77.27%) were males.

The etiologies were dominated by public road accident (72.73%).

The average blood pressure of our patients was 93.25 mmHg, ranging from 80 to 111 mm Hg.

More than 90% of patients had a Glasgow score > 5.

77% of patients had associated injuries (11 otorrhagia and 6 epistaxis; facial trauma, thoracic trauma, abdominal trauma, peripheral fracture).

CT scans were performed at 100% of patients, revealing 0 subdural haematomas, 3 extradural haematomas, 2 cerebral contusions, 7 meningeal haemorrhages and 12 intracerebral haemorrhages.

Thoracic and abdominal-pelvic CT scans were performed in 40% of patients.

All patients were intubated and ventilated.

A tracheostomy was performed secondarily in 18% of patients.

Catecholamines were used in 59.10% of cases.

Overall mortality was 72.73%.

Only 6 patients underwent surgery (3HSD, 1HSD and 2 embarrures).

50% of our patients presented with a pulmonary infection, the most common isolated germs being

streptococcus pneumonia, pseudomonas aeroginosa and staphylococcus aureus.
20% of patients have bedsores
Finally, we stress the importance of preventing road accidents by complying with the Highway Code, wearing seatbelts, regularly checking the mechanical condition of vehicles, improving road infrastructure and raising awareness of the risks and consequences of road accidents.

INTRODUCTION

Head trauma is a direct or indirect mechanical stress on the skull, immediately complicating or later of disorders of the awareness, synonymous of a suffering encephalic diffuse or localized going from lack of consciousness to a coma [1].
In Morocco, according to the accident statistics analysis report of the traffic road of the year of 2024, the incidence of the RTA East of 67 926 accidentswith 3 832 dead And a average of 10.4 death every day And more 102 040 woundedincluding 11,641 injured serious [2].
Several scores of gravity of the Head trauma, allow the classification at mild, moderate or severe head trauma, which will guide the course of action to be taken by the sequel.
It considered as benign if the Glasgow coma score ≥ 13 and serious if Glasgow ≤ 8 [3]
Major steps have been taken in recent years towards better understanding pathophysiological of trauma cranial, facilitating a approachdiagnostic And therapeutic multidisciplinary, THE causes And THE factors of mortality, will contribute not only to a better assessment of HTs, but also to reveal new avenues of research to improve the prognosis at short and medium term of these patients.
The goal of our work East to describe, through a series of 302 cases of Head trauma, collected At service of resuscitation surgical emergency:

- The aspects epidemiological.
- The clinics aspects.
- The organic and radiological particularities.
- The therapeutic management.
- Evolution.

MATERIALS AND METHODS

I. KIND AND FRAME OF STUDY

It is about a retrospective descriptive study.

- It consists of a study carried over a 6-year period (2019-2024), which involved 302 patients with a Head Trauma After an accident during this period. And Who concerned The following subjects :

- Epidemiology of traumatized cranial.
- Become of traumatized cranial.
- The pneumonias nosocomial at the head trauma victim.
- The hematoma extra Dural at the head trauma victim.
- The bruises in the head trauma victim.
- The need of a transfusion at the head trauma victim.

The exploitation of The files in our study consisted of a collection, dThe archives of service (P33), of all

the files of The sick hospitalized during The study period.
The data were collected by a form operating pre-established including epidemiological, clinical, biological, bacteriological data,profile of resistances germs and evolution.

II. PLACE OF STUDY :

Our study has summer conducted at service of The EMERGENCIES surgical ICU (P33) of HU Ibn Rochd of Casablanca.
Please note that The service is made up of 10 separate boxes, each box with a sink, liquid soap and hydroalcoholic solution dispenser. The medical charge is assured by six teachers, seven residents and two interns.
On the paramedic plan, the service includes 24 nurses, 2 physiotherapists and 4 agents.

III. ANALYSES STATISTICS :

The analysis carried out during this study focused on several points, namely:

- The incidence of head trauma.
- The kind and mechanism of the accident.
- The epidemiological data.
- The clinical data.
- The radiological data.
- The biological data.
- The assessment of HT patients.
- The treatment of HT.
- And finally, their developments.

IV. CRITERIA OF INCLUSION AND EXCLUSION :

1. Criteria of inclusion :

Have been included in The study:

- All patients who were hospitalized in the intensive care unit of emergency surgery having had a head trauma during The period of study.
- The patients over 15 years.

2. Criteria of exclusion :

Have been excluded from our study

- The patients aged of less then 15 years
- Another pattern hospitalization
- Admitted in a other service
- The patients arrived deceased at hospital
- The patients outgoing against notice medical.

RESULTS

I. EPIDEMIOLOGY:

1. Distribution in the time:

1.1.According to The year:

The number of traumatized cranial is variable from year to year, 29 in 2019 and 61 in 2024, an average of 50 cases/year.

Table I: Distribution of The cranial traumatized in function of The year.

Years	N	%
2019	29	10%
2020	44	15%
2021	53	17%
2022	55	18%
2023	60	20%
2024	61	20%
Total	**302**	**100.00%**

1.2.According to The month:

We demonstrated that the higher number of HT was observed in the month of June (50 case) and the lowest during month of May (9 case).

1.3.According to The day of The week:

The higher rate of Head trauma was observed The Saturdays with 82 cases (27%), and The lower rate was observed The Fridays with 29 case either 10%.

1.4.Distribution according to The time admission

We noted that The majority of The head traumatized 34% are admitted between 6 p.m and 00h, and that The lower 19% a summer observed between 2 p.m. And 6 p.m.

2. Distribution according to age:

It was found that 66% of patients were between The ages of 21 and 40 who represents The slice of age with The higher percentage (199 case), 5% between 16 and 20 years or 15 cases, 21% between 41 and 60 years or 63 cases while we only find 8% of HT with an age superior at 60 years old or 25 case.

3. Distribution according to The sex:

Our study has watch 83% of The patients either 250 case was of sex male so that only 17% of patients or 52 cases were female, which gives us a sex ratio of 4.80%.

4. Circumstance And mechanism of Head trauma :

4.1.Circumstance of Head trauma :

Our study has watch that the RTA(road traffic accident) represented the first cause of HT with64% or 192 cases, followed in second place by falls with 13% or 39 cases, As for The attacks, they represented 10% of all our patients, i.e. 30 case. And in last position we find the accidents of work with 4% (12 case). THE percentage of HT with A mechanism undetermined was of 9% either 29 case.

4.2.Mechanism of The head trauma:

4.2.1. Road traffic accident:

Among The 192 cases admitted for a HT following a road traffic accident, 121 of them aremotorcyclists, 63%, which is the main cause of Head trauma in our study.

4.2.2. The assaults:

In The 30 cases of HT following an assault, the causative agent was a stick in 33% or 10 cases, in 23% it was a stone or 7 cases, in 17% it was an armed white either 5 cases and in The 8 cases remaining The agent causal born was not determine 27%.

4.2.3. Fall of a height:

We have split our patients' victims of a HT caused by a fall in 2categories: less of 6m with 30 case either 77% and more of 6m with 9 case either 23%.

5. Breakdown by time to treatment:

Our study has find that there majority of the HT have being admitted with a deadline of time to treatment lower than 3 hours (56% of The cases) either 169 case, so that The longest deadline was from 6 p.m. with a deadline average of 3.88h.

Background:

76 % of our patients had no background pathological notables (either 230 patients),

7 % suffered of HTA (either 21 patients), 5 % had diabetes (either 16 patients) and 2% had asthma.

6. The notion of initial loss of awareness:

The initial loss of awareness was observed at 205 case either 68% of the patients.

7. Distribution according to the toxic factors of risk:

Toxic risk factors found in patients admitted for HT are smoking 15% or 45 patients, alcohol abuse 8% or 24 patients and The use of cannabis 6% either 17 patients.

The remaining 71% of patients, or 216, did not have toxic habits. notables.

II. CLINICAL APPEARANCE:

1. Neurological state:

In our series the assessment of neurological status was based on the following elements:

- The assessment of GCS admission.
- Pupil status.
- Deficit signs
- Seizures

1.1.State of consciousness:

Our study has distributed The HTs in three groups according to The score from initial Glasgow score:

The 1st group: severe HT with a score less than or equal to 8, concerning 52 patients either 17%.

The 2nd group: moderate HT with a score between 9 And 13,concerning 200 patients or 66 %.

The 3rd group: light HT with score of 14 Or 15, concerning 50 patients either 17%.

1.2.Size and reactivity of The pupils :

Our study has found that 257 case had of symmetrical and reactive pupils on admission, 20 cases had anisocoria, 16 cases had bilateral mydriasis and9 cases had A miosis.

1.3.Deficit signs:

We found A neurological deficit in 12 of our patients either 4%. The deficit signs were present at 8

patients of which 7 presented hemiplegia.

Table II: Distribution of HT according to the deficit signs presented at admission.

Signs loss-making	N
Motor deficit	8
Monoplegia	1
Hemiplegia	7
Sensitive deficit	4
Monoparesis	1
Hemiparesis	3

1.4.Seizures:

5 cases have presented seizures at their admission, it represents 2% of studied cases.

2. hemodynamics state:

2.1.Arterial Pressure:

To have an idea on the arterial pressure of our patients, we have calculated their average blood pressure. 5% presented a hypotension either 226 case had an adequate arterial pression either 75% And 61 case were in hypertension arterial.

2.2.Cardiac Frequency:

Our study classified our cases into 3 categories according to their heart rates, patients with bradycardia 6%, patients with tachycardia 12% and patients with normal heart rate who represent The majority of cases with a percentage of 82% either 247 patients.

3. respiratory Frequency:

The majority of our patients 217, were eupneic either 72%, 30 were in bradypnea either 10% and 55 were in tachypnea either 18%.

4. General Exam:

4.1.Lesions of scalp:

Two types of lesions were noted on the scalp, hairy leather wounds which represent 48% or 144 cases and scalp hematomas 32% or 96 case.

4.2.Flows through orifices:

25 case (8%) had associated earaches, 15 case have presented epistaxis either 5% and we noted only 1 case of rhinorrhea.

In the majority of the cases, the HT were isolated with a rate of 63%, the rest of the patients had associated lesions :

- Facial trauma was found at 32 patients either 11%.
- Spinal lesions were noted at 19 patients either 6%.
- Members fractures have been found at 28 patients either 9%.
- Thoracic lesions in 17 patients either 6%.
- Abdominals lesions at 13 patients either 4%.

- Basin trauma at 3 patients either 1%.

III. RADIOLOGICAL DATA:

1. Cerebral CT scan:

On The 302 patients, 266 case either 88% have benefited of a cerebral CT scan has at their admission and 12% does not, either 36 patients.

The CT scan identified parenchymal and bone lesions.

Cerebral contusions are the most common injuries with a rate of 45% or 136 cases of cerebral contusions followed by subdural hematoma with a rate of 37% And in 25% of The case either 76 case the CT scan was normal.

2. Others radiological exams:

Regarding other radiological examinations carried out, it was noted that 32% of patients had benefited of a Body scanner (cerebral + thoracic +abdominal + pelvic CT scan).

The X-ray thoracic has being realized at 66% of The patients and an abdominal ultrasound in 12% of The patients.

Table III : Distribution according to the different radiological exams carried out.

Exam radiological	Number	Percentage
X-ray thoracic	199	66%
Thoracic CT scan	167	55%
Ultrasound abdominal	35	12%
Abdominal CT scan	166	55%
X-ray of basin	8	3%
Members X-ray	95	31%
Uro-CT scan	4	1%
Cerebral MRI	10	3%

IV. BIOLOGICAL DATA:

1. Natreamia and Kaleamia

The 302 hospitalized patients had their potassium and sodium levels measured.

Sodium levels were normal in 70% (211 cases), hypernatreamia in 26% (78 cases) and hyponatreamia in 4% (13 cases).

With regard to potassium, 75% (226 cases) had normal potassium levels, while 13% (39 cases) had hypokalaemia and 12% (37 cases) had hyperkalaemia.

2. Complete blood count

All hospitalized patients had a complete blood count (CBC). Our study found that 56%, or 169 patients, presented with normocytic normochromic anaemia, 105 of whom required a blood transfusion.

In terms of white blood cells, 201 cases (67%) were found to have hyperleukocytosis.

Our study also found that 99 patients (33%) had thrombocytopenia.

3. renal Function:

All our patients had right to a dosage of creatinine and of urea to evaluate their renal function, our study has find that 23% of the case either 69 patients had an alteration of their renal function during

their hospitalization.

4. Transaminases

Our study has find that 46 % either 140 case had A high rate of ASAT and 53% or 160 case had a high rate of ALAT only.

5. Haemostasis work-up:

Our study has find that 40 % of The case either 120 patients have suffered of a disorder of hemostasis during their hospitalization.

6. CRP.

Our study has find that 229 patients either 76% had a high CRP level in their hospitalizations.

V. THE TREATMENT :

1. Medical Treatment:

The care received by our patients on admission has included:

- 2 peripheral veins of good caliber.
- Vascular filling with 0.9% saline or Haemaccel if in shock; 229 patients (76%) required this.
- 30° proclivity position.
- Stabilization of the cervical spine while keeping it aligned with The axis of The body.
- Suturing of bleeding wounds
- Injection of anti-tetanus serum.
- Prevent gastric ulcers.
- Analgesic treatment.
- Anticonvulsant treatment for all patients presenting with convulsive seizures.
- Additional biological and radiological tests.
- Intubation, ventilation, sedation and controlled ventilation for all head trauma patients.
- Blood transfusion for 105 patients, a rate of 35%.
- Antibiotic prophylaxis and antibiotic therapy.
- Sodium Valproate-based anti-coma treatment in 268 patients, witch is 89% of patients.
- Monitoring.

2. Surgical Treatment:

Only 28% either 85 patients hospitalized have needed surgical treatment, of which 66% was for evacuation of an extradural hematoma or a subdural hematoma.

3. Nutrition:

We noticed that 63% or 190 of our patients benefited from an enteral nutrition, 22% or 65 patients benefited from oral nutrition, 12% either 35 patients have benefited of a food, as to toremaining patients, 3%, benefited from a combination of enteral and parenteral.

To evaluate the role of the early nutrition, we have used albumin as marker, our study showed that 60% of our patients or 181 cases had a decreased albumin level on admission compared to 40% who had a normal level.

The albumin dosage on day 3 of hospitalization showed that 80% of our patients had a normal rate of

albumin.

VI. EVOLUTION :

1. hospitalization Duration:

The duration average of stay at service was in average of 33 ± 34 days withextremes going minimum 14 days has 87 days.

2. Favorable Evolution:

48% (N=145) of The patients have I had a evolution favorable And are gone out Aftera duration average of 23.56 days.

3. Transfer:

96 patients either 32% have summer transferred At service of neurosurgery For Acomplement of taking in charge

4. Complications during the hospitalization:

4.1.Short term Complications:

In our series, 136 patients either 45% have presented complications illustrated by the table below.

Table IV: Distribution of The HT according to short term complications.

Complication	Number	Percentage
Refractory ICHT	51	17%
Nosocomial Infection	105	35%
-Respiratory	63	21%
-Neuro-meningeal	21	7%
- Urinary Infection	14	5%
-Others	7	2%
-Bedsores	31	10%
Thromboembolic Complication	2	1%

4.2.Neurological sequelae:

They are found in 43% or 130 patients, with at The top of The list, The headaches found in 75 patients or 25% followed by motor deficits found in 18 patients or 6%.

5. Mortality:

5.1.Global mortality:

The mortality at SHT (severe head trauma) was 77% either 40 patients.
The mortality of the light and moderate HT was 8% either 21 patients.
Which means that mortality rate in our series was 20% either 61 patients.

5.2.Cause of death:

The first cause of mortality identified in our study is the neurological aggravation with a rate of 80% or 49 cases, followed by infectious and hemodynamics causes.

DISCUSSION

I. REMINDERS

1. cerebral Hemodynamics:

1.1.The intracranial pressure [4] :

The ICP results of a balance of pressure between three sectors represented by the cerebral parenchyma, the fluid compartment and the vascular compartment.

The equation of Monro-Kell establishes a ICP constancy at price of a stability of The volumes and that all increase of volume of The intracranial compartments must necessarily be accompanied of a decrease of volume of The two other compartments to hold this constancy.

The brain parenchyma has no capacity for anaerobic metabolism, that is for this reason its functioning is controlled by cerebral circulation, therefore its contribution in oxygen depends on The balance of The PPC.

After A Head trauma, the abilities of go reactivity arteriolar may be affected to The detriment of cerebral perfusion. The lower limitof plateau self-regulation himself find SO deviated towards the high values of pressure.

Under conditions of preserved self-regulation, an increase in PPC is responsible for cerebral vasoconstriction, leading to a reduction in VSC, to achieve stabilization, or even a reduction in ICP. On The other hand, under these same conditions, a decrease in PPC within The limits of The plateau self-regulation conduit has a vasodilatation cerebral, responsible of aincrease of The VSC And by consequent of The PEAK.

II. THE DIFFERENT CRANIOENCEPHALIC LESIONS:

There are two types of lesions that are often associated, the lesions that we direct calls in relation to The impact of The skull against a traumatic agent and those which are secondary to phenomena of acceleration or deceleration which we calls lesions indirect and which follow to movements of the head [7].

1. THE direct lesions:

A direct impact is responsible of lesions of contact.

He can train in first place of The wounds of leather hairy, a deformation Ora breakup of The envelopes being able to drive has of The lesions of kind fracture of skull, EDH Or SDH. Energy of shock can be transmitted the underlying encephalon and be the cause of intra-parenchymal lesions (contusions): EDH, SDH, cerebral contusion, hemorrhage subarachnoid post-traumatic [7].

1.1.THE wounds of leather hairy :

In anatomy, The scalp represents a synonymous For leather hairy, And in traumatology he designates a wound imposing below shape of detachment subcutaneousextended by tearing rather than by cutting clear by armed white.

The wound of scalp is a vascular emergency and stop hemorrhage by all the means is an absolute emergency in order to avoid a shock.

All wound of scalp must be considered as a severe wound and be sutured before the transfer of a traumatized patient.

The large wounds of scalp must be sutured at OR in ensuring an inspection of the skull, a disinfection before considering the suture.

1.2.The fractures:

The cranial traumas can generate simple Or displaced fracture, which are called embarrures, The

fractures can in turn be responsible else lesions such that THE reached vascular has The origin of hematoma, of fistula of LCR Who are has The origin of The meningitis post traumatic[6].

1.3.The hemorrhage subarachnoid:

Subarachnoid hemorrhage (SAH) is common after Head trauma severe of origin usually venous. They exhibit At riskhydrocephalus acute, Or more often delayed by disorder of there resorption of LCS,at The level of The Pacchioni granulations or The lower orifice of The fourth ventricle in case intrauterine hemorrhage ventricular [8]

1.4.The extradural hematoma:

Extradural hematoma (EDH) is a collection of blood formed between The internal bony table and dura mater, often associated with arterial damage middle meningeal [9], explaining the topographical predilection of EDHs for the temporal area. EDH mainly affects men between the ages of 10 and 50, less easily detachable from the dura mater in the elderly, explains its rarity after 65 years.

Figure 1 : Brain ct scan Picture of an extra dural hematoma

In infant, it's far to be exceptional despite elasticity of the vaulted linked to age [10].

The EDH begins generally by a loss of initial awareness, A back the a normal state of awareness (interval free), Then a recurrence of The disorders ofvigilance can lead to coma; in The absence of treatment, a worsening contralateral motor deficit, ipsilateral mydriasis (engagement temporal with suffering of The common ocular motor nerve), finally signs of decerebration (suffering of The trunk cerebral). Of The shapes with coma straight away sign The high-pitched character of The HED [11]

1.5.The subdural hematoma:

Acute subdural hematoma (ASH) is a collection of blood formed between The hard way mother and THE brain, of origin traumatic in 71% of The case [12].

Her expression clinical occurs immediately After THE trauma either InThe 72 hours following The trauma; The HSDA blames The rupture of a vein, of a artery Or Again THE bleeding of a hearth attrition cerebral (combining tissue destruction, ischemic focus, blood clots and affecting The cortex And The

white matter) [11].
Traffic accidents represent The most serious contingent of injured (24% of cases), falls and assaults are responsible for 72% of HSDA [7].
Most often, HSDA manifests itself with a coma at The outset, but it can display The symptoms of an extradural hematoma with free interval, then disordersvigilance, contralateral deficit, coma, homolateral mydriasis, finally signs of decerebration ; Sometimes The HSDA is relatively good tolerated with presence of signshypertension intracranial And disorders of minimal vigilance.
A such painting can himself stabilize Or to get worse secondarily. In All state ofcause, THE diagnosis of The HSDA born can rely on alone on The painting clinical [11].

1.6.Cerebral contusions and hematomas intracerebral [6,10]:

Result from The contact between there surface cerebral and The bone of skull.

They are often associated with focal ischemia, intracerebral hematomaAnd edema that can cause a commitment intracranial.
They represent 10% of serious head injuries. Intracranial injuries brain are related to damage to The blood-brain barrier. All degrees are possible between simple edematous contusion and destruction parenchymatous. THE lesions vascular associated can to train there constitution of a intracerebral hematoma.
Ventricular hemorrhage causing hydrocephalus may also accompany these lesions.

2. THE indirect lesions:

THE movements acceleration and/deceleration Or of rotation linked At shift fast of there head are going to provoke of The lesions diffuse And multifocals. Thisphenomenon observes itself has The state pure during of The accidents of there traffic with belt ofsafety, where The head will tilt around The hinge of The cervical spine without hitting an obstacle, these types of lesions cause neuronal, glial ormore or less significant vascular [13].
High pressures cause direct bruising at The point of impact.
Opposite The point of impact, negative pressures generate forces of tensions responsible for identical lesions called “contusion contusion” [13].

III. PATHOGENIES OF THE LESIONS CRANIO-CEREBRALS :

We distinguished The primary lesions, immediately present After the trauma, and secondary lesions that develop in a delayed manner in a few hours.
If the primary lesion is inevitable, the secondary must be thoroughly screened and, if that is possible, treated.

1. Primary Lesions:

THE lesions direct Or indirect are going cause of The lesions cell phones (neuronalOr glial) or vascular more Or less important either reversible or No.
At moment of The impact we can note of The phenomena temporary(vasospasm acute initial, depletion of The stocks energy) And, very quickly, InThe seconds following The impact, a neuro-excitatory phase, then inhibitory, linkedhas mechanical stimulation of The activity synaptic [14].

2. Secondary Lesions:

The neurological damages do not occur immediately at the moment of the impact (primary lesions) but

can evolve secondarily, It's what we call The secondary lesions.
There most of secondary lesions are caused cerebral by edema, who give an increase of the intracranial pressure and a subsequent decrease of The cerebral infusion driving ischemia [15].
THE secondary cerebral lesions represent the first cause of death in hospital of The HT[15].

Table V : Secondary brain damage [14].

Extracranial	Intracranial
Arterial Hypotension	Process expansive
Hypoxemia	Intracranial Hypertension
Hypercapnia	Vasospasm
Deep Hypocapnia	Seizures
Arterial Hypertension	Hydrocephalus
Acute Anemia	Infection
Hypo- Or hyperglycemia	
Hyperthermia	
Hyponatremia/ Hypernatremia	

2.1.The intracranial lesions:

2.1.1. Intracranial Hypertension:

Intracranial hypertension (ICH) results from increased volume cerebral by consequent of The PIC has The interior of The cranium rigid.
The risks caused by this anomaly are twofold: cerebral engagement leading to brain death and worsening or development of lesionscerebral ischemic in report with The decline of cerebral blood Speed (CBS). It creates a vicious circle with self-aggravation between cerebral edema, ICHT and decrease in CBS [16].

2.1.2. Edema cerebral (CO)[17]:

CO is defined by The net accumulation of water and solutes in The area intracellular and/or In THE sector extracellular cerebral, has The origin of a increase of volume of The mass cerebral.
The classification proposed in 1967 by Igor Klatzo remains The most viable. This classification is founded on two types of CO: cytotoxic edema, called edemacellular, is observed when neurons or glial cells are unable to maintain their ionic gradient, so that The regulation of cell volume is lost. cytotoxic Edema itmself encounter the more often during an ischemic or hypoxic achievement, and predominates in there substance gray; vasogenic edema, where The opening of The HEB causes a passage of water, electrolytes AND proteins In the interstitial sector, this edema appears when the vessels are enough injured to allow a liquid, rich in proteins, of flee in interstitial Inspace under the influence of the intravascular hydrostatic pressure.
In there most of The case, the two types of CO, cellular and vasogenic,coexist, with an onset time between 1 and 6 hours, a maximum amplitude at24-48 hours, And a resolution spontaneous between 5 And 15 days in The absence ofcomplications.

2.1.3. THE vasospasm:

The post-traumatic vasospasm is due to meningeal hemorrhage, the risk, the duration and the consequences of post-traumatic vasospasm vary greatly in the literature. The risk of vasospasm is

evaluated between 27 And 40 % For the anterior circulation and 20% of patients for the posterior circulation [18].

2.1.4. The seizures:

Management of seizures secondary to head trauma should be brought to the forefront, because they can be harmful, by aggravating the existing lesions by The elevation of the intracranial pressure, of the arterial pressure and of cerebral metabolism.

The risk factors known responsible of The seizures are a cortical contusion, a fracture of The skull bone with a sinking, a SDH, an extradural hematoma, an intracerebral hematoma and a penetrating Head trauma.

It's important to think, in front of a late awakening, of subclinical seizures which will be detected by a electroencephalogram (EEG) [6].

Most hypnotic agents have anticonvulsant action. They are equally effective on simple crises but also in status epilepticus. This property is important since convulsive seizures constitute a cerebral assault which is not always clinically detectable [19].

2.1.5. Cerebral Ischemia:

The brain's vulnerability results from The low energy reserves does it have and its high metabolic needs. A decrease in CBF (frequent in during HT) creates a situation of ischemia responsible for a cascade of reactions At cellular level at The origin of The extension lesions.

The fight against ischemia involves reducing consumption metabolic as well as improving cerebral tolerance to ischemia. sedation deep And hypothermia moderate are, In these terms, THE main axes therapeutic [19].

2.2.The systemic secondary assaults:

This are pathological situations at course of which, it exists an inadequacy between the oxygen cerebral deliverance and the oxygen cerebral consumption. The main determinants of cerebral blood flow (CBF), of a part, are cerebral perfusion pressure (CPP) and arterial partial pressure in CO2 (PaCO2) via cerebral vasoreactivity. On The other hand, The Cerebral metabolism and cerebral oxygen consumption (CMRO2) are linked for 80% to cortical electrical activity.

Therefore, any situation affecting PPC (therefore blood pressure average Or there pressure intracranial), there PaCO2 Or The activity cerebral East likely to disrupt The balance between DSC and CMRO2 and induce ischemia cerebral.

The main secondary cerebral injury of systemic origin (SCISO) are: hypocapnia, hypotension, acidosis, hypoxia, hyperglycemia, hyperthermia, intracranial hypertension, hypothermia, hypercapnia, the occurrence of coagulopathy or epilepsy [21].

IV. HEAD INJURY ASSESSMENT SCALES:

1. The clinical evaluation scales:

1.1.The Glasgow coma scale:

the Glasgow coma scale (GCS) is the prognosis classification of The comas most widely used traumatic in the world developed by Teasdale and Jennet in 1974. It consists of testing three settings: The opening of The eyes, the verbal answer and the motor response [6]. It should not be forgotten that in children, the verbal response must be adapted to age.

To optimize it use, we must respect a few rules:

- The validated nociceptive stimulation method is pressure applied to The supraorbital level or The pressure of The nail bed with a pen
- In case of bruise or edema of the eyelids, The opening of The eyes is not evaluable. In this case, a global GCS cannot be calculated, the calculation will focus on the answers that remain evaluable.
- In case of asymmetry, we will hold account of the best answer obtained.
- Possible arterial hypotension and/or hypoxia must be corrected. For to have a score of reference.

It should be remembered that Teasdale and Jennet recommend assessing The GCS six hours After THE trauma, because its value initial can Sometimes be random [22].

1.2.THE others scales of The assessment clinical :

The study of brainstem reflexes called The Liège score can be usedto improve The prognostic value of The GCS, and which becomes The GCS-cork. This ladder has as setting, The assessment of The reflexes fronto-orbicularis, oculocephalic, photomotor, and oculocardiac. Unfortunately this scaleis little used in current practice, because of possible damage to The cervical spine makes it dangerous THE maneuvers necessary has these examinations [23].

Table VI: Score of cork [72].

Front orbicular	**5**
Oculo-cephalic vertical	4
Photomotor	3
Oculo-cephalic horizontal	2
Oculocardial	1
None	0

2. The CT scan scale:

The classification that is currently used is that of The TCDB. It has The advantage of simplicity and validation on one of The largest and recent patients series. Intracranial lesions were divided into two categories which are the diffuse lesions and the lesions including a picture of highdensity of a volume greater than 25 ml (mass lesions). Diffuse lesions are classified into four stages according to The visibility of The perished encephalic cisterns and The deviation of The midline**.** The compression of The cisterns of The base is a well-known sign of poor prognosis, indicating a high risk of HIC severe. THE lesions of mass are divided in lesions evacuated surgically and lesions not evacuated. THE prognosis clinical it closely related hasthere TDM class of patients [22].

3. THE score of exit GOS:

A other ladder devaluation of The team of Glasgow East THE Glasgow OutcomeScale (GOS) which should not be confused with The GCS. The GOS is a scale allowing of define THE become of The patients After A Head trauma severe. THE Data from this scale are often grouped into three categories: patients deceased, THE patients suffering of after-effects severe Or in state vegetative, And THE patients having of The after-effects minimal Or without sequel. This score East measure usually hasthere exit of there resuscitation, then to 3 and 6 months After THE trauma [22].

Painting XXVI : *Glasgow Outcome Scale*

1 Good recovery	Not of after-effects Or after-effects minors
2 Inability moderate	After-effects but patient independent
3 Inability severe	Aware but addicted
4 State vegetative	
5 Deaths	

V. EPIDEMIOLOGY :

1. Distribution In THE time :

1.1.In function of The year :

OUR study has watch that despite improvement of The means of awareness,THE number of HT does not stop increasing across the years.

This concord with the results of The others series Shtouki [24], AitSoltana [25], Babacar Diop [26] And F. Haddar [27].

1.2.In function of month:

According to our study, the distribution of HT according to the month of the year was mainly during The summer months (June, July, August, September) with a frequency of 53.97%.

Which is consistent with The work carried out in Morocco [24, 25, 27, 28, 29], these results can be explained by the importance of road traffic and The return of Moroccans residents abroad.

1.3.In function of The days of there week :

The majority of HT were recorded during the weekend with a rate of 43%And the beginning of the week with A rate of 14%, Who agree with the days of rest and the resumption of activities.

The same observation is made by other authors A. Chtouki [24], A. Ait Soltana [25], F. Haddar [27], LJJ. Lawson [31] And P. Skippen [32]

1.4.In function of The time admission :

Unlike A. Ait Soltana [25], who found that The majority of TCs (60.5%) were admitted between 1 p.m. and 2 a.m., our study found that The majority of Head trauma (61%) were arriving between 6 p.m. - 8 a.m.

There even observation was described by F. Haddar [27].

This can be explained by The increase in alcohol consumption and drug use illicit during these hours [33].

2. Distribution according to age :

In our series, we found that The majority of cases were young patients, in fact 71% of patients were under 40 years old, with a predominance of the age group ranging from 20 to 40 years which represents 66% with an average age of 37.15 years. According to W. Peeters [43], in a study carried out between 2003 and 2012, which covered all Belgian hospitals, the highest incidence rates are observedIn THE groups of age more young people (0-4 years) And the more aged (85+). The rate of incidence For the groups of age 65-84 years and 85+ have increased from 237.2 to 450.4 for 100,000 in 2003 and from 252.8 to 653.3 for 100,000 in 2012.

This is explained by The demographic curve of ages on The one hand, and on The other share by The activity of subjects young people.

Table VII : Age AVERAGE according to THE results of the literature.

Authors	Year	Age AVERAGE
I PM Rouxel [36]	1999	36 ± 18
Helm And al [37]	1999	37 ± 21
HAS. Martin And al [38]	2000	35 ± 15.4
F. Bernard [39]	2006	37 ± 14
I. Sadeq [40]	2006	38
K. Stylianos [41]	2010	34 ± 14.4
Z. Charani [28]	2013	36
S. Belachi [34]	2013	40
H. Khay [42]	2014	39 ± 18.4
F. Haddar [27]	2016	37.73 ± 15.7
In there series studied	2020	33

3. Distribution according to sex :

As in The majority of studies 36, 37, 27, 43, 35, 39, 28, 34, 40], our work has note the clear predominance masculine (83%), with a sex ratio of 4.80%.

In a study carried out by W. Peeters [43], we note a predominance of men in all age groups except The two oldest groups (65-84and 85+). In The 85+ age group, there is an average male/female ratio of 0.5. SO that In there population Belgian, THE report East of 0.4 For This band of age.There preponderance related of The women can to explain by their distribution of The SexesIn The oldest age group. However, data from W. Peeters [43] agree with our results, because his work found an incidence rate of HT more raised at home THE men in all The groups of age.

This East explain by the prevalence of The activities has risk at The house of The man.

Painting XXVIII : Distribution of The HT according to THE sex In there literature.

Authors	Number	Men	Women	Sex ratio (M/F)
I PM Rouxel [36]	304	228	76	3
Helm And al [37]	122	93	29	3.9
G. Bouhours [35]	114	91	23	4
Bernard And al [48]	113	89	24	3.7
Z. Charani [28]	101	96	5	19.2
S. Belachi [34]	55	49	6	8.16
I. Sadeq [40]	393	313	80	3.9
F. Haddar [27]	10236	8224	2012	4.08
Mr. Ejjail [43]	30	27	3	9
OUR series	302	250	52	4.80

Circumstances of trauma :

Public road accidents (PRA) come first. In our series, theyconstitute 64% of The causes. This East noted by their majority of The authors [36, 35, 39,28, 34, 40, 27, 43].

According to A. Capizzi [9], The most common HTmechanisms, in order decreasing of frequency, are

THE following THE falls involuntary, THE shocksinvoluntary against A object, THE accidents of the road, THE assaults and self-mutilation intentional.

Table VIII : Variation circumstances of HT according to THE authors

Authors	RTA (%)	Fall (%)	Assault (%)	Others (%)
J PM Rouxel [36]	65	17	8	10
G. Bouhours [35]	86	6	5	3
Bernard And al [39]	63	24	-	7
Z. Charani[28]	56	23	19	2
S. Belachi [34]	65.5	21.8	9.1	3.6
I. Sadeq [40]	72.56	18	4.89	5.55
F. Haddar[27]	63.75	20.78	11.88	3.59
Mr. Ejjail [43]	83.33	16.66	-	-
OUR series	64	13	10	13

4. Distribution in function of deadline of socket in charge :

The immediate post-traumatic period is The one that presents The greatest risksecondary ischemic aggravation. Now we know that episodes of ischemia determine a large part of The prognosis and that patients who died after TCG are all deceased with of many lesions ischemic [45].

English speakers talk about The golden hour. In fact, The care in The first hour Who suit THE trauma decreases THE risk of ischemia cerebral [46,47].

In effect, there most of The studies, have proven THE role of hospitalization early Andthere socket in charge fast And codified In improvement of evolution of The HTby therelimitation of risk of aggravation secondary of The lesions cerebral initials (ACSOS) [48, 49, 50].

In The study of S. Belachi [34], THE deadline AVERAGE of there PEC was more of 6 a.m. at The house of90.9%. In I. Sadeq [40], 64% of patients were treated in less than 6 hours.

SO that In The study of G. Bouhours And Al [35], has watch that THE deadline AVERAGEwas 1:55 ± 48 min.

And finally we note that in The study of F. Haddar [27], The time takenin charge varied of 30 min to 72 hours with a average of 5.12 ± 8.22h.

In our series, The majority of TCs were admitted with a delay in treatment charge less than 3 hours, that is to say 56% of cases or 169 cases, while The delay THE greater was of 6 p.m., with a delay AVERAGE of 3.88h.

5. Background :

76% of our patients had no notable pathological history (i.e. 230 patients), and only 24% had background.

In The literature, there are few studies that mention The antecedents of sick. At The Lille University Hospital [51], 21.7% of patients have a history psychiatric, 39.1% have a history of alcoholism, 52.2% have instability family, and 34.8% have instability professional or school.

In The study of F. Haddar [27], 82.1% of The patients born presented not of background pathological

against 17.9%, his results agree with those of Mr.Ejjail [44], who found in his series that 76.67% of cases did not present of background individuals.

Table IX : Distribution of The patients according to THE ATCDs.

Author	Background	Not of background
F. Haddar [27]	82.1%	17.9%
Mr. Ejjail [44]	76.67%	23.33%
In OUR series	76%	24%

6. There notion of loss of awareness initial :

In case of initial loss of consciousness, one should try to estimate its duration. can not to have initial loss of consciousness but post-amnesia traumatic which has broadly The same meaning, because both testify to a significant trauma, i.e. a transient impairment of functioning cerebral. A study on there population from Ohio [56] has find a prevalence of there lossof awareness by 21.7%, of which 9% had less 15 years old.

7. Distribution according to THE factors of risk toxic :

Our study found that The most common toxic habits among people with head trauma are smoking with 15%, alcohol with 8% And The use of cannabis with 6%.

In a study by Ponsford J, at The Epworth Rehabilitation Centre in London [33], 31.4% of patients with HThad consumed alcohol before The accident. So only 9% had A drug problem.

In The study of F. Haddar [27], 3944 of The cases were intoxicated at The moment of The accident either 38.53% of all of The case.

So alcoholism is a major cause of RTA, this is confirmed by THE data of The literature (table 29).

Table X : Distribution of The patients according to The ATCD of alcoholism

Author	Country of study	Year	ATCD of alcoholism (%)
F. Dance And al. [53]	France	1994	15
HAS. Ait Soltana [25]	Morocco	1997	22
A. El Qadiri [54]	Morocco	2001	7
E. Wong And al. [55]	Singapore	2002	18.7
O. Kozlowski [51]	France	2002	39.1
Sadeq I. [40]	Morocco	2006	23
F. Haddar [27]	Morocco	2016	38.53
OUR series	Morocco	2020	8

VI. CLINICAL APPEARANCE:

The diagnostic and therapeutic strategy for severe head injuries andisolated is based on an initial clinical assessment of The trauma victim which collects The anamnestic data, THE settings vital and establishes THE balance sheet lesional [35,57].

1. Respiratory State:

In The first hours, respiratory problems are The consequence of easily overlooked chest trauma in head

and neck trauma patients clutter upper airway related has The state comatose.

1.1.Hypoxemia:

Hypoxemic episode is defined by a PaO2 less than 60 mm Hg or SpO2 < 90% [58].

Hypoxemia remains a surprisingly common ACSOS, therefore its management charge must be quickly corrected speak control of The ways aerial and there ventilation mechanics [5].

E. Tentillier et al [58] found in his study that more than half of The TCGs have a SaO2 < 97%, of which 12% have severe hypoxemia or a SaO2 < 92%. The study of G. Bouhours and al [35], found that 33 patients or 29% have a SaO2

< 92%. This Who confirmed there frequency of hypoxemia at The house of THE TCG.

Charani explained that hypoxia was mostly due to a simple release incomplete ways aerial superiors [28].

In OUR series, all THE patients benefited of monitoring of Sp02.

1.2.Hypercapnia:

The deleterious effect on ICP is well known. However, little work has been done interested in its incidence and impact during TC. The authors conclude that The TCG with coma East systematically accompanied of a hypoventilation,directly correlated has the depth of coma, without that he either possible of determineThe relative share of partial upper airway obstruction to that of central neurological depression [21]. The PaCO2 value observed at reception has a prognostic value: among a cohort of 77 severe head trauma patients, mortality was multiplied by four at The house of THE patients hypocapnic or hypercapnic [59].

1.3.Hypocapnia:

Hypocapnia severe accentuates THE risk of ischemia cerebral: in underneath of 25mm Hg, She induced a vasoconstriction enough important For reducedangerously The DSC.

This risk also exists for higher Paco2 values. In case of TCG, we therefore recommend moderate hypocapnia called safety hypocapnia with The following: objective a PaCO2 located in The surrounding area of 35 mm Hg.

Hypocapnia controlled more deep it has considered as of The therapeutic of hypertension intracranial documented, ideally below continuous monitoring of SVJO² or PIC, or in The presence of a clear clinical sign of isolation cerebral [13].

2. State hemodynamics:

The primary goal of HT management is The restoration of a proper cerebral perfusion through prevention and treatment of any disorders hemodynamics [60].

2.1.Hypotension:

The episode hypotensive East defined by a pressure arterial systolic inferiorat 90 mmHg for more than five minutes or a MAP less than 60 mmHg [61].

It has been clearly demonstrated that a single episode of low blood pressure (SBP) inferior has 90 mmHg) in pre-hospital, double there mortality And that THE patients of whichThere NOT born was not corrected during THE transportation have THE prognosis THE more dark. This explain Why all THE recommendations published contraindicate formally of tolerate a lower PAS has 90 mmHg [36].

The first cause of hypotension found is hemorrhage. Some cases have have been described after osmotherapy (mannitol) and appear to be linked to hypovolemia caused by The diuretic properties of osmolar products. And by analyzing retrospectively in detail The causes of low blood pressure in a group

of 59 patients, Chestnut and al born find none cause of hypotension obvious For21 of them, and they believe that this hemodynamic failure is purely of central origin and then raises The question of The precocity of The introduction of catecholamines [62].

In the series of Benign, 12% of The patients had a hypotension [47]. And For

E. Tentillier et al 8% [58].

In a retrospective study by Van Haverbeke L et al [61], episodes of hypotension were noted at The house of 70 patients (i.e. 2.7 %).

In The study of F. Haddar [27], 2.4 % (either 246 patients) had a hypotension, SO that 88% presented a state stable hemodynamics has The entrance.

Regarding Mr. Ejjail's study [44], 60% of severe head trauma patients presented a hypotension requiring a expansion volemic immediate associated with administration of catecholamines (dopamine, adrenaline or noradrenaline) but never in first intention.

In our study, 5% or 15 patients had hypotension, SO that 75% presented a state stable hemodynamics at The entrance.

2.2.Arterial Hypertension:

Hypertension arterial must more often be respected,

Two cases can arise: HBP associated with bradycardia or HBP associated with tachycardia. • HBP associated with bradycardia is a sign of the severity of The brain trauma, it treatment is the same of The ICHT flare-up that it revealed.

Hypertension associated with tachycardia is rare when sedation and ventilation are properly assured. It is then The sign of an adrenergic discharge and may be self-limiting. When MAP is greater than 120 mmHg or that The PAS is higher than 180 mmHg, it is permissible to use an antihypertensive ifblood pressure did not return to normal spontaneously after 15 minutes. Nicardipine or Urapidil can be administered cautiously, in titrated boluses [63].

According to Lamiree Martin [64], among 268 patients, 258 or 96.27% presenteda stable hemodynamic state, only 10 patients or 3.73% presented a unstable hemodynamic state. B. Diarra noted a clear predominance of The state hemodynamics normal Who was found at The house of 89.9 %, However The statehemodynamics unstable is of 10.1 % [65]. F. Olatoundji has found that 647 (58.7%) patients were stable and 108 (14.3 %) sick was unstable [66].

In The series of F. Haddar [27], only 9.6% of patients had a hypertension to their admission.

In OUR series, 20% of The patients presented a hypertension at the admission.

3. Neurological State:

The neurological exam of a HT is primordial in case of Head trauma, it must be repeated and carried out meticulously, by assessing the level of awareness, the pupillary state and research of the localization signs [13].

We must not forget to search for the notion of immediate loss of awareness in place of accident, The free interval, the progressive orrapid neurological deterioration, The notion of convulsive crisis [67, 68], as well as The agitation which remains frequent during trauma, and which can have several causes (intoxication alcoholic, pain, hypoxia, etc.). It is not predictive of an injury intracerebral, but can be a demonstration of hypertension intracranial. Thissign must so be mentioned when he East found [22].

3.1.Glasgow coma Scale :

In 1974, The assessment and management of traumatic brain injuries were revolutionized by Teasdale

and Jennett, who created a notation system clinical has three components, known as The Glasgow coma scale [69].

It is the system of quoting THE more used to appreciate the level of awareness of The patients with a neurological achievement, mostly post traumatic [5, 70] provided that it is used in a very rigorous manner to preserve all its objective value. It is The result of 3 items: The response motor (side sure 6), there answer verbal (on 5) and The answer ocular side (out of 4).

Grace At GCS, we can distribute The HTs in 3 groups :

- A SHT is defined by A patient with a GCS less than 8/15
- A moderate HT corresponds to a GCS between 9 And 13
- A light HT is defined by a GCS of 14 Or of 15

Several authors have suggested of simplify THE GCS, Healey And al [77], has find on 20,000 patients that The motor component of The Glasgow score was also precise For predict The survival that The score of Glasgow total.

Meredith and al [78] has find that one threshold (notably there component motorGlasgow score less than 6) could predict mortality with a sensitivity of 59%And a specificity of 97%, However a comparison with the total scale of Glasgow hoes not been done.

Our results agree with those of the literature, with a predominance of the slices of patients with a GCS included between 9 and 13 either moderate HT.

A study recent in 2020 conducted by Lefevre C [128], has watch that there majorityTCs in The United States and in Europe was of The moderate HT.

In revenge a study done by Dewan M [127] on sixty-six country, has findthat 81% cases were light HT, 11% moderate and 8% severe.

3.2.Size And reactivity pupillary:

The exam of The pupils must to understand, there size, there symmetry and their responsiveness.

Unilateral or bilateral mydriasis observed in a HT may have several explanations, a mechanical compression of The third cranial pair during of a commitment temporal, but it can Also be explain by a drop of Speedblood flow to The brainstem, direct injury to The ocular nerve, lesions peduncular or brainstem disorders, metabolic or ischemic disorders, or an intoxication [79].

It is for this reason that the exam of the pupils is essential to the diagnosis and the prognosis.

Our study is consistent with observations reported in The literature, i.e. a predominance of normal pupils, because The majority of our patients are taken in charge in a stadium early.

3.3.Sign of focu:

The neurological examination must also systematically look for The presence of a sign of focus neurological. The element THE more fluently appreciated Eastthere motor skills of members.

3.4.Local exam:

It allow of to research the physical craniofacial signs, the scalp lesions and the orificials flows. In our series, We have note a clear predominance of scalp wounds found in 144 patients or 47.68%, followed by scalp hematoma found in 96 patients either 31.78%.

As for The orifice flows, we noted The predominance of otorrhagia found in 25 patients or 8.27%, followed by epistaxis and rhinorrhea found respectively in 15 patients or 4.96% and only 1 patient either 0.33%.

In The study of I. Sadeq [40], 8.44 % of The patients had a otorrhagia, 8.4%presented with epistaxis and 0.67% had rhinorrhea. S. Chouki [80] noted that 246 patients or 40.5% had scalp wounds, 225 cases or 37% had a scalp hematoma, 26 cases of otorrhagia or 4.3%, 24 cases of epistaxis or 4% and 13 cases of rhinorrhea or 2.1%. According to H. El Jaafari [71], otorrhagia was found in 21 cases (7.8%), epistaxis in 18 patients (6.7%) and rhinorrhea at a only one person.

3.5.Lesions associated:

Extracranial lesions must be systematically sought because they will play an important role in The management, in particular hypoxia which may be due to chest trauma or hypotension from shock hemorrhagic which are frequent in this context and which can be sources of aggravation of The lesions cerebral And Who darken considerably THE prognosis [62].

VII. BALANCE SHEET RADIOLOGICAL :

In The context of severe head trauma, The first question to be asked imaging early must answer East that of existence of a emergency neurosurgery. The medical-surgical team must confront The conditions of occurrence of trauma and The state clinic of The patient to The data radiological.

1. The standard X-ray:

In cranial traumatology, plain radiography has long served as a tool diagnostic main.

Currently, most teams rightly consider it useless due to its weak profitability diagnostic And The absence of a real value predictive, positive Ornegative [84].

The discovery of a fracture of The vault or base is a priori The witness of a trauma violent, susceptible to generate lesions intracranial, butX-ray has no predictive value as to to The coexistence of lesions cerebral [85, 86].

Overall, standard radiography is useless for brain exploration in THE Head trauma notably THE TCG And her interest himself limit has exploration ofcervical spine, thorax, pelvis and in case of suspected prophetic fracture in The polytrauma.

2. The cerebral Computed tomography.

Currently, the scan remains the most appropriate diagnostic means to offer urgently because of its speed and ease of access, even at acomatose patient and agitated. The examination will be performed without injection of product of contrast, by contiguous cuts of 5 to 9 mm thickness extending from the foramen magnum at vertex. More fines cuts will be carried out if request, depending on the images obtained. It does not seem desirable to make cuts straight awaycoronals looking for skull base lesions, as they require a mobilization of The head [6].

Early CT scan analysis can, in some cases, help to expect the presence of a deficit subsequent but cannot answer the crucial question of the return of consciousness [87].

CT scan in The initial phase (within The first 6 hours after CT) allow to establish A balance sheet lesional And of to set down THE indications surgical urgent.However, it is recognized that too early a CT scan may miss lesions. significant, even surgical. The second CT scan is more predictive of The become patients that The first, it is indicated in case :

- In THE 24 firsts hours above all if the first CT scan has been realized less then three hours after the trauma.
- During of the appearance of signs of deterioration clinical or in The absenceimprovement clinical.
- During an increase of there PEAK [81].

Ilker Solmaz's study involved 442 patients who all benefited from a TDM cerebral to their admission [75].
I. Sadeq [40] has note that there TDM cerebral has summer realized at The house of 308 patients on450 either 68.45% of The patients.
For The study realized At University Hospital Sfax [88], a TDM initial has summer realized at The house of98.1%.
During of her study, Hamdani has noted on THE 654 case observed that 144 patientsor 22.02% have do TDM [89].
Ouni has appreciated on 266 Numbers of Head trauma that 234 either 87.96%have benefited from CT [90].
K. ANIBA has counted In her work that 92.7% of The patients have benefitedbrain CT scan [29].
Mr. LAMIREE noted that 290 patients, or 73.98%, suffered head injuries.have benefited from brain CT scan [64].
For The study realized by G. Bouhours And al [35].
Initial computed tomography (CT) was performed in 89% of patients and repeated In The 24 hours at The house of 25%, And THE deadline AVERAGE of realization of there firstCT scan was 58 minutes after admission. For The study by Van Haverbeke et al. [61], A first exam by TDM cerebral has summer carried out at The house of 190 patients At courseof The first 24 hours (88.4% of cases). The other 25 patients died in The first hours of their care, before The examination is carried out by CT scan. And during The first 24 hours, a second CT scan was accomplished at The house of 58 patients (30.5 % of The case).
In The F. Haddar study [27], 88.5% or 9059 patients benefited from a brain CT scans upon their initial admission, several lesions were objectified isolated or in association.
As for our study, out of The 302 patients, 266 cases or 88% benefited from a scanner cerebral to their admission.

2.1.CT scan scale of classification:

Traumatic Coma Data Bank (TCDB) or Marshall's classification suggests a classification of TCs based on CT data. Its advantage is its simplicity and its validation on one of The largest recent series of patients. Intracranial lesions have been divided into two main categories: diffuse And THE lesions including a picture of high density of a volume superior25 ml (lesions of mass). THE lesions diffuse are classified in four stadiums according to the revisibility of The perimes encephalic cisterns and midline deviation. Mass lesions are divided into surgically evacuated lesions and non-surgically evacuated lesions. evacuated. THE prognosis clinical is closely related has the class CT scan patients.
There mortality of The patients having A trauma diffuse of kind 1 (TDM normal)is 10% and increases with The CT class. The mortality of patients with a lesion of surgically evacuated mass is between 40 and 50%. The diffuse lesionThe most common is type II lesion. In this category, The prognosis is strongly linked to The age of The patients. Recovery without sequelae or with sequelae moderate is observed in 39% of patients under 40 years of age, while it is only 8% for The others. In category IV, 75% of patients die or are in a state vegetative to the exit of The hospital [10, 91, 92].

2.2.THE CT scan lesions:

2.2.1. Extradural hematoma (EDH) :

Extradural hematoma results from The formation of a blood collection between The skull and The dura

mater, and most often associated with a fracture of The skull opposite. It is The consequence of an injury to an artery or veinmeningeal, more rarely of The breaking of a venous sinus.

Its diagnosis is CT-based and its presentation is that of a lens biconvex spontaneously hyperdense, GOOD limited, accompanied of a effect of masson The adjacent parenchyma. The constitution is generally rapid, even if The clinical decompensation may take several hours. However, The possible appearance delayed of this lesion, And that in particular at The house of THE children, imposed acareful interpretation of The images and repeating The examination when The realization of this one East very early [87, 93].

2.2.2. Acute subdural Hematoma (ASDH):

Resulting from lesions of small veins crossing The subarachnoid space, The subdural hematoma constitutes itself between The arachnoid And there dura mater. This collection is most often located at The level of The convexity of The cranial box [87]. The association of a subdural hematoma and a parenchymal contusionunderlying is very common, making The prognosis of subdural hematomas treble traumatic more severe that The one of The hematomas extradural. Her diagnosisrests on the realization of a scanner cerebral without injection of product of contrast, revealing a homogeneous biconcave hyperdense blade spread out in a crescent molding The surface of brain and less limit. The effect of mass on THE structures parenchymatous is often larger than The thickness would suggest The hematoma subdural: this testifies edema reactive to The contusion associate [10, 87].

He exists also a small blade subdural in interhemispheric posterior (arrow in b) as well as a small blade at The level of The floor of The fossa temporal and pericerebellar (arrows in a). The axial FLAIR slice visualizesby elsewhere A important erasure of The furrows cortical and of The homes hyperintense subcortical, particularly in The posterior regions, reflecting lesions of contusion cerebral.

2.2.3. Contusion cerebral :

Hemorrhagic contusions represent areas of brain destructionresulting from The direct impact of The brain against protruding parts of The structure bone of The cranium. For this reason, they are most often formed at The level of The frontal and temporal lobes. They are frequently associated withcontrecoup injuries. Hemorrhagic contusions consist of a core central hemorrhagic, hyperdense, surrounded of a area of fabric cerebral hypo infusedhypodense And has risk ischemic. In THE hours And days Who follow THE trauma himself shape A halo edematous pericontusional of mechanism ischemic (cytotoxic) And vasogenic. [87, 96, 97].

2.2.4. Cerebral Edema:

cerebral Edema is developed in the hours and days that follow the trauma. Whether vasogenic or ischemic (cytotoxic), it exposes to two major risks: that of displacement and engagement of brain structures in cases of intracranial pressure gradients and that of hypoperfusion and ischemia cerebral diffuse in the case the HIC is homogeneous. This last phenomenon translated to long term by cerebral atrophy. [87].

Its CT scan presentation associates a disappearance of The cisterns of The base And of The furrows cortical, a decrease of the size of The ventricles and, more rarely,a cortico-subcortical dedifferentiation. It diagnosis is Sometimes difficult at the young.

A good clue is The pinching of The ventricular frontal horns which are not Never virtual at The subject healthy [87].

2.2.5. Subarachnoid Hemorrhage:

The representation CT scan of subarachnoid hemorrhage (SAH) is that of a spontaneous homogeneous

hyperdensity of the subarachnoid spaces [101, 62]. It initial diagnosis on the CT scan holds a place in reason of role prognosis net so that its presence is not socket in account In there classification TCDB [8]. This hemorrhage aggravates THE prognosis, in determinant aischemia (Or The role of vasospasm seems important) [10].

A SAH is found In 14 % of The case After HT of all gravities [59] butup to 30% or even 40 % at The house of the serious HT [102, 103].

For be has The evidence of origin traumatic, She must predominate At level ofcerebral convexity. A hemorrhage of The basal cisterns must be put in cause THE diagnosis of trauma primary And must, if THE circumstances of The accidentare not clear, look for a rupture of a sub-vascular malformation adjacent (aneurysm) by an arteriography [87].

This is The depression of The cranial vault in relation to The impact. Lesions Skin lesions are often associated which can make clinical diagnosis difficult. Careful palpation of The skull looks for a pop (associated with exquisite painat The house of The conscious traumatized person [104].

The suspected diagnosis is confirmed by computed tomography examination in windows bone Who allow to analyze THE different fragments bony. We research SO particularly a splinter bone himself leader towards THE meningesand towards The brain parenchyma. And The parenchymal window looks for lesions associated.

THE embeddings closed, that's to say without wound cutaneous in glance, can beoperated delayed.

Surgical indication is given if there is a depression of The vault cranial compressing The cerebral cortex, if there is a bone splinter causing a cerebral cortical lesion. The indication also takes into account The location on an exposure area causing aesthetic damage. The wall jamb posterior frontal sinus should raise The possibility of communication between space endo sinus And endocranial with A risk infectious (meningitis) [104].

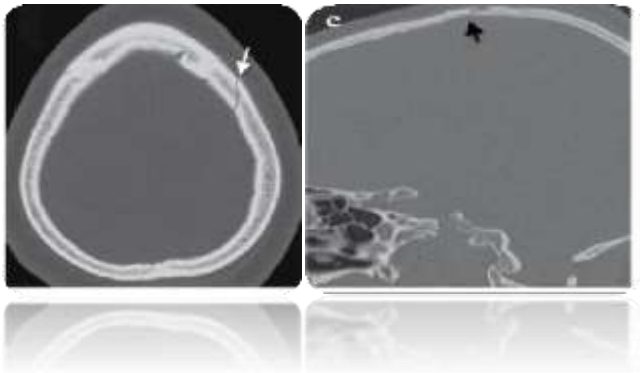

Figure 2 : Cuts CT scan in window bone showing a doorframe.

3. MRI magnetic resonance Imaging:

Brain MRI is still underutilized in trauma assessment severe cranial. Its certain difficulties of realization are however perfectly controllable by a team of radiology And of resuscitation GOOD trained. A few sequences fast (T1, Flair, T2) allow of realize A balance sheet lesional a lotmore accurate than that provided by brain scanning and to obtain an assessment prognosis precise of most of The patients [87].

Indeed, MRI can detect diffuse axonal lesions and should be realized at The house of all THE patients presenting A coma deep contrasting with The absenceof lesions on brain scan. Diffuse axonal lesions of The hemorrhagic typeare visible in MRI on THE sequences in echo of gradient in weighting T2*. They

appear in The form of a hyposignal that is all The more intense The closer one is todistance of trauma Who translated there presence of deoxyhemoglobin paramagnetic. MRI allow also of detect of The lesions axonal diffuseNo hemorrhagic [106]. These lesions can explain the disorders of there awareness observed After Head trauma despite A appearance normal At scannercerebral. Typically, these are hypersignals in FLAIR which are accompanied of hypersignals in broadcast (decrease of coefficient of broadcast) without anomaliesin T2* [107].

Of The sequences more recently developed (SRM, tensor of broadcast) allow From now on of realize A balance sheet extensive of The attack cerebral And shouldcontribute has impose this method imaging as there technical of reference ForThe assessment prognosis Head trauma severe [87].

Currently MRI East Above all used For there research of lesions anatomical in patients with neuropsychological sequelae, after a Head trauma, even moderate [108, 109, 110].

In OUR series, MRI has summer done uniquely at The house of ten patients in out of The EMERGENCIES seen there unavailability of plateau technical (respirator And scope compatible with MRI).

4. Balance sheet biological.

Biological assessment is not necessary in mild head trauma, it is by against systematic in case of severe HT. it understand :

- NFS + brochure
- Balance sheet of hemostasis
- Ionogram blood
- Gas from The blood
- Grouping + Rhesus.

Blood counts can detect anemia that constitutes a ACSOS that he will be necessary to correct For avoid ischemia cerebral. Hyperglycemia, hypernatremia and acidosis lead to ICH, requiring correct all disorders electrolytic [135].

In OUR series, all THE patients have benefited of a balance sheet biological.

VIII. THE TREATMENT :

There socket in charge of The traumatized cranial must to start on THE places evenof The accident. There precocity And there quality of there resuscitation would allow of reduce of significantly reduced HTmortality while careful conditioning by a trained team does not significantly delay arrival at The hospital [111]. The main objective of this resuscitation East there prevention of The ACSOS Above all hypotension And hypoxia Who are associated to a bad prognosis [92].

1. Medical Treatment:

The first therapeutic line has For goals an infusion cerebral adapted, maintaining correct tissue oxygenation associated with the reduction of cerebral oxygen consumption and finally strict metabolic control. The goals of the second line therapeutic being has the times of maintain a PEAKless than 20 mmHg and an adapted cerebral flow, the first treatments to be implementedin place must respect these objectives while having an acceptable tolerance. initiation of sedation, optimization of PPC or Osmotherapy are The treatments currently offered. The tolerance of these therapies depends on The times of ground of patient but Above all of The lesions cerebral And of The state of there barrierblood-

brain [87].

1.1.Conditioning :

The conditioning of a severe head injury victim involves The establishment of an arterial catheter and a central venous line. This allows for The detection and treat blood pressure drops quickly and measure blood pressure frequently gas blood.

The continuous monitoring of the saturation of hemoglobin in oxygen measured by pulse oximetry (SpO2), expired CO2 and core temperature and a hourly diuresis are also important elements [23] in addition to The implementation place of a peripheral venous line, a nasogastric tube and a probe urinary.

1.2.The control of there breathing :

In order to prevent The risk of inhalation, to avoid hypercapnia which can increase HIC and hypoxemia which can aggravate cerebral ischemia, The patient must be quickly intubated And artificially ventilated [87].

Recommendations for The management of GCTs [112,113] recommend for tracheal intubation in emergency an anesthetic fast induction sequence (Crush induction) with at best three medical professionals [114].

Rapid ventilation control is essential. Ventilation goals are to obtain an SpO2 ≥ 95% or a PaO2 ≥ 60 mmHg, and a normocapnia with a PaCO2 between 35 And 40 mmHg. Hyperventilation prophylactic deep(PaCO2 < 35 mmHg) should be avoided since it can compromise perfusion cerebral.

There ventilation mechanical (VM) East A AVERAGE therapeutic used For geta ventilation alveolar able to ensure THE maintenance of a oxygenation Cerebralsufficient [101, 115]. In A context of suffering Cerebral acute ; a VM Noadapted can be responsible of hypoxia, hyper Or hypocapnia deep, The origin of a worsening secondary of The lesions neurological susceptible to darken vital prognosis or functional [47, 51,116].

In Taleb Sid Ahmed's study, 28.5% of patients benefited from a intubation ventilation At service of CHU intensive care unit from Tlemcen [97].

For The work done by EL Jaafari H. showed that 15.24% of The whole of patients admitted to The study had received intubation ventilation at course from their hospitalization to service resuscitation [71].

Sadeq has watch that 27.25% of The whole of The patients admitted In The study done to services emergency And resuscitation had benefited of a intubation ventilationAt during their hospitalization [40].

In the studied series, all our patients have benefited of an artificial intubation and ventilation.

Painting XV : rate of patients intubated ventilated.

Authors	Intubation / ventilation
S. Taleb [97]	28.5%
EL Jaafari H [71]	15.24%
Sadeq [40]	27.25%
In OUR series	100.00%

1.3.Hemodynamics management:

An isolated HT is not a cause of cardiovascular collapse, hemodynamics instability must therefore lead to The search for either spinal cord injury or, more frequently, hypotension due to hemorrhage (scalp wound, etc.). Filling vascular is therefore a priority with The objective maintaining pressure systolic blood pressure ≥ 90 mm Hg, in case of active bleeding, and 120 mm Hg if The head trauma is isolated.

And a cerebral perfusion pressure of 70 mm Hg Thus than one transportation of adequate O2 [87, 40]. The solute of reference East THE serum dirty has 0.9%. It is A crystalloid which is iso-osmolar And therefore isotonic. These isotonic crystalloids are as effective as other solutions but require volumes 2 to 4 times larger. They diffuse quickly into THE fabric interstitial And provide a expansion volemic of 30% approximately [117]. The solutes hypotonic are outlawed in case of severe HT because risk to induce cerebral edema; as well as glucose solutions which are contraindicated in The firsts 48 hours post traumatic (risk acidosis cerebral) [118].
In The studied series, 76% or 229 patients benefited from volemic expansion has base of serum salted 0.9%.

Painting XVI : Distribution according to the control of the hemodynamics function

Authors	Filling	Catecholamines	Transfusion
Van Haverbeke and al [61]	98.1%	26.95%	_
Y. Coulibaly and al [76]	78.8%	_	_
University Hospital of Sfax [88]	_	6.7%	19%
G. Bouhours [35]	_	_	36%
OUR series	76%	11%	35%

1.4.Transfusion :
Low blood pressure most often results in a decrease in hematocrit, because of The fact of The losses blood and of hemodilution secondary to vascular filling. It is possible to transfuse group O rhesus blood negative in The absence of grouping.
In practice, The hemoglobin concentration will be maintained above 10 g/dl by transfusion of bases erythrocytes. This objective East important, This all The more sothat The lesions associated At trauma are often hemorrhagic [47].
In our series, transfusion was used in 35% of TCG patients, i.e. 105 patients.
In The study of Rouxel And al [36] 126 patients (44 %) had a Hb inferiorat 11 g/dl. In The work carried out by G. Bouhours [35], The use of transfusion haswas necessary in 36% of cases. And for The study of The Sfax University Hospital [88], 19% need a transfusion.

Painting XVII : Transfusion blood at The house of THE traumatized cranial.

Authors	Transfusion blood
Rouxel And al [36]	44%
G. Bouhours [35]	36%
Sfax University Hospital [88]	19%
In OUR study	35%

1.5.Sedation :
Sedation is justified by The need to ensure good cerebral relaxation in reducing THE needs metabolic cerebral, of allow a Good adaptationof The patient to mechanical ventilation and to control The phenomena of agitation and THE stimulations painful. THE agents used must decrease there PEAK,

decreasecerebral metabolism, respect The DSC/metabolism coupling, possess properties anti convulsive And must to present a half-life contextual shortin order to of allow there realization of reassessments neurological clinics [87].

The use of agents hypnotics (Propofol, Midazolam, Etomidate, barbiturates) allow a decrease of there PEAK in decreasing of way dose dependent brain consumption in oxygen.

These agents cause a decrease in CBF, VSC and therefore ICP while preserving THE coupling flow rate/metabolism, self-regulation And there reactivity of The CO2 vessels. However, The adrenal effects of Etomidate prohibit her administration extended. As to to barbiturates, THE risks hypotension at injection, immunosuppressive effects as well as half-life long contextual born allow not to consider their use of first intention [87].

Painting XVIII : cerebral Effects of The drugs used For the sedation of The cranial traumatized patients.

	PEAK	PPC	CMRO2	Antiepileptic	Prevention of The increase of there PEAK
Morphinics	= Or □	□	=	**0**	+
Benzodiazepines	= Or □	□	=	+	+
Propofol	= Or □	□	□	+	+
Barbiturates	□	□	□	+	+
Etomidate	□	=	□	**0**	+
Curares	= Or □	=	**0**	**0**	+
Ketamine	= Or □	=	=	**0**	+

THE recommendations current recommend The use of Midazolam inassociation with a morphine derivative. Sedation being prolonged with strongdoses, sufentanil is The most suitable morphine. In practice, and since The major reduction in The cost of Propofol, this is most often associated with The Midazolam, even at The house of of The patients presenting a PEAK controlled. This associationis synergistic and allow of reduce The doses of Midazolam.

This allows for more flexible management of sedation and provides The possibility of evaluations clinics more frequent [87]. THE appeal to curares, as to has him, mustbe limit at most And born himself designs that in case of syndrome of distress respiratory acute (SRDA) with pressures of ventilation No controlled Or if appear, despite the sedation, of The chills secondary has of The variations thermal And Who can be responsible of a increase brutal of the CMRO2. Curare is sometimes necessary if therapeutic hypothermia is used [87].

The procedures for stopping neurosedation, however, remain imprecise [119]. Final termination should be considered as soon as The brain-damaged patient meets certain conditions [120] :

- The absence of HIC Since more than 48 hours.
- The absence hypoperfusion cerebral estimated by the measure of The velocitiescerebral arteries by Doppler transcranial.
- The absence of aggravation of The brain damage.
- The absence of failure severe respiratory And hemodynamics.

- The stop of a possible administration of curares Since more of 24hours.
- Not of convulsion.
- Not of hypothermia.

In The series studied, sedation was systematic in all TCGs.maintained for at least 48 hours or until clinical signs disappear of HTIC.
THE Midazolam was used at The house of all THE patients preferably in associationto Fentanyl.
In The work carried out by Van Haverbeke et al [61], sedation was performed at The house of 188 patients (87.4 % cases), has help of hypnotics and of The morphine.
In The study of B. Aabydi [121], almost there totality of The sick have summer intubatedventilated (221 sick either In 98.22 %), with sedation during a duration average6 days.
In The study by F. Haddar [27] 19.05% benefited from sedation based onMidazolam And Fentanyl.
And In The study Of Z. Charani [28], all THE patients have suffered a sedation withan average duration of 5 days and The most used products are Fentanyl followed of Midazolam And thiopental.

Painting XIX : Distribution according to there realization of a sedation

Authors	Sedation	Products used
Van Haverbeke and al [61]	87.4%	Hypnotics And morphine
F. Haddar [27]	19.05%	Midazolam And Fentanyl
Z. Charani [28]	100%	Fentanyl follow up of Midazolam And of thiopental
B. Aabydi [121]	98.22%	-
OUR series	100%	Hypnovel And Fentanyl

2. Surgical management:

Surgery is actually uncommon in the acute phase, but conditions directly the prognosis. THE deadline between the trauma and The evacuation of a hematoma, at the patients that presenting a syndrome of commitment, is determinant [122].
Emergency neurosurgery can thus intervene on The front line in The resuscitation of severe head injuries in The initial phase and is discussed with The team neurosurgery.
Of The indications neurosurgical formal has there phase early have summer recognized :

- Focal lesions causing a midline deviation of more than 5 mm.
- Mass lesions with a volume >25 ml. Surgery may be indicated (even if The lesions have a volume of 5mm), if they cause aincrease in ICP >20 mm Hg or a decrease in CPP <70 mm Hg [123].
- Drainage of hydrocephalus acute ;
- THE trimming And closing of The embeddings open [5].

In our series only 28% or 85 hospitalized patients required a treatment surgical, of which 66% For evacuation of a hematoma extra dural Or of ahematoma under dural acute.

IX. EVOLUTION :

1. Favorable Evolution:

Roughly we distinguished two large issues, Either She East unfavorable (GOS1-2-3): this situation may be marked by The absence of return to consciousness orThe absence of reaction which is qualified as akinetic mutism before 18 months and a state vegetative persistent beyond 18 months.
Either The evolution is favorable (GOS 4-5) which results in a more or less long due date has a recovery satisfactory functionality allowing a activity socio-professional normal [59, 124].
This favorable development is marked by The improvement of clinical signs (hemodynamic, respiratory and neurological), by normalizing The signs organic (metabolic, gasometric) y Understood there sterilization of The samples bacteriological post therapeutic And there regression see there disappearance radiological lesions. And it is conditioned by The measures reducing The occurrence of ACSOS therefore The risk of cerebral ischemia causing The after-effects [125].
The evolution was favorable at The house of 145 of our patients either 48%.

2. THE complications :

2.1.Infectious complications:

We speak of nosocomial infection when this infection appears after The ^{48th} time of hospitalization and who was neither present nor incubating upon admission [105].

2.1.1. Nosocomial pneumonia:

THE patients put below ventilation mechanical have a higher risk of develop nosocomial pneumonia. They are mainly iatrogenic because of a part, due to lack of rigorous asepsis during manipulation of The endotracheal tube and during of the maneuvers of bronchial aspirations, and else share by default humidification of the air inspired, This Who disturbed the mucociliary function and favors bronchial congestion without forget the inhalation issue [121, 130].
The incidence of these pneumonias bacterial acquired below ventilation mechanics is very variable, from 3.5 to 84% in The literature, with an average of 30%. Mortality varies from
12 has 61 %. From a part, this difference East linked has there diversity of The patients studied,And else part, She East linked has there difference of The criteria diagnostics And bacteriological tests used [130].
In there series of G. Benhayoun [81], She has occurred at The house of 48 patients either 54
% with A rate of mortality reaching 48%.
In a study published in 2007, carried out at The Fez University Hospital, which involved 282 patients, there prevalence of The pneumonias nosocomial was of 11% And represented25% of infections acquired in The services resuscitation [132].
In The study by F. Haddar [27], 581 patients developed pneumonianosocomial either 5.6% of total of The TC.
In The study of M. Ejjail [44], it was quite frequent with a percentage of 46.66%.
In OUR study, we note 63 patients having developed a pneumonianosocomial, or 21% of The TCs in our study. This rate can be explained in our study of The complementary use of honey in medical treatment, and its effect antimicrobial demonstrated, by The action of peroxide of hydrogen, its large osmolarity, her acidity and certain substances No identified [133].

2.1.2. The urinary infection:

In most cases, urinary tract infection is linked to The introduction of a catheter In there bladder. THE catheterization bladder East A gesture inevitable In GOOD of The cases. A quarter of hospitalized patients have a bladder catheter for a period average of two days [131]. These infections have as consequences potentially serious pyelonephritis acute And The shock septic [131].

In OUR study, 14 patients have developed a infection urinary either 5%.
In there series of F. Haddar [27], 186 patients have developed a infectionurinary or 13.19% of The infections nosocomial found in his study.
In there series of S. Jaffel [134], 11.8% of The patients have developed a infectionurinary either 16 patients.
We can explain This rate down found In OUR study by THE respect of The rulesof asepsis within The department of surgical resuscitation.

2.2.The thromboembolic complications:

These are very serious complications that can be life-threatening.patients, hence The need to establish preventive treatment by means physical and medicinal depending on The clinical situations and The report risk/benefit.
In the studied series, 2 patients have developed a thromboembolic disease either less than 1% of our sick.

Painting XXI : The favorable evolution according to The literature

Authors	Country	Evolution favorable (%)
E Tentillier And al [58]	France	36
V. Haverbeke and al [61]	France	52.2
AR Aguèmon and al [47]	Benign	30
HAS. Ait Soltana [25]	Morocco	36
S. Belachi [34]	Morocco	47.2
Mr. Lamiree [64]	Madagascar	87
H. Khay [42]	Morocco	77.1
A. El Qadiri [54]	Morocco	61.3
In OUR series	Morocco	48

3. The mortality :

3.1.Overall mortality :

THE rate of mortality of The SHT has tendency has regress in the Westerners country and seems be improved by the progress realized In this domain. IT stay currently set at around 40%. Significantly lower figures have been published in different contexts. But it is likely that they reflect differences in recruitment from one study to another, difference in age distribution and difference in distribution of GCS more that of The differences In efficiency of socket in charge [57].
In a study by Ghajar J [15], in The United States, on The 1.6 million of victims of HT by year, we can count 52,000 dead.
There mortality at The house of THE TCG was of 77% either 40 patients.
There mortality at The house of THE traumatized cranial light And moderate was of 8% either 21patients.
Which means that The overall mortality rate in our series was 20% or 61patients.
That can be explain according to Read I [129], by The absence of administration ofneuroprotectors pharmacological.

3.2.Causes of death :

The worsening of The neurological condition following HTIC is responsible for death at 80%, followed infectious causes And hemodynamics.

In The B. Aabydi study [121] The main cause of death was The aggravation neurological status in 53.44%, followed by nosocomial infection in 41.37%deaths And In 5.1% by others causes including The ARDS and The shock hemorrhagic.

In The study of K. Aniba [29] The aggravation neurological constitutes there firstcause of mortality with a frequency of 86.5%.

In The series of H. Hadiri [74], mortality is also high, The main causes of death were distributed as follows: worsening of The neurological condition and engagement in 53%, then infection in 20% of cases, hemorrhagic shock in 18% of The case And Finally embolism greasy in 9% of case.

CONCLUSION

Every year, an estimated 69 million people worldwide will suffer from head trauma [127], The victims are most often young people, with a majority of head trauma moderate and light.

Grace to progress realized in matter of socket in charge, imaging And of careintensive, this has made it possible to considerably reduce The pronounced number of deaths, and disabilities resulting from traumatic brain injuries [15], this improvementEast due in large part has there acknowledgement And At treatment early of hypoperfusion cerebral.

Our study has permit of report The incidence of The HT at service of emergency surgical ICU At University Hospital Ibn Rochd of Casablanca And of to show their link narrow with THE accidents from The road public accident.

We have also demonstrated The importance of early nutrition in takingin charge of The head trauma victim.

We have put in evidence A rate pupil of mortality concerning The severe HT by several factors (transport often not medicalized, sometimes too long delays, delay of The taking in charge …), Who compete has the occurrence of secondary lesions rendering the darker the become of the patients.

Therefore, establishing a better relationship between The different services taking in charge THE traumatized cranial, must be favored for potentiateThe waking phases and early recovery, and to avoid complications (notably infectious in report with an extended stay in intensive care.

BIBLIOGRAPHIE

1. B Irthum, JJ Lemaire. Hypertension intracrânienne. Encyclopédie médico-chirurgicale 17-035-N-10
2. Le Ministère de l'Equipement et du Transport et de la logistique Recueil des Statistiques des Accidents Corporels de la Circulation routier 2013. http://www.mtpnet.gov.ma/
3. Comité de pilotage, Jehlé E, Honnart D, et al. Traumatisme crânien léger (score de Glasgow de 13 à 15) : triage, évaluation, examens complémentaires et prise en charge précoce chez le nouveau-né, l'enfantet l'adulte : Société française de médecine d'urgence. Ann Fr Med Urgence. 2012;2(3):199-214.doi:10.1007/s13341-012-0202-4
4. Proust F, Derrey S, Lavabre O, Castel H. Hypertension intracrânienne. EMC - Neurologie. 2012;9(2):1-14.
5. Bouhours G, Terminassian A, Beydon L. Traumatismes crâniens graves : prise en charge à la phase initiale.Réanimation. 2006 ;15(7-8) :552-560.
6. B Aesch, M Jan Traumatismes cranioencéphaliques. Encyclopédie médico-chirurgicale 17-585-A-

10 (2007).
7. Mrozek S, Srairi M, Geeraerts T. Traumatisme crânien grave à la phase aiguë. Journal Européen des Urgences et de Réanimation.2017 ;29(3) :241-254.
8. Alliez J.–R et al Hématomes intracrâniens post traumatique en phase aigue Encyclopédie médico-chirurgicale (Elsevier, Masson, SAS, Paris), Neurologie,17–585–A– 20, 2008
9. Capizzi A, Woo J, Verduzco-Gutierrez M. Traumatic Brain Injury. Medical Clinics of North America.2020 ;104(2) :213-238.
10. J. Albanèse, F. Portier, M. Léone Tomodensitométrie du traumatisme crânien Conférences d'actualisation 2000, p. 367–387
11. Hématomes intra crâniens - Campus de Neurochirurgie. Visitée le 6 mars 2021. http://campus.neurochirurgie.fr/spip.php?article419
12. Vega RA, Valadka AB. Natural History of Acute Subdural Hematoma.Neurosurgery Clinics of North America. 2017;28(2):247-255.
13. O. Mutschler, G Bouvard, P. Ravussin Concept d'agression cérébrale secondaire d'origine systémique Annales française d'anesthésie et réanimation 1995 ; | , 14 :114121
14. kamran samii Anesthésie-réanimation chirurgicale Flammarion Médecine–Sciences (2003) 1488pages
15. Ghajar J. Traumatic brain injury. The Lancet. 2000 ; 356(9233) :923-929.
16. J.-C. Orban, C. Ichai Hiérarchisation des traitementl'hypertension intracrânienne chez le traumatisé crânien grave. Annales Françaises d'Anesthésie et de Réanimation 26 (2007) 440–444.
17. Payen JF, Fauvage B, Falcon D, Lavagne P. Œdème cérébral par lésion de la barrière hématoencéphalique : mécanismes et diagnostic. Annales Françaises d'Anesthésie et de Réanimation.2003 ;22(3) :220-225.
18. Bruder N. Hémorragie sous-arachnoïdienne non anévrismale. Annales Françaises d'Anesthésie et de Réanimation.2007 ;26(11) :954-958.
19. Abdennour L, Puybasset L. La sédation-analgésie du patient cérébrolésé. Annales Françaises d'Anesthésie et de Réanimation.2008 ;27(7-8) :596-603.
20. Deplanque D. Physiopathologie de l'ischémie cérébrale. Encycl Méd Chir (Editions Scientifiques et Médicales Elsevier SAS, Paris, tousroits réservés), Neurologie, 17-045-A-80,2003, 10 p.
21. Payen D, Welschbillig S. Lésions ischémiques secondaires : comment les prévenir ? Annales Françaises d'Anesthésie et de Réanimation. 2007 ;26(10) :878-882.
22. K. Tazarourte, C. Macaine, H. Didane, H. Dékadjevi. Traumatisme crânien non grave.EMC 2007 25-200-C-10.
23. Nicolas Bruder Surveillance et traitement des patients traumatisés crâniens.EMC 1996 36-910-B-10
24. A. Chtouki Prise en charge du traumatisme crânien à l'hôpital provincial de Tétouan.Thèse de Médecine- Casablanca 1999, n°322
25. A. Ait soltana Les traumatismes Crâniens à l'hôpital provincial de Beni Mellal.Thèse de médecine - Casablanca 1997, n° 160
26. B. Diop Serigne Les traumatismes crâniens graves de l'enfant. Thèses de médecine – Casablanca 2001, n°114
27. F. Haddar Prise en charge des traumatismes crâniens au CHU Mohamed VI de Marrakech.Thèse de

médecine- Marrakech 2016, n°206
28. Z. Charani Prise en charge des traumatisés crâniens graves isoles dans les premières 24heures. Thèse soutenue à la faculté de médecine et de pharmacie de Fès N° 72/13
29. K. Aniba La prise en charge des traumatismes crâniens au CHU med VI de Marrakech, Thèse soutenue dans la faculté de médecine et de pharmacie de Casablanca N°136 en 2005
30. E. LeCorre Danniel Approche épidémiologique et itinéraire des traumatisés crâniens graves adultes dans le département du FINISTERE. Ann. Réadap. Méd. Phys., 1994, 34 : 335-347
31. L.J.J. Lawson The evaluation of transport safety. Rapport de transport Canada TP 10569, direction de l'évaluation économique :Mai 1989.
32. P. Skippen Effect of hyperventilation on regional cerebral blood flow in head injuredchildren. Crit Care Med. 1997 Aug ; 25(8) :1402-9.
33. Ponsford J, Whelan-Goodinson R, Bahar-Fuchs A. Alcohol and drug use following traumatic brain injury : A prospective study.Brain Injury. 2007 ;21(13-14) :1385-1392.
34. Belachi Sara Les facteurs pronostiques du traumatisme crânien gravent en réanimation Thèse soutenue à la faculté de médecine et de pharmacie de Fes N° 115 en 2013
35. G. Bouhours, T. Lehousse, J. Mylonas, G. Lacroix, C. Gondret, C. Savio, C.Couillard, L. Beydon
36. Evaluation de la régulation préhospitalière et prise en charge initiale destraumatisés crâniens graves dans la région des Pays-de-la-Loire. Annales Françaises d'Anesthésie et de Réanimation 27 (2008) 397–404
37. J.-P.M. Rouxel a, K. Tazarourte b, S. Le Moigno a, C. Ract a, B. Vigué Prise en charge préhospitalière des traumatisés crâniens Annales Françaises d'Anesthésie et de Réanimation 23 (2004) 6–14 A. Helmy, M. Vizcaychipi and A. K. Gupta Traumatic brain injury : intensive care management British Journal of Anaesthesia 99 (1) : 32–42 (2007)
38. M. A. Schreiber, N. Aoki, B. G. Scott, J. R. Beck. Determinants of mortality in patients with severe blunt head injury.Archives of Surgery, 2002, vol. 137, no 3, p. 285-290.
39. F. Bernard, J. Outtrim, D. K. Menon, B. F. Matta Incidence of adrenal insufficiency after severe traumatic brain injury variesaccording to definition used : clinical implications. British Journal of Anesthesia 96 (1) : 72 6 (2006).
40. Sadeq Imane. La prise e charge des traumatismes crâniens aux urgences du CHU IBNROCHD. Thèse soutenue à la faculté de médicine et de pharmacie de Casablanca N° 67en 2006
41. K. Stylianos Rapport d'un bureau de l'état civil sur le traumatisme crânioencéphalique enGrèce. Ecole de médecine à Athènes, 2010 :59-63.
42. H. Khay Les traumatismes crâniens dans la région de l'orientalThèse de médecine, Fès, Maroc : 29-2015
43. Peeters W, Majdan M, Brazinova A, Nieboer D. Changing Epidemiological Patterns in Traumatic Brain Injury : A LongitudinalHospital-Based Study in Belgium. Neuroepidemiology. 2017 ;48(1-2) :63-70.
44. M. Ejjail Epidémiologie et prise en charge des traumatismes crâniens graves isolés au service de réanimation polyvalente à l'hopital militaire Avicenne. Thèse de médecine- Marrakech 2015, n° 80
45. D. I. Graham, I. Ford, J. H. Adams, D. Doyle, G. M. Teasdalet, A. E. Lawrence, D. R. Mclellan Ischaemic brain damage is still common in fatal nonmissile head injury Journal of Neurology, Neurosurgery, and Psychiatry 1989 ;52 :346-350

46. J.-F. Chateil, B. Husson, M. Brun, F. MallemoucheImagerie des traumatismes crâniens chez l'enfant. EMC 31-622-A-20(2007).
47. A.R. Aguèmon a, J.L. Padonou b et al. Traumatismes crâniens graves en réanimation au Bénin de 1998 à 2002.Annales Françaises d'Anesthésie et de Réanimation 24 (2005) 36–39.
48. Oh Te, R. Hutchinson, S. Short, T. Buckley, E. Lin, D. Leung. Verification of the Acute Physiology and Chronic Health Evaluation scoringsystem in a Hong Kong Intensive care unit Critical care medicine, 1993, vol. 21, no 5, p. 698-705.
49. P. Gosling, S. Brudney, L. Mcgrath, S. Riseboro, M. Manji. Mortality prediction at admission to intensive care : a comparison ofmicroalbuminuria with acute physiology scores after 24 hours Critical care medicine, 2003, vol. 31, no 1, p. 98-103
50. J.I. Sánchez-Olmedo, J.M. Flores-Cordero, Rincón-Ferrari, M. Pérez-Alé,M.A. MuñozSánchez, J.M. Domínguez-Roldán, F. Murillo-Cabezas Brain Death After Severe Traumatic Brain Injury : The Role of SystemicSecondary Brain Insults. Transplantation Proceedings, 37, 1990–1992 (2005).
51. Kozlowski O., Pollez B., et al. Devenir et qualité de vie à trois ans dans une cohorte de patients traumatiséscrâniens graves. Ann de réadaptation et de méd physique 2002, 45, 8 : 466_73.
52. Langlois, J. A., Rutland-Brown, W., & Wald, M. M. The Epidemiology and Impact of Traumatic Brain Injury.Journal of Head Trauma Rehabilitation, 21(5), 375–378. 2016 ;33(22) :2000-2010.
53. F. Danze, M. Brule Evolution en centre de rééducation des comas post-traumatiques supérieurs ou égaux à 8 jours : 289 cas. Ann. Réadapt., Méd., 1994, 37 : 449-462.
54. A. El Qadiri Traumatisme crânien à l'hôpital provincial d'Agadir.Thèse Médecine., Casablanca 2001 n°300.
55. E. Wong, MK Leong, V. Anantharaman, TC. Chao Road traffic accident mortality in Singapore.J. Emerg Med ; Feb 2002, 22 (2) : 139-146.
56. Corrigan JD, Yang J, Singichetti B, Manchester K, Bogner J. Lifetime prevalence of traumatic brain injury with loss of consciousness.Injury Prevention. 2018 ;24(6) :396-404.
57. François Cohadon Les traumatismes crâniens, de l'accidet à la réinsertion2e édition Arnette 1vol. p 358
58. E. Tentillier a, M. Dupont a, et al. Description d'un protocole de prise en charge préhospitalière du traumatisécrânien grave. Annales Françaises d'Anesthésie et de Réanimation 23 (2004) 109–115.
59. Travis M. Dumont, Agostino J. Visioni, Anand I. Rughani, Bruce I. Tranmer, Bruce Crookes Inappropriate Prehospital Ventilation in Severe Traumatic Brain Injury Increases In-Hospital Mortality Journal of neurotrauma 27 :1233–1241 (july 2010)
60. V. Bhadri Narayan Intensive care management of the head injued patient. Indian journal of neurotrauma (INJT) 2006, vol.3, No.2, pp81=94
61. L. Van Haverbeke, S. Deraedt, B. T. Lemoine, J. Joly, J.J. Weiss, R. Fourgon, B. Trutt Traumatismes crâniens graves de l'adulte : prise en charge à la phase précoce enÎle-deFrance. Revue Médicale de l'Assurance Maladie volume 35 n° 1 / janvier-mars 2004.
62. R. M. Chesnut, T. Gautille, B. A. Blunt, M. R. Klauber, L. F. Marshall. Neurogenic hypotension in patients with severe head injuries. Journal of Trauma and Acute Care Surgery, 1998, vol. 44, no 6, p. 958-963

63. C Martini, R Domergue, et le groupe d'experts en médecine d'urgence du Sud-Est Les neurotraumatismes graves isolé. Prise en charge préhospitalière et au Sas d'urgence Annales Françaises d'Anesthésie et de Réanimation 1998 ; 17 : 1267-731
64. Lamiree Martin Sandrine Etude épidemio-clinique, para clinique et thérapeutique Du Traumatisme crânien au Cenhosoa. Madagascar ; Thèse de médecine, Antananarivo 2013, N° 8532.
65. B. Diarra Osmothérapie au mannitol chez les traumatisés crâniens à propos de79 cas admisau SUC de l'hôpital Gabriel Touré, Bamaki Thèse de médecine, 2003.
66. Fatigba O et Padonou J. Epidémiologie des traumatismes crânioencéphaliques à Parakou. BENIN Afr J Med Med Sci. 2010 ; 29 : 163-7.
67. N. Bruder, G. Boulard, P. Ravussin Surveillance et traitement des patients traumatisés crâniens Encycl. Méd. Chir., 1996 36-910-B-10.
68. N. Bruder, F. Gouvitsos Remplissage vasculaire au cours des 24 premières heures après traumatisme crânien grave Annales Françaises d'Anesthésie et de Réanimation 2000 ; 19 : 316-25
69. Gill M, Windemuth R, Steele R, Green SM. A comparison of the Glasgow Coma Scale score to simplified alternative scores for the prediction of traumatic brain injury outcomes Annals of Emergency Medicine. 2005 ;45(1) :37-42.
70. L. Kosakevitch-Ricbourg Traumatismes crâniens légers : définition, classificatons, pronosticRev Stomatol Chir Maxillofac 2006 ;107 :201-205 H. El Jaafari Prise en charge des traumatismes crâniens à l'hôpital Hassan II d'AgadirThèse de Med, Casablanca 2003, n°15.
71. J. Chazal, S. Puget, E. Schmidt, D. Sinardet Prise en charge du traumatisé crânien grave dans les 24 premières heures, dans le service des urgences, par la neurochirurgie. Annales Françaises d'Anesthésie et de Réanimation 2000 ; 19 : 299-303
72. R. Hani Prise en charge des traumatismes crâniens à l'hôpital Al Farabi d'OujdaThèse Méd, Casablanca, 2000, n 108. H. El Hadiri L'agression cérébrale secondaire d'origine systémique chez le traumatisécrânien. Thèse de pharmacie, Casablanca, 2007
73. Ilker S et Cahit K. Traumatisme crânien par balle à propos de 442 malades. Turquie: Gülhane Académie Médicale Militaire, Département de la Neurochirurgie, Ankara, 2009 ; 19 : 216-23.
74. Coulibaly Y. Le traumatisme crânien à l'hôpital du point G, à propos de 80 casMali médical : MAN. 2004 ; T XIX : 3-4.
75. Healey C, Osler TM, Rogers FB, et al. Improving the Glasgow Coma Scale score : motor score alone is a betterpredictor. J Trauma. 2003 ;54 :671-678.
76. Meredith W, Rutledge R, Hansen AR, et al. Field triage of trauma patients based upon the ability to follow commands : astudy in 29,573 patients. J Trauma. 1995 ;38 :129-135.
77. Dewitt DS, Jenkins LW, Prough DS. Enhanced vulnerability to secondary ischemic insults after experimentaltraumatic brain injury. New Horizons. 1995 ;3 :376-3
78. S. Chouki Traumatismes crâniens au sein du service de neurochirurgie à l'hôpital de Béni Mellal Thèse de médecine-Casablanca 2005, n°136.
79. Ghita Benhayoun. Le traumatisme crânien grave en réanimation.Thèse de Med, Casablanca 2000, n°144.
80. Y. Allali Facteur pronostic du traumatisme crânien grave.Thèse Méd, Casablanca 1997, n 155.
81. R. Eckhard Une étude de population basée sur épidémiologie, causes, traitement et résultat de tous

les degrés de sévérité de traumatisme crânien dans deux régions distinctes en Allemagne. 2010 :1-89.
82. L. Mailhan Qualité de vie après traumatisme crânien sévèrePratiques psychologiques 11 (2005) 343–357
83. ANAES. Prise en charge des traumatisés crâniens graves à la phase précoce.Recommandations pour la pratique clinique. Janvier1998 :109-15.
84. YS. Cordoliani, JL. Sarrazin, L. Hauret, P. Goasdoué, C. Lévèque, P.Sabbah Imagerie des traumatismes crânio-encéphaliques.Feuillets de radiologie 1999 ; 39 :242-59
85. V. Degos, T. Lescot, L. Abdennour, A.L. Boch, L. PuybassetSurveillance et réanimation des traumatisés crâniens graves Encyclopédie médico-chirurgicale 36-910-B-10
86. M. Bahloul, C. Ben Hamida, H. Chelly, A. Chaari, H. Kallel, H. Dammaka, N. Rekik, K. Bahloul, K. Ben Mahfoudh, M. Hachicha d, M. BouazizSevere head injury among children : Prognostic factors and outcome. Injury, Int. J. Care Injured (2008) 2008.04.018.
87. H. Hamdani Les traumatismes crâniens chez l'enfant (645 cas)Thèse de médecine : Casablanca. 1990
88. M. Ouni Les traumatismes crâniens chez l'enfantThèse en médecine 2009. Rabat. Réf m132, 2009.I.
89. K. Moppett Traumatic brain injury : assessment, resuscitation and early management.British Journal of Anaesthesia 99 (1) : 18–31 (2007).
90. Andrew I R Maas, Nino Stocchetti, Ross Bullock Moderate and severe traumatic brain injury in adultsLancet Neurol 2008 ; 7 : 728–41
91. Bruce Lee, Andrew Newberg Neuroimaging in Traumatic Brain Imaging The American Society for Experimental NeuroTherapeutics. Vol. 2, 372–383, April 2005
92. Seidou Guidah, Samuila Sanussi, B A. Abass, El Hadj Niang. APPORT DE L'IMAGERIE MEDICALE DANS LE BILAN DES TRAUMATISMES CRANIENS AU NIGER : à propos de 311 cas à l'hôpitalnational de Niamey. J Afr Imag Méd 2012 ; (4), 1 :4-11.
93. Jean-Louis Dietemann Neuro-imagerie diagnostique - 2rd Edition
94. D. A. Lang, D. M. Hadley, G. M. Teasdale, P. Macpherson, E. Teasdale Gadolinium DTPA Enhanced Magnetic Resonance Imaging in Acute HeadInjury Acta Neurochir (Wien) (1991) t09 :5-11
95. H. Kushi, Y. Katayama, T. Shibuya, T. Tsubokawa, T. Kuroha. Gadolinium DTPA-enhanced magnetic resonance imaging of erebral contusions. Acta Neurochir (Wien) 1994 ;60 :472-4.
96. MC Kinley Bruce A ; PH D Standardized management of intracranial pressure : A preliminary clinical trial.J Trauma, 1999 ; 46 (2) ; 271-279.
97. Taleb Sid Ahmed Prise en charge et devenir des traumatisés crâniens hospitalisés au CHU de Tlemcen en 2009 et 2010. Tlemcen : Universite aboubekr belkaid. Faculté de Médecine B. Benzerdjeb. Thèse Médecine.2014
98. J. EL Houdzi Prise en charge des traumatismes crâniens à l'hôpital Ibn Tofail de Marrakech.Thèse. Méd. Casablanca, 1998, n 110.
99. A. Ter Minassian. Traitement de l'œdème cérébral. Annales françaises d'Anesthésie et de Réanimation 22 (2003) 336 348.

100. H. M. Eisenberg, H. E. Gary, E. F. Aldrich, C. Saydjari, B. Turner, M. A.Foulkes, J. A. Jane, A Marmarou, L. F. Marshall, H. F. Young. Initial CT findings in 753 patients with severe head injury A report from the NIH Traumatic Coma Data Bank J Neurosurg 73 :688-698, 1990
101. K. A. Greene, F. Marciano, B. A. Johnson, R. Jacobowitz, R. F. Spetzler, T. R. Harrington. Impact of traumatic subarachnoid hemorrhage on outcome in nonpenetrating head injury J Neurosurg 83 :445–452, 1995
102. Laurent Riffaud Traumatismes cranio-encephaliques Service de Neurochirurgie, CHU de Rennes
103. H. Kallel, H. Chelly, M. Ghorbel, M. Bahloul, H. Ksibi, N. Rekik, H. Ben Mansour, M. Bouaziz La méningite post-traumatique : incidence, microbiologie et pronostic.Neurochirurgie, 2006, 52, n° 5, 397-406
104. K. Paterakis, A. H. Karantanas, A. Komnos, Z. Volikas. Outcome of patients with diffuse axonal injury : the significance and prognosticvalue of MRI in the acute phase. J Trauma 2000 ;49 :1071-5.
105. Amon Y. Liu, Joseph A. Maldjian, Linda J. Bagley, Grant P. Sinson, and Robert I. Grossman Traumatic Brain Injury : Diffusion-Weighted MR Imaging Findings AJNR Am J Neuroradiol 20 :1636–1641, October 1999
106. DD. Blatter, ED. Bigler, SD. Gale et al. MR-based brain and cerebrospinal fluid measurement after traumatic braininjury : Correlation with neuropsychological outcome.Am J Neuroradiol 1997 ; 18 : 1-10.
107. JT. Wilson, KD. Wiedmann, B. Condon, G. Teasdale, DG. Brooks Early and late magnetic resonance imaging and neuropsychological outcome after head injury. J Neurol Neurosurg Psychiatry 1988 ; 51 : 391-396.
108. D. Plantier, E. Bussy, A. Rimbot, P. Maszelin, H. Tournebise La neuro-imagerie après traumatisme crânien léger : mise au point et recommandations pratiques. Rev Stomatol Chir Maxillofac 2006 ; 107 :218- 232
109. Y Matillon Recommandations pour la pratique clinique Prise en charge des traumatises raniens graves a la phase précoceL'Agence Nationale d'Accréditation et d'Évaluation en Santé
110. A. Robin, C. Kiefer, K. Cochepin-Martins. Troubles psychiques des traumatisés crâniens sévères. EMC (2008)37-546-A-10
111. Abdennoura L., T. Lescota, et al. Traumatisés crâniens graves : jusqu'où aller ? Annales Françaises d'Anesthésie et de Réanimation 26 (2007) 445–451
112. K. Tazarourte, O. Kleitz, S. Laribi, B. Vigué.Prise en charge des traumatisés crâniens graves.EMC 2007 MC 25-200-C-20.
113. A. Pasco-Papon a, b, D. Darabi a, et al. Apports de l'IRM conventionnelle et de l'IRM de diffusion à la phase aiguë dutraumatisme crânien grave. Annales Françaises d'Anesthésie et de Réanimation 24 (2005) 510–515
114. J. S. David, M.P. Cresta', et al. Traumatismes crâniens graves : effets de la ventilation mécanique Préhospitaliére sur la capnie. Annales Françaises d'Anesthésie et deRéanimation 1999, 17, 398,402.
115. A. Soummer, O. Langeron Conduite à tenir devant un état de choc Encyclopédie médico-chirurgicale 2 (2005) 245–252
116. J.P Bleichner et al. Evaluation de la restriction glucidique dans la prévention de l'hyperglycémie chez le traumatisé crânien grave, risque hypoglycémique. JEUR. 1996 ;9 :126-30.
117. R.L. Chioléro, P. Schoettker Arrêt de la sédation en neuroréanimation Annales Françaises

d'Anesthésie et de Réanimation 23 (2004) 541–545
118. J. F. Payen, G. Francony, C. Canet, F. Coppo, B. Fauvage Neurosédation en réanimation Annales Françaises d'Anesthésie et de Réanimation 28 (2009) 1015–101
119. Bouchra Aabydi L'intérêt des scores de gravite non spécifiques dans l'évaluation du devenir du traumatisme crânien grave Thèse soutenue à la faculté de médecine et de pharmacie de Marrakech N° 23en 2010
120. F. Lagneau Indications et utilisation des curares en réanimation. Annales Françaises d'Anesthésie et de Réanimation 27 (2008) 567–573.
121. Bullock R ; Chesnut RM : 9. Guidelines for the management of sever head injury.New york, brain foundation 1995
122. Mathieu MARTIN Fréquence et facteurs de risque de l'hypertension intracrânienne à la phase précoce du traumatisme crânien grave Mémoire pour l'obtention du DES d'Anesthésie-Réanimation, UniversitéPierre et Marie Curie (2012)
123. C Marescal, P Adnet, N Bello, I Halle, AP Forget, P Boittiaux Agressions cérébrales secondaires d'origine systémique chez les enfantstraumatisés craniocérébraux graves. Annales Françaises d'Anesthésie et de Réanimation 1998 ; 1 I : 234-9.
124. Dewan MC, Rattani A, Gupta S, et al. Estimating the global incidence of traumatic brain injury. Journal of Neurosurgery. 2019 ;130(4) :1080-1097.
125. Lefevre-Dognin C, Cogné M, Perdrieau V, Granger A, Heslot C, AzouviP. Definition and epidemiology of mild traumatic brain injury. Neurochirurgie. Published online May 2020 : S0028377020300606.
126. Lu J, Marmarou A, Choi S, Maas A, Murray G, Steyerberg E.Mortality from traumatic brain injury. Intracranial Pressure and Brain Monitoring XII. Vol 95. Acta Neurochirurgica Supplementum. Springer Vienna ; 2005 :281-285.
127. 5e Conférence de Consensus en Réanimation et Médecine d'Urgence Paris,le 13 octobre 1989 Diagnostic des pneumopathies nosocomiales en réanimation
128. Léone M, Arnaud S, Boisson C, Blanc-Bimar MC, Martin C. Infections urinaires nosocomiales sur sonde en réanimation : physiopathologie, épidémiologie et prophylaxie. Annales Françaises d'Anesthésie et de Réanimation. 2000 ;19(1) :23-34.
129. El Rhazi K, et al. Prévalence et facteurs de risque des infections nosocomiales au CHU Hassan II de Fès. Eastern Mediterranean Health Journal. 2007 ; 19(5) : 256-264.
130. Wahdan HAL. Causes of the antimicrobial activity of honey.Infection. 1998;26(1):26-31.
131. S. Jaffel, B. Mahdi, L. Thabet, M. Boussofara Les infections nosocomiales chez les traumatisés en réanimation. 18 ème journées nationales d'infectiologie/ Médecine et maladies infectieuses2017.
132. O. Moeschler, G. Boulard, and P. Ravussin, "Concept d'agression cerebrale secondaire d'origine systemique (ACSOS)Secondary systemic insults to the brain,"Ann Fr Anesth Roanim, vol. 14, pp. 114–121, 1995.