COURSE GUIDE

PHS 403
ACCIDENT AND EMERGENCY

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MODULE1 ACCIDENTS AND EMERGENCY

UNIT 1 INTRODUCTION

1.0 INTRODUCTION

This is also known as emergency department(ED),Emergency Room (ER), or Casuality Department. This unit will assist you acquire the basic understanding of accident and emergency and related issues.

2.0 OBJECTIVES

By the end of this unit ,you should be able to:

- define accident and emergency
- know what is triage in emergency
- know the entry points of some categories of some patients
- know how to handle patients with serious conditions
- describe resuscitation area and play therapist.

3.0 MAIN CONTENT

3.1 Definition of A&E

Accident and Emergency(A&E) also known as Emergency Department(ED), Emergency Room(ER), or casualty department, is a medical treatment facility

specialising in emergency medicine ,that is, acute care of patients who present without prior appointment, either by their own means or by ambulance. The emergency department is usually found in a hospital or other primary care center. Due to the unplanned nature of patient attendance, the department must provide initial treatment for a broad spectrum of illnesses and injuries, some of which may life-threatening and require immediate attention. In some countries, emergency departments have become important entry points for those without other means of access to medical care.

3.2 Triage

Today, a typical hospital has its emergency department in its own section with its owndedicated entrance. As patients can present at any time and with any complaints, a key part of the operation of any emergency department is the prioritisation of cases based on clinical need, this process is called Triage. Triage is normally the first stage the patient passes through, and consists of a brief assessment, including a set of vital signs, and the assignment of a chief complaint (eg, chest pain, abdominal pain, difficult breathing, etc). Most emergency departments have a dedicated area for this process to take place, and may have staff dedicated to performing nothing but to triage role. In most departments, this role is fulfilled by a nurse , community health practitioner,although, dependent on training levels in the country and area, other healthcare professionals may perform the triage sorting ,including the paramedics or physicians.

Triage is typically conducted face-to-face when the patient is present, or a form of triage may be conducted via radio with an ambulance crew, in this method, the paramedics will call the hospital‘s triage center with a short update about an incoming patient, who will then be triaged to the appropriate level of care. Most patients will be initially assessed at triage and then passed to another area of the department, or another area of the hospital, with their waiting time determined by their clinical need. However, some patients may complete their treatment at the triage stage, for instance, if the condition is very minor and can be treated quickly, if only advice is required, or if the emergency department is not a suitable point of care for the patient. Conversely, patients with evidently serious condition, such as cardiac arrest, will bypass triage altogether and move straight to the appropriate part of the department.

3.3 Resuscitation Area

This is commonly referred as Trauma or Resus, is a key area in most departments The most seriously ill or injured patients will be dealt with in this area, as it contains the equipment and staff required for dealing with immediately life-threatening illnesses and injuries. Typically, resuscitation staffing involves at least one attending physician, and at least one and usually two nurses or community health officers with trauma and advanced cardiac life support training.

Patients whose conditions are not immediately life-threatening will be sent to an area suitable to deal with them, and these cases might typically be termed as a prompt care or minor area, such patients may still have been found to have significant problem including fractures, dislocation, lacerations requiring suturing.

3.4 Play Therapist

Children can present particular challenges in treatment. Some departments have dedicated Paediatric areas, and some department employ a Play therapist whose job is to put children at ease to reduce the anxiety caused by visiting the emergency departments, as well as provide distraction therapy for simple procedures.Many hospitals have a separate area for evaluation of psychiatric problem. These are often staffed by psychiatrists and mental health nurses and social workers. There is typically at least one room for people who are actively at risk to themselves or others (eg suicide).

4.0 CONCLUSION

An emergency department, also known as Accident and Emergency, Emergency Room or Casuality Department is a medical treatment facility specialising in emergency medicine, that is, acute care of patients: Treating serious injuries, accidents or sudden illnesses, using a priority system where the most seriously ill patients are seen first, a frontline venue for the delivery of emergent medical care. You can now explain what accident and emergency is, describe Triage, Resuscitation and Play therapist in your own words and even explain them to others.

5.0 SUMMARY

6.0 TUTOR-MARKED ASSIGNMENT

(a) Define accident and emergency.

(b) Explain the term Triage.

(c) Describe Resuscitation area and Play therapist.

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UNIT 2 ACCIDENTS

CONTENTS

1.0 INTRODUCTION

Accident is an unfortunate incident that happens unexpectedly and unintentionally, typically resulting in damage or injury or harm. This unit will assist youacquire the basic understanding of accident and injury related issues.The unit's objectives below will also give you a better guide on what anaccident is.

2.0 OBJECTIVES

At the end of this unit, you should be able to:

- define the term accident
- differentiate between accident,trauma and injury
- classify injuries
- define road traffic accident
- discuss the preventive measures on accident and injuries.

3.0 MAIN CONTENT

3.1 Definition

Accident is an unfortunate incident that happens unexpectedly and unintentionally, typically resulting in damage or injury or harm, loss, casuality, mishap, fatality. It is synonymous to mishap, misfortune, misadventure, mischance, injury, disaster, tragedy,

catastrophe, calamity, blow trouble, it can as well be defined as an event that happens by chance or that is without apparent or deliberate cause. While emergency is a serious, unexpected, and often dangerous situation requiring immediate action.

3.1.1 Kinetics/ Kinds of Trauma

Trauma results from the collision of two or more bodies in motion. As a result the study of trauma is related to a branch of physics known as Kinetics. To grasp the kinetics of trauma , you need to understand two basic principles of physics- the law of inertia and the law of conservation of energy. Application of these two principles can help you to identify the mechanism of injury and the probable effects of Impact, or Collision, upon the victim or victims. A grasp of these principles can help you better analysetrauma .
When kinetic energy is to human anatomy, it is called TRAUMA. Trauma is defined as a wound or injury that is externally or violently produced by some outside force. The wound may either be blunt (closed) or penetrating (open).

Blunt trauma occurs when a body area is struck by, or strikes, an object. The transmission of the energy, rather than the object, damages the tissues or organs beneath the skin as they collide with each other. An example of this is hitting your thumb with a hammer. The is compressed between the hammer (which pushes the tissues) and the board (which resists the motion). Tissue injury results as flesh and bone become trapped between these two forces(acceleration and deceleration). Muscle cells stretch, blood vessels tear, and become may fracture.Blunt trauma can also induce internal injury deep within the body cavity. Forces of compression cause hollow organs like the bladder or bowel to rupture, spilling their contents and haemorrhaging. In the thorax, alveoli, or small airways may burst, permitting air to enter the pleural space. Solid organs, such as the spleen, liver and kidney, contuse or lacerate, leading to swelling, blood loss, or both.

Further, trauma may result from the effects of rapid speed change and organ attachment. An example includes the liver, which is suspended the ligamentumteres. During severe deceleration, the liver may be sliced by the ligament similar to cheese when cut by a wire cheese cutter. Similarly, the aorta may be injured as the chest slows and the heart, which is suspended from this great vessel, twists upon impact. Layers of the vessel are torn apart, and blood enters the injury with the force of the systolic blood pressure. The aorta balloons like a defective tire, leading to a tearing chest pain, circulatory compromise, and immediate, or delayed exsanguinations (severe blood loss).

Wounds that break the skin are classified as penetrating trauma. Penetrating trauma occurs when the energy source(such as a knife) progresses into the body. Energy may also be transmitted to surrounding body tissues, thus extending the trauma beyond the open wound. This frequently happens with gunshot wounds. Blunt trauma most commonly results from motor vehicle accidents involving automobiles, motorcycles,

pedestrians, or recreational vehicles. It also can be caused by falls, sports injuries, and blasts.
Events of Impacts: There are basically five vehicle impacts- frontal, lateral, rotational, rear-end, and rollover. Each progresses through a series of four events. These events include : Vehicle collision, body collision, Organ collision and secondary collisions.

3.1.2 Classification of Injuries

The emphasis is now more on the outcome of the event (i.e. injury) than the event itself . Injuries are broadly divided into two main categories:

Unintentional injuries

These are subdivided into:

(a) Road traffic injuries
(b) Poisoning
(c) Falls
(d) Burns
(e) Drowning and "other unintentional injuries" e.g. exposure toanimate and inanimate mechanical forces like firearms, electriccurrent, radiation and extreme ambient temperature, pressures,forces of nature, contact with heat and hot substances, venomous plants and animals.

Intentional injuries
These are subdivided into self-inflicted injuries (i.e. suicide),interpersonal violence (e.g. homicide), war-related injuries, and "otherintentional injuries" (includes injuries due to legal intervention).

3.1.3 The Burden of Injuries

Injuries accounted for 12% of the global burden of disease and 90% ofdeaths in the year 2002. As projected, road traffic injuries will by 2020move from the 9th position to the 3rd position as the leading cause ofglobal disease burden (WHO Global Burden of Disease Project, 2002).In many parts of the world, injury related database is thin and the realburden may be heavier than the estimate. For every injury-related death,several thousand more require hospital treatment; and victims sufferwith impairments, frequently with disabling consequences.

Injuries by age and sex
Injury mortality rate among men is twice as high as that concerningwomen. In some regions, however, mortality rates for suicide and burnsin females are as high as or even higher than in males. Males in Africaand Europe have the highest injury-related mortality

rates.Young people between the ages of 15 and 44 years account for almost 50% ofthe world's injury-related mortality. Mortality from road traffic injuriesand interpersonal violence in males is almost 3 times higher than that infemales. Children under five years of age account for approximately 25%of drowning deaths, and 15% of fire-related deaths worldwide.

3.1.4 Epidemiological Considerations

Probability of occurrence of an injury and its severity primarily depends upon the agent, reservoir, vector, host, response, environment and other risk factors.

Agent
That agent is energy. Injuries are seen in a variety of different physicalways. Energy can be delivered in a way as to cause blunt trauma totissues or organs; or it can be in form of projectiles, which may producepenetrating wounds. It may also exist in the form of mechanical,electrical, chemical, radiation and thermal force.

Reservoir
The reservoir is the place where the agent is usually found. For example,generators/power houses are the reservoirs of man-made electricity;petrol/diesel is a reservoir of energy converted by vehicles into kineticenergy.

Vectors
These are inanimate objects like motor vehicles, bullets, cigarettes,flammable cloths, etc, that transfer the energy from its reservoir topotential or actual host. An exposure may or may not result in injury,depending upon whether the amount of contact between a susceptiblehost and the energy involved is outside the band of tissue tolerance. Ahuman being or animal can also exert mechanical energy, hencebecoming an animate object or a vector of energy e.g. the kick of ahorse.

Host response
There are limits beyond which energy delivered to the host can beabsorbed or tolerated. The host response depends upon its age, medicalcondition, diet, physical conditions, etc. Males are as much at risk asfemales of being victims of an accident. Young males are at maximum risk of injury while the case fatality following an injury is highest in the elderly.

Environmental factors
Poor road conditions, inadequate public transport system, excessive heatand cold, poor illumination of road or work place, poor enforcement oflaw, lack of devices, easyavailability of poisonous and hazardoussubstances, overpopulation, illiteracy,

prevalence of stray animals onroad are some of the environmental factors contributing to injuries

Risk factors

Risk factors may be causative (exposure to hot fluids in burns) orcontributory (driver fatigue in a road injury). They may be modifiable(speed of a vehicle) or non-modifiable (age and sex of the victim).Application of the "risk concept" can help identify individuals who areprone to a particular type of injury by virtue of having one or more riskfactors.A cluster of subjects with similar risk factors constitute a "risk group".This group is vulnerable to a particular injury and need to be givenpriority in prevention programmes.

3.1.5 Prevention of Injuries

Persons are injured on roadways, in work places, in homes, and duringleisure activities. No group within a society is spared, although some areat more risk than others. A significant injury can change the lives ofvictims and other family members, often permanently. It is thusimportant to prevent injuries.

Primordial and primary prevention

Efforts are directed to remove the circumstances leading to injuries. Theusual strategies consist of IEC (Information, Education andCommunication) activities on prevention of injuries and specificenvironmental modification geared to protect people from injuries.

Secondary prevention

Immediate care is needed once an injury has occurred. Immediateresuscitation, first aid triage and quick referral, as well as early hospitalcare are the cornerstone of secondary prevention. This is aimed atreducing the severity and complication of injuries, as well as preventingdeath.

Tertiary prevention

This is aimed to reduce the long-term disabilities by physical andpsychological rehabilitation of the injured and restoration of bodilyfunctions to the maximum extent possible.

Road traffic accidents (Injuries)

Road traffic accidents are crashes originating, terminating, or involvinga vehicle partially or fully on a public highway.

Magnitude of the problem

Globally, road traffic crashes account for one-fourth of the total injurydeaths, killing 1.2 million persons per annum. These deaths are expectedto increase to 2.3 million (rise of over 85 %) by 2020 with more than 90% of these deaths occurring in low and middle income countries (WHO,2004).

Risk factors
There are many risk factors associated with road traffic accidentsworldwide; however, some are discussed below.

Motor vehicle
Growing number of motor vehicle is one of the main factorscontributing to the increase in global road crash injury.

Speed
The speed of a vehicle is controlled by factors related to road, vehicle,traffic, environment, driver and the occupants in the vehicle.

Driver factors
Alcohol concentration of more than 0.04g/dl in the blood of the driver isassociated with significant risk of crash. Fatigue or deprivation of sleep,use of hand-held mobile phones while driving etc. are also responsiblefor road crashes.

Environment Factors
The traffic on most roads is a mix of slow (pedestrians, cycles, bullocksand mopeds) and fast vehicles (motorcycles, vans, cars, trucks andbuses). There are no lanes dedicated to a particular kind of traffic evenin bigger metropolitan cities, inadequate visibility due to weathercondition is also a major environmental risk factor.

Preventing road traffic accidents
Road traffic injury should be considered alongside heart disease, cancerand stroke as a public health problem; appropriate intervention that canprevent much of it from occurring.

Managing exposure
Eliminating the need or desire to travel is not possible, but length andintensity of exposure to types of road traffic that put people at risk canbe reduced.To achieve this, the places where people live, work and relax shouldentail minimum travel. Unnecessary trips should be discouraged. Someof the following should be adopted towards managing exposure to trafficaccidents:Provide shorter, safer routes for vulnerable roadusers.
Encourage the use of safer modes of travel such as affordable publictransport, by rail and/or bus and coach,segregating slow and fast moving traffic and regulating motor vehicle use by young riders and drivers.

Preventing crashes
This can be done by better designing roads (having a hierarchy of highspeed highways, transitional roads, rural roads and residential roads);insisting on crash protective road sides, promoting lane driving andenforcing road safety rules with respect to speed and

alcohol limits,mandatory use of seat belts and helmets; and improving safetyawareness among road users.

Preventing injury in event of a crash
The ultimate option is to design a "smart" vehicle that can sense andcombat danger of crash, vehicles should be designed to withstandcrashes and minimise injury to the occupants. The vehicle should haveseat belts, frontal and side impact protection bars; the passengercompartment should not collapse in a crash and be devoid of elementsthat can cause injury.

Delivery care after crash
The aims of care after crashes are to avoid death and disability, to limitthe severity and suffering caused by the injury and to ensure optimalfunctioning of the crash survivors and reintegration into the community.

4.0 CONCLUSION

In this unit, you have learned that accident is an unfortunate incident that happens unexpectedly and unintentionally, typically resulting in damage or injury or harm, loss, casualty, mishap, fatality while, trauma is a wound or injury that is externally or violently produced by some outside force. You have also been told what impact and events are. You have also realised that injuries are classified asunintentional and intentional. You should at this point be able to explainin your own words, trauma,impacts, events, road traffic accidents/injuries and how to preventroad traffic accidents/injuries.

5.0 SUMMARY

This unit has been able to define the term accident, which is traditionallyreferred to as unintentional, unplanned event.Also, the unit has classified injuries, the burden of injuries and itsepidemiological considerations, the prevention of road trafficinjuries/accidents on our roads.

6.0 TUTOR-MARKED ASSIGNMENT

(1) Define the term trauma.
(2) Mention five types of impact.
(3) Give a brief explanation of the term injury.
(4) Explain the levels of injury prevention methods.
(5) Define the term road traffic accidents.

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UNIT 3 EMERGENCY MEDICAL SERVICES(EMS) SYSTEMS

CONTENTS

1.0 INTRODUCTION

The emergency medical services(EMS) systems is a complex health care system made up of personnel, equipment and resources. The EMS system has several components. The prehospital component includes: Lay persons, the first Responder , the emergency medical technician and the paramedic. The hospital component includes: The emergency department nurse, the emergency physician and the speciality physicians(eg: trauma surgeons, cardiologists). Support personnel also help the EMS system operate smoothly. In the hospital phase, they include EMS dispatchers, law enforcement personnel, firefighters, and other public-safety workers. In the hospital respiratory therapists, radiologic technicians, and other specialists provide important support services.

All components of the system must work together to assure quality patient care. Usually, the first EMS person to respond to medical emergency is the first Responder. The first Responder may be a Police officer, firefighter or lay person who has received basic emergency medical training in an approved First Responder Program. First Responders are trained in basic airway management and other basic skills. The next component is the EMT. The EMT may respond in a fire department vehicle or in an ambulance. He or She should either continue the stabilisation started by the first Responder or initiate life support measures. The next component, the paramedic, should provide advance life support care, if indicated. Upon arrival at the hospital emergency department, patients are usually assigned priorities for care by a nurse or physician. If needed, a surgeon or other specialist will be summoned to the emergency department.

2.0 OBJECTIVES

At the end of this unit , you should be able to :

- describe the general development of ems system
- list and define the components of an ems system
- explain the oversight duties of an ems administrative agency
- discuss the responsibilities of the physician medical director regarding direct-line and indirect-line medical control
- describe public involvement in an ems system, with regard to system access, recognition of emergency and initiation of basic life support
- describe the components of an effective medical and operational communications system
- describe the components of emergency medical dispatching, system status management, interrogation guidelines, response protocols, pre-arrival instructions and dispatcher training
- describe the use of patient transfer protocols for ground and air transport services
- describe the importance of quality evaluation in ems, and discuss the similarities and differences between quality assurrance and quality improvement programs
- discuss the value of research in ems
- describe the categorization of receiving facilities and explain how the coordination of resources is attained
- list the components of mutual aid mass-casualty planning
- outline various designs and financing methods for an ems system.

3.0 MAIN CONTENT

3.1 History of Emergency Medical Services Systems

Emergency Medical services is a health care system requiring the integration of several components. It is designed to provide appropriate care for all emergency patients. Until the late 1960s, few areas provided adequate prehospital emergency medical care. The prevailing thought was that care began in the hospital emergency room. Rescue techniques were crude, ambulance attendants poorly trained, and equipment minimal . There was no radio communication and no physician involvement.Eventually, as costs forced many mortician-operated ambulance services to withdraw, local police and fire departments had to provide this service. In many areas, volunteer groups were formed. The result was a poliferation of local , independent EMS provider agencies that could barely , if at all communicate with each other. In addition, it was impossible to coordinate response activities on a scale larger simple, local calls.

The publication in 1966 of "Accidental death and Disability: The neglected disease of modern society", by the National Academy of Sciences, National Research Council, focused national attention on the problem. "The white paper", as the report came to be called, spelled out the deficiencies in prehospital emergency medical care. It also suggested guidelines for the development of EMS system, the training of prehospital emergency medical providers, and the upgrading of ambulances and their equipment. Although, many improvements have been made since the report's publication, a surprising number of inadequate services still exist. This landmark publication set off a series of Federal and private initiatives.

They include:

1966-Congress passed the National Highway Safety Act, which forced the states to develop effective EMS systems or risk losing federal highway construction funds.

1971-The White House funded nearly $9 million toward EMS demonstration projects. These projects were designed to be models for subsequent system development.

1972-The Robert Wood Johnson Foundation provided grants for establishing regional EMS projects and communication systems.

1973-Congress passed the Emergency Medical Services Systems Act, which provided funding in a series of projects awarded to develop regional systems.

In order to be eligible for this funding, an EMS system had to include the following 15 components specified by the act:

(a) Manpower (b) Training (c) Communications (d) Transportation (e) Emergency facilities (f) Critical care units (g) Public safety agencies
(h) Consumer participation (i) Access to care (j) Patient transfer (k) Standardised record-keeping (l) Public information and education (m) System review and evaluation (n) Disaster management (o) Mutual aid.

Unfortunately, the designers of this legislation left out two major components: system financing and medical control. When federal funding was significantly reduced in the early 1980s, many systems faced economic disaster and lacked a solid plan for financial recovery. Even worse, many systems were operating without physician direction. The legislative oversight have meant a long, uphill battle for medical directors attempting to re-establish authority and accountability for medicine practiced by EMTs and paramedics in the streets. In many ways, the EMS system Act paved the way for system development. But in some respects, it sents EMS in the wrong direction. This Act was amended in 1976 and again in 1979. A total of $215,000,000 was appropriated over a seven year period toward the establishment of regional EMS systems.

In 1981, the passage of consolidated Omnibus Budget Reconciliation Act (COBRA) wiped out all federal funding for EMS,except block grant programs administered by the department of Transportation and the Health and Human services Administration. Only a small portion of this money , however, is available for EMS activities. These funds are given to the states, which in turn disburse the money to formally established regional systems.

3.1.1 System Approach

The efficient delivery of emergency medical care requires a team effort and a systematic approach to get the best use of existing resources. There is no best method for providing prehospital emergency medical care in a given area. However, certain essential elements are considered standard for ensuring the best possible patient care in any region. Each system must develop operational policies for its components.

System administration
An administration agency should first be established. This agency will be responsible for managing the local systems's resources and for developing operational guidelines and standards for each component. A budget is created to operate the system and to select a qualified administrative staff. The agency should incorporate a planning bond-composed of providers, representatives of the medical community, emergency physicians, and consumers-that will advise and assist the agency in setting policy.Once established, the agency will designate who may function within the system. It will develop policies consistent with the existing state requirements. The EMS agency must develop a quality assurrance or quality improvement program to evaluate the system's effectiveness and to ensure that the best interest of the patient is always top priority.

In short, the needs of the patient are determined first; then the system is designed to meet those needs. The coordination of the system components to meet the patient's needs is the responsibility of the EMS agency.In addition to regional and municipal EMS agencies, EMS agencies also exist on the state level. The state EMS agencies are typically responsible for allocating funds to local systems, enacting legislation concerning the prehospital practice of medicine, licensing and certifying field providers, enforcing all state EMS regulations, and appointing regional advisory councils. In essence, EMS is a series of systems within a system. The integration of these systems and the coorporation of all participants result in a better quality of emergency medicl care.

Medical control
The EMS system will retain a medical director, who will be actively involved in, and ultimately responsible for, all clinical and patient care. All prehospital medical care provided by non-physicians is considered an extension of the medical director's license. Every prehospital ambulance or rescue service must have a medical director who is responsible for that service. Prehospital care providers are designated agents of the

medical director, regardless of whose employees they may be. For this reason, the medical director determines which providers may care for patients within the system. The medical director is the ultimate authority in all direct-line and indirect-line medical control issues.

Direct medical control

Direct medical control exist when prehospital providers communicate directly with a physician at a medical control or resource hospital . The physician's direction is usually based on established protocols for managing specific problems. This physician assumes responsibility and gives treatment orders for patients. Direct medical control physicians should be experienced in emergency medicine. They should have completed a training program that emphasizes system particulars, treatment protocols, and communication policies and procedures. Once they have become proficient in these areas, they should go through a formal certification process. They should also be required to ride with crews to get a feel for the realities of prehospital field medicine. Medical control communications are sometimes delegated to a mobile intensive care nurse(MICN), physician assistant or paramedic. In all circumstances, however, ultimate on-line responsibility rests with the medical control physician.

Control of medical emergency scene should go to the individual the most knowledge and best training in prehospital emergency stabilization and transport. When an advanced life support unit, under medical direction, is requested and dispatched to the emergency scene, a physician/patient relationship is established by the physician providing medical control. The paramedic is responsible for the subsequent management of the patient and acts as the medical control physician's agent, unless the patient's physician is present. If a private physician is present and assumes the responsibility for the patient's care , the paramedic should defer to the physician's orders.

If an intervener physician is present and on-line medical control does not exist , the paramedic should relinquish responsibility to the physician. But first, the physician must identify himself or herself and demonstrate a willingness to accept responsibility and to document the intervention as required by the local EMS system. If the treatment differs from established protocol , however, the physician should accompany the patient to the hospital. If the intervener physician is present and direct medical control does exist, the on-line physician is ultimately responsible. In case of disagreement between the intervener physician and the on-line physician , the paramedic must take orders from the on-line physician and put him or her in contact with the intervening physician.

Indirect medical control

Indirect medical controlincludes training and education , protocol development, audit, chart review and quality assurance. To be effective , medical control must have official and clearly defined authority, with power to discipline , or limit the activities of, those who deviate from the established standard of care.

Protocols are designed by the off-line medical control system to provide standardized approach to common patient problems and a consistent level of medical care. When treatment is based on such protocols, the on-line physician assists prehospital personnel in interpreting the patient's complaint, understanding the findings of their evaluation, and applying the appropriate treatment protocol. Protocols will be designed around the four T's of emergency medical care. They include:

(i) Trage
(ii) Treatment
(iii) Transport
(iv) Transfer

Triage guidelines help the physician make decisions involving patient flow through an EMS system. This simply means allocating the system 's resources to the needs of the victims. The EMS dispatcher will determine the type , level , and priority of response to the victim. The victim will then be directed to the appropriate receiving facility, which can provide definitive care or stabilize the victim until he or she is transfered to a definitive care facility .
Treatment often includes emergency interventions by field personnel . Some procedures will be done upon a direct order from the medical control physician. Others will be standing orders.

Treatment protocols should be kept current with new research.
Transport involves decisions about mode (air versus ground) and the level of care during transport. Most plans call for taking the patient to the closest appropriate facility, as designated by the system. Transport plans are based on three factors:

(a) Nature of the injury or illness
(b) Condition of the patient
(c) Estimated transport time.

Transfer protocols cover the management of inter-hospital patient transfer. They allow for region wide continuity of care when patients are transferred. Agreement between receiving facilities within the system ensure that the patient is admitted to the definitive care facility.Protocols will also be established for special circumstances, such as the proper handling of "Do Not Resuscitate" orders, patients who refuse treatment , sexual abuse , abuse of children or elderly persons and intervener physicians. Although, protocols standardize field procedures , they should allow the paramedic flexibilty to improvise and adapt to special circumstances. Protocols establish a basis for medical care and a standard for accountabilty in an EMS.

3.1.2 Public Information and Education

The public is an essential , yet often overlooked, component of an EMS system. An EMS system should have a plan to educate the public about recognising an emergency, accessing the system, and initiating basic life support.The American Heart Association (AHA) estimates that 350,000 cardiac arrests per year occur before the patient reaches hospital. Most happen within two hours of the onset of cardiac symptoms, because many patients deny that something is wrong and delay calling for help. If the patient , a family member, friend , or bystander can recognize the emergency and intervene in time, many cases of sudden death can be prevented. Families of patients with coronary artery disease should be targeted for instruction in recognizing emergency symptoms. The second aspect of public education is system access. Systems with telephone service make emergency access easy.

The phone number should be well publicised, and citizens should be taught how to give information to the emergency medical dispatcher. After recognizing that a medical emergency exists, the bystander must initiate basic life support procedures such as cardiopulmonary resuscitation (CPR). This could also include initiatial patient care after major trauma and hemorrhage control. Abundant research in the last 20 years indicates a relationship between response times and the patient's ultimate recovery. Communities that have many rapid paramedic response , have proven that a large number of patients can be successfully resuscitated. On the other hand , communities weak in prompt bystander CPR have shown a much lower rate of recovery. The role of the bystander in emergency medical care is critical to successful resuscitation of a cardiac arrest victim.

3.1.3 EMS Communications

The communications network is the heart of any regional EMS system. Coordinating the components into an organised response to urgent medical situations requires a comprehensive , flexible, communications plan. The basic components of a communications systems include the following six elements:

(i) Citizen access
(ii) Single control center
(iii) Operational communications capabilities
(iv) Medical communications capabilities
(v) Hardware
(vi) Software

Any citizen with an urgent medical need should have a simple and reliable mechanism for accessing the EMS system. Multiple community telephone numbers add life-threatening minutes to any emergency response system. A single communications center that can communicate with and direct all emergency medical units in the system is the best.

Emergency medical dispatching
Emergency medical dispatching is the nerve cente of an EMS program. The activities of medical dispatchers are crucial to the efficient operation of the system. Dispach is the means of assigning and directing appropriate medical care to the victim. An emergency medical dispatching plan should include pre-established interogation protocols, pre-assigned response configurations, system status management , and pre-arrival telephone instructions. Medical direction for the emegency medical dispatcher should be given the EMS medical director. Quality assurance of the EMS dispatch program is an EMS agency responsibility.An effective emergency medical dispatching system has certain key components. EMS dispatchers do more than send ambulances; they make sure the system's resources are in constant readiness to respond. System status management relies on projected call volumes and locations, not geographical or political tradition, to strategically place ambulances and crews. The system is used to reduce response times.

Education and certification
The two kinds of education program for EMS personnel are original education and continuing education. Original education programs are the initial training courses for pre-hospital providers. They involve the completion of a standardised course that meets or exceeds the United States Department for Transportation national curriculum for that level. Continuing education programs include refresher courses for recertification and periodic in-service training sessions. All education programs should have a medical director who is involved in EMS system. The EMS agency is responsible for assuring funding for these programs.

3.1.4 Quality Assurance and Quality Improvement

An EMS system must be designed to needs of of the patient. In EMS, patients are the users of the system and hence the customer. The quality of an EMS is reflected in the daily medical practices of its prehospital care providers. In EMS, the only acceptble quality is excellence. Because of this, the EMS system of today must have a program to evaluate the quality of service provided. For a quality evaluation program to be effective , it must be comprehensive and continuously ongoing. In addition, the EMS system must be willing to react to the quality evaluation program and to adjust its practices and procedures accordingly.

A quality assurance (QA) program is primarily designed to maintain continuous monitoring and measurement of the quality of the clinical care delivered. QA programs emphasise evaluation of objective clinical data such as response times, adherence to protocols, patient survival, and other key indicators of system performance. QA programs document the effectiveness of the care provided. They also help identify problems and select areas that need improvement. Quality Improvement (QI) for evaluating system performance. A QI program focuses on customer perceptions of the service provided. It is an ongoing effort to refine and improve the system in an attempt to provide the highest

level of service possible. In addition to clinical issues, a QI program evaluates other aspects of EMS system such as billing, maintainance, supply, and so on. EMS quality can be divided into two broad categories: Take-it-for granted quality and service quality.

3.1.5 Research

In order to provide a scientific basis for prehospital EMS, a formal ongoing research program is an essential component of the system.Existing and future procedures, techniques, and equipment must be evaluated scientifically. For moral , educational, medical, financial reasons, an EMS system design must include research. Unfortunately, many protocols and procedures that paramedics use today have evolved without clinical evidence of their usefulness, safety, or benefit to the patient. Future EMS research prjects must address the following issues:

(i) Which prehospital field interventions actually reduce morbidity and mortality?
(ii) Are the benefits of certain paramedic field procedures worth the potential risk?
(iii) What is the cost of/benefit ratio of sophisticated prehospital EMS system?
(iv) Is field stabilisation possible, or should paramedics begin immediate transport in every case.

Receiving facilities
Not all hospitals are equal in emergency and support service capabilities. Hospital categorisation identifies the readiness and capability of a hospital and its staff to receive effectively treat emergency patients. Categorisation originated from the realisation that patients have varying degrees of illness and injury and that receiving facilities have varying capabilities to provide initial or definitive care. A facility categorisation system lets the EMS system's coordinators know in advance of specialty areas within EMS delivery system . This knowledge expedites the transportation of emergency patients to hospitals that will provide definitive treatment, or live saving stabilisation, until transfer can be arranged.

3.1.5 Mutual Aid/Mass-Casuality Preparation

No system is an island , and the resources of any region will sometimes be overwhelmed. A formalised mutual-aid agreement policy will ensure that help is available when needed. Mutual-aid agreements can be between neighboring departments, municipalities, systems and even states. Coorperation among all EMS agencies must supersede geogrphical, political, and historical boundaries.

System financing
EMS services can be hospital-based, fire department or police department –based, municipal and third service, private commercial business, volunteer or some combination thereof.

4.0 CONCLUSION

In this unit, you have learned that EMS is a complex health care system made up of personnel, equipment, and resources. The EMS system has several components. You have also learned about medical control, EMS communications and others.
You have also realised that education and certificate, quality assurance, and quality improvement have been discussed. You should at this point be able to explainin your own words, EMS, Receiving facilities, mutual-aid/mass-casuality preparation, and explain the importants of quality valuation and EMS very well.

5.0 SUMMARY

An EMS system comprises a number of components , all of them crucial to providing emergency medical care for sick and injured people. The system is a continuum of care: from the Emergency medical team who conducts public education classes; to the mechanic who keeps the ambulance fleet running; to the emergency medical dispatcher who calms a distressed caller and provides a lifesaving instructions over the phone; to the paramedic who provides field intervention; to the emergency department physician, surgeon and physical therapist who will see the patient through definitive care and rehabilitation. No one component, no one person, is more important than another. EMS a total team effort. EMS systems are designed with the patient as the highest priority. They begin with a strong administrative agency, which structures the system around the patients' needs and grants the medical director ultimate authority in all issues of patient care. The medical director is an emergency physcian who remains actively involved in all components of the system.

6.0 TUTOR-MARKED ASSIGNMENT

(i) Describe quality assurance and quality improvement.
(ii) Describe medical dispatching.
(iii) What is medical control? Mention two types of medical control and discuss.
(iv) Describe system approach.

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MODULE 2

UNIT 1 FALL- RELATED INJURIES

CONTENTS

1.0 INTRODUCTION

Mechanism of Injury: This is usually apparent through careful evaluation of the trauma scene. Identifying the mechanism of injury can help you anticipate both the location and the seriousness of injuries. Some of the mechanisms of injury are fall and sports related injuries. Falls are the 2nd leading cause of unintentional injury after road traffic accidents. This unit will help you acquire some basic understanding of fall and its related injuries as indicated in the objectives below.

2.0 OBJECTIVES

At the end of this unit, you should be able to:

- define the term fall
- mention factors relating to fall
- state methods of preventing fall-related injuries.

3.0 MAIN CONTENT

3.1 Definition of fall

Fall is an event which results in a person coming to rest inadvertently onthe ground or flooror other lower level. According to the WHOdatabase, fall-related deaths and non-bullet fatal injuries exclude thosedue to assault and intentional self-harm. Falls from animals, burning
buildings and transport vehicles, and falls into fire, water and machineryare also excluded.

3.2 Factors Relating to Fall

Falls are responsible for the largest number of hospital visit for non-fatalinjuries, especially for children and young adults, and even among theelderly.Falls from roof tops, balconies, windows, staircases, trees, constructionsites, slippery floors, etc. are rampant. Falls emanating from these canalso lead to serious head injuries, fractures of hip, vertebrae, forearm,leg, ankle, pelvis, upper arm, hand, etc.

3.3 Preventing Falls and Related Injuries

Safer playground design including use of mud andsand surfaces instead of hazardous paved ones should beadopted.Enactment of safety regulations for playgroundsshould be enforced.Safety guidelines and standards should be adoptedin designing chairs and other furniture for children and theelderly.Sensitisation of architects, builders and masons forsafer designs of stairs, balconies and rooftops with appropriaterailings, grab bars and landings.Encouragement of public use of safety standards forprotectors on windows.Safer furniture and household design guidelines forthe elderly and disabled.Encouragement/evolution of safer workingtechniques and use of harnesses for construction workers, treeclimbers, window cleaners and all those who work at heights.

3.4 Sports-Related Injury

Sports medicine is growing very rapidly, it is an extensive field, which certainly cannot be covered completely here,however, some basic principles will help you to better understand and care for athletic injuries.Sports injuries are most commonly produced by extreme exertion ,fatique, or by direct forces of trauma. Injuries can be secondary to acceleration , deceleration , compression, rotation, and hyperextension or hyperflexion. These forces leave behind soft-tissue damage to the skin and muscle, connective tissue injury to tendons and

ligaments, skeletal trauma to long bones or the spinal column, as well as internal damage to either hollow or solid organs.When a debilitating sports-related injury occurs, the athlete should be transported to an emergency department and examined before further participation is allowed. Injuries that display minimal pain may be significantly worsened by the stress of further competition. Such stress may cause complete rupture of ligaments or other soft-tissue injury and increase the potential for permanent disability.

In some contact sports, athletes may experience severe impact. If collision leads to any period of unconsciousness, neurologic deficit, or lowered level of orientation, the individual should be evaluated by emergency department personnel. There is a strong desire by coaches and players alike to return to the game. However, until a head and cervical spine injury can be ruled out, discourage such action.Protective gear reduces the chance for, and significance of, injury. However, gear can sometimes be a contributing factor in sports injuries. In major contact sports, for example, shoes are designed to give maximum traction using cleats to lock the foot firmly in position. In football, a player might be struck, forcing the body to turn on an immobile foot.. Ligaments may tear in the knee, resulting in a severe and disabling leg injury. In other cases, protective gear may hinder your complete assessement and patient stabilisation. Therefore, it is important to remove all gear from the area of injury.

4.0 CONCLUSION

This unit has exposed you to the term fall and its related injuries, sports related injuries. It also exposed you to the preventive measures to be adopted in order to avoidincessant falls. You should at this point be able to explain in details andin your own words, fall-related injuries including sports related injuries.

5.0 SUMMARY

This unit has focused on the definition of fall and related injuries. It alsohighlighted themethods to be adopted in preventing falls and fall-relatedinjuries.

6.0 TUTOR-MARKED ASSIGNMENT

1. Mention five fall-related injuries that you know.
2. State at least five preventive falls related to injuries.
3. Briefly explain sports related injuries.

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UNIT 2 DROWNING

CONTENTS

1.0 INTRODUCTION

Drowning is the respiratory impairment from being in or under a liquid, it is further classified by outcome into death, ongoing health problems and no ongoing health problems, and it occurs more frequently in males and young. In 2013 it was estimated to have resulted in 368,000 deaths down from 545,000 deaths in 1990. Of these deaths 82,000 occurred in children less than five years old. It accounts for 7% of all injury related deaths (excluding those due to natural disasters), with 96% of these deaths occurring in low-income and middle-income countries.

Drowning is the second leading cause of accidental death in people ages 1-44. It causes unintentional injury deaths worldwide. (Approximately 85 percent of near-drowning victims are male, and two-thirds of these do not know how to swim). There has been an attempt made to differentiate between the terms drowning and near-drowning. The term drowning means that death occurred within 24 hours of submersion(, while, the term near-drowning indicates that death either did not occur or occurred more than 24 hours after submersion). This unit will help you acquire understanding of what drowning is and its basic components as indicated in the objectives below.

2.0 OBJECTIVES

At the end of this unit, you should be able to:

- define drowning and near-drowning

- discuss the risk of drowning and management of drowning
  - discuss pathophysiology of drowning, Sea and fresh water drowning
  - list various ways to prevent drowning.

3.0 MAIN CONTENT

3.1 Definition of Drowning

Drowning is the process of experiencing respiratory impairment from unintentional submersion/immersion in liquid. Drowning is death Occasioned through submersion in water. Drowning outcomes are classified as:Morbidity and Non-morbidity.

3.1.1 Pathophysiology of Drowning

As a public health scientist, you will need to understand the sequence of events in drowning or near-drowning**.** Following submersion, if the victim is conscious, he or she will undergo a period of complete apnea (the abscenceof breathing) for up to three minutes.
This apnea is an involuntary reflex as the victim strives to keep the head above water. During this time blood is shunted to the heart and brain, in a fashion similar to the primitive, diving reflex, present in certain lower animals.

When the victim is apneic, the PaCo2(partial pressure of carbondioxide in the blood) in the blood rises to greater than 50mmHg. Meanwhile, the PaO2(partial pressure of oxygen in the blood) of the blood falls below 50mmHg. The stimulus from the hypoxia ultimately overrides the sedative effects of the hypercabia(an increased level of carbon dioxide in the body), resulting in central nervous system stimulation.

Until unconscious, the victim experiences a great deal of panic. During this stage the victim makes violent inspiratory and swallowing efforts. At this point, copious amounts of water enter the mouth, posterior pharynx and stomach, stimulating severe laryngospasm and bronchospasm. In approximately 10 percent of drowning victims, and in a much greater percentage of near-drowning victims, this laryngospasm prevents the influx of water into the lungs. If a significant amount of water does not enter the lungs, it is referred to as dry drowning. Conversely, if a laryngospasm does not occur, and a significant quantity of water does enter the lungs, it is referred as to a wet drowning. The laryngospasm, or airway obstruction due to aspirated water, further, aggravates the hypoxia, with coma ultimately ensuing. Persistent anoxia results in a deeper coma.

Following unconsciousness, reflex swallowing continues, resulting in gastric distension and increased risk of vomiting and aspiration. If untreated, hypotension, bradycardia, and death result in a short period. Drowning and near-drowning are primarily due to asphyxia from airway obstruction in the lung secondary to the aspirated water or laryngospasm. If, in a near-drowning episode this process does not end in death, the fluid may cause lower airwaydisease. Expect different physiological reactions in cases of fresh-water and sea-water drowning or near-drownings.

3.1.2 Fresh-Water Drowning

In fresh-water drowning or near-drowning, the large surface of the alveoli and small airways allow a massive amount of hypotonic water to diffuse across and into the vascular space. This results in haemodilution. Hemodilution produces a thickening of the alveolar walls with inflammatory cells, hemorrhagic pneumonitis, and destruction of surfactant. Surfactant is a substance in the alveoli responsible for keeping the alveoli open.

When the capillaries of the alveoli are damaged , plasma proteins leak back into the alveoli, resulting in the accumulation of fluid in the small airways, this in turn leads to multiple areas of atelectasis(a collapse of the alveoli, which in turn decreases ventilator effectiveness) with shunting and hypoxemia.

3.1.3 Sea-Water Drowning

In sea-water drowning, the hypertonic nature of the fluid draws water from the blood stream into the alveoli. In near-drowning, the hypertonic nature of sea-water, which is 3-4 times more hypertonic than plasma, draws water from the blood stream into the alveoli. This produces pulmonary edema leading to profound shunting. The result is failure of oxygenation, producing hypoxemia, since the blood is travelling through the lung tissues without being oxygenated. Additionally, respiratory and metabolic acidosis develops due to the retention of Co2 and developing anaerobic metabolism. Since all of the above factors disrupt normal pulmonary function, initial field treatment must be directed toward correcting the profound hypoxia.

Factors affecting survival

There are other factors that may have an impact on drowning and near-drowning survival rates. These include such things as the cleanliness of the water , the length of time submerged, and the age and the general health of the victim. Children have a longer survival time and a greater

probability of a successful resuscitation. Even more significant is the water temperature. The concept of developing brain death after four to six minutes without oxygen is not applicable in the cases of near-drowning in cold water. Some patients in cold water can be resuscitated after 30 minutes or more in cardiac arrest. However, persons under water 60 minutes or longer usually cannot be resuscitated.
A possible contribution to survival may be the mammalian diving reflex. When a person dives into cold water, he or she reacts to the submersion of the face. Breathing is inhibited, the heart rate becomes bradycardic, and vasoconstriction develops in tissues relatively resistant to asphyxia. Meanwhile, cerebral and cardiac blood flow is maintained. In this way, oxygen is sent and used only where it is immediately needed to sustain life. The colder the water, the more oxygen is diverted to the heart and brain . A common saying in emergency medicine states: The cold water drowning victim is not dead until he or she is warm and dead.

3.1.4 Risk of Drowning

Drowning can occur during swimming, boating, hunting, fishing andeven while taking a bath. Approximately 10% of childhood drowningtakes place in bathtubs. Small children can drown in as little as one inchof water and are therefore at risk of drowning in wading pools, bathtubs,buckets, diaper pails, toilets, spas and hot tubs.

Important risk factors for drowning are:

Age
Children under five years have the highest drowning mortality rateworldwide. Children under this age range constitute over half of theglobal mortality and 60 % of the total number of deaths arising fromdrowning. Drowning rate is also high among children aged between 0-14 years.

Sex
Males have higher drowning rates than females due to increasedexposure to water and riskier behaviours such as swimming alone,drinking alcohol before swimming and boating.

Occupation
Communities dependent on water bodies for their living e.g. fishermenhave a high risk of drowning.

Floods
Large numbers of drowning deaths are associated with floods, especiallyin China.

Access to water
Unfenced homes in proximity to bodies of water increase the risk ofdrowning; pools, farm dams, irrigation channels and wells are alsoimportant risk factors for children drowning. Infants left alone or withanother child in an adult bathtub are at significant risk.

Transportation vessels
Vessels that are unsafe or overcrowded and poor weather conditions areassociated with large number of deaths through drowning. Unavailability or unawareness of a Personal Flotation Device (PFD) on a transportation vessel is another risk factor.

3.1.5 Management of Drowning

The patient should be removed from the water as soon as possible by a trained rescueswimmer. Ventilation should be initiated while the patient is still in water. Rescue personnel should wear protective clothing if water temperature is less than 70 F. In addition, a safety line should be attached to the rescue swimmer. In fast water, it is essential to use personnel specifically trained for this type of rescue. Suspect head and neck injury if the patient experienced a fall or was diving. Rapidly place the victim on a long backboard and remove him or her from the water.

Examine the near-drowning victim for airway patency, breathing and pulse.If indicated, begin cardiopulmonary resuscitation (CPR) . Airway management should include proper suctioning and use of airway adjuncts. C-spine injury should be considered and treated accordingly. Administer oxygen at a 100 percent concentration. If available, and if transport time is longer than 15 minutes, respiratory rewarming should take place. The Heimlich maneuver is contraindicated. Next, establish an intravenous(IV) of lactated Ringer’s or normal saline for venous access and run at 75ml/hour. If indicated, carry out defibrillation. If the patient is hypothermic, treat him or her according to hypothermia protocol . Unless hypothermia is present, resuscitation is not indicated if there is evidence of putrefaction (decomposition) or if immersion has been extremely prolonged.

More than 90 percent of near-drowning patients survive without sequelae. All near-drowning patients should be admitted to hospital for observation. Some of these patients have problems with pulmonary parenchymal injury, destruction of surfactant, aspiration pneumonitis, or pneumothorax. A number require an extended hospital stay due to hypoxia, hypercarbia, and mixed metabolic and respiratory acidosis. Treatment of the effects of cerebral hypoxia occasionally continues throughout and even after hospitalization.

3.1.6 Prevention of Drowning

Victims of drowning have a very slim chance of survival afterimmersion, the victim losses consciousness after approximately twominutes of immersion and irreversible brain damage can take place after4 – 6 minutes.Different preventive strategies include:Collection of data on drowning, identification of vulnerablepopulations, undertaking research to identify risk factors,protective factors and exposure measures, promotion, facilitation andimplementation of drowning prevention measures and policies.

Monitoring and evaluating interventions which include removalof hazard (e.g. drain unnecessary accumulation of water baths,ponds, buckets etc.).Creating barriers to water bodies, especially in villages.Promoting "learn to swim" activities andTraining the community in resuscitation.Other preventive measures include development of strategies to ensureeffective inspection and certification system for safe surfacetransportation on water and creation of safety standards for public andprivate swimming pools.Sensitisation of policy makers and community leaders on the need toprovide life jackets and floatation devices at swimming pools, boats andbarges is also important towards preventing deaths arising fromdrowning. Training of life-guards, and fencing of deep parts of lakes andponds.

4.0 CONCLUSION

In this unit you have learnt what drowning is , fresh- water drowning ,sea-water drowning are and the risk involved in drowning. You have also learnt about the pathophysiology and management. You should at this point be able to define drowning as process of experiencing respiratory impairment from unintentionalimmersion. You have also known the various ways of preventingdrowning in the Nigerian context.

5.0 SUMMARY

This unit focuses on an important aspect of life which is often neglectedby the society. The risk of drowning has been discussed extensively foryour understanding.

6.0 TUTOR-MARKED ASSIGNMENT

1. Drowning can occur during swimming. Discuss

2. Enumerate five preventive measures to reduce drowning related morbidity and mortality.

3. Discuss in details the pathophysiology of drowning.

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UNIT 3 STRESS

CONTENTS

1.0 INTRODUCTION

Stress is an inherent aspect of emergency medical services. Only in recent years has stress been recognised as a bona fide hazard of emergency work, hospital workers/personnels must learn to manage stress, as well as the conditions that cause it. Dealing with stress in a positive manner promotes emotional and physical health and prevents burnout. This unit will discuss stress and how to cope with its effects.

2.0 OBJECTIVES

At the end of this unit, you should be able to:

- define stress
- know the causes and management of stress
- discuss physiology of stress and different types of reactions.

3.0MAIN CONTENT

3.1 Stress

The word stress literally means a hardship, force, or strain. From a psychological standpoint, stress can be defined as a state of physical and psychological arousal. It exists to some degree in everyone. physiological or biological **stress** is an organism's response to a stressor such as an environmental condition or a stimulus. Stress is a body's method of reacting to a challenge. According to the stressful event, the body's way to respond to stress is by sympathetic nervous system activation which results in the fight-or-flight response. Because the body cannot keep this state for long periods of time, the parasympathetic system returns the body's physiological conditions to normal (homeostasis). In humans, stress typically describes a negative condition or a positive condition that can have an impact on a person's mental and physical well-being.A stressor is any agent or situation that causes stress. Often stress results from a perceived imbalance between the demands of the job and our ability to meet those demands. Inaddition, stress may be increased by the demands imposed by the people and events around us.

3.1.1 Physiology of Stress

The body’s response to stress is termed a Stress Reaction.The stress reaction is complex and involves many body systems. After the detection of the stressor. Information received from the various senses is processed and interpreted in the cerebral cortex . The cerebral cortex is the part of the brain that controls higher mental functions and intellect. The limbic system, located in the mid- brain is subsequently activated and an emotional reaction (fear, anger, rage, hostility etc)occurs. The response then proceeds to lower brain centers, including the hypothalamus. The hypothalamus controls many bodily functions, including temperature and sleep. It also regulates the endocrine system. Once stimulated by the hypothalamus, the endocrine system releases hormones such as epinephrine (adrenalin) and norepinephrine (noradrenalin) in response to the stress. The release of epinephrine further stimulates the brain. The body quickly enters a hyperalert state commonly called the, fight or flight, response or alarm reaction. The fight- or- flight response is characterised by an increase in heart rate and blood pressure, papillary dilation, excessive perspiration, increased muscle tension, increased blood glucose level, and a sense of anxiety.

This response prepares the body to deal with any threats, real or perceived.

3.1.2 The Body's Response to Stress

The body is under various levels of stress at all times. However, after repeated exposure to the same stressor, the body will adapt by suppressing the typical emotional and physical responses. The body's response to stress generally goes through the following stages.

Stage I: Alarm; an alarm reaction occurs at the first exposure to the stressor. The signs include increased pulse rate, papillary dilation, and other responses of the sympathetic nervous system. If resistance to stress is diminished, the physiological and emotional response can be overwhelming.

Stage II; Resistance; this stage starts when the individual begins to adapt to the stress. Resistance is often brought about by the use of various coping mechanisms. Physiological parameters, such as pulse and blood pressure, may return to normal. As adaptations develops, resistance increases above the normal level.

Stage III: Exhaustion: Prolonged exposure to the same stressors leads to the exhaustion of an individual's adaptation energy. The signs of the alarm reaction reappear, but they are now much more difficult to reverse.

3.1.3 Types of Stress Reactions

A stress has both physical and psychological components. There are three types of stress reactions that can occur; acute, delayed and cumulative stress:

Acute stress reaction; the**acute stress reaction** usually occurs after a catastrophic event. The reaction has a powerful emotional impact on the rescuer. Catastrophic events capable of evoking such a response have been labelled**critical incidents.** The acute stress reaction that follows is called **critical incidents stress.** The acute stress reaction may begin at the scene or shortly after the event. Several warning signs or symptoms characterize its onset. Some signs and symptoms require immediate intervention, while others do not. Early intervention will help minimise the long- term effects of the stress. Critical Incident Stress Debriefing (CISD) helps to effectively deal with stress and prevent post-traumatic

stress disorder. If indicated, the CISD team may refer the rescuer for professional counselling.

Delayed stress reaction: Delayed stress reaction also called post - traumatic stress disorder, occurs, days, weeks, months or even years after a critical incident. Not all personnel who experience a critical incident will suffer a delayed stress reaction-but some will. The following signs or symptoms characterise post-traumatic stress disorder:

(i)Re-experiencing of the traumatic event

(ii)Recurrent and intrusive recollections of the event.

(iii) Recurrent dreams and nightmares related to the event.

(iv) Flashbacks, or sudden feelings that the event is recurring, usually after exposure to some triggering stimulus.

Diminished responsiveness to the external world

(i) Decreased interest in life.

(ii) Feeling detached or estranged from others.

(iii) Suppression of normal emotional responses such as love, anger, or fear.

Physical and cognitive symptoms

(i) Hyper alertness

(ii) Difficulty sleeping.

(iii) Survivor guilt.

(iv) Memory impairment and difficulty concentrating

(v) Avoidance of any activities that may cause recall of the event.

(vi) Avoidance of thoughts or feelings associated with the incident.

(vii) Problems with interpersonal relationships.

Post-traumatic stress disorder can interfere with your life. It can provoke marital problems, alcohol and drug abuse, personality changes, and even suicide. Because of this, persons suffering from a post-traumatic stress disorder should seek expert professional help soon.

Cummulative stress reaction: The third type of stress reaction is called a cumulative stress reaction, or **Burnout.**Cumulative stress, unlike the type of stress discussed earlier, does not result from a single critical incident. Instead, it stems from recurring minor stressors, both work and non-related.A cumulative stress reaction typically takes years to develop. Initially, the person will experience depression, boredom, apathy, emotional fatigue. As the reaction progresses, the person will experience sleep disturbances, increasing loss of emotional control, frequent physical complaints, physical and emotional fatigue, irritability, and worsening depression. Ultimately, the person will develop extreme fatigue, severe depression, feelings of paranoia, crying spells, loss of sexual drive, inability to perform a job, significant problems managing his or her personal life, occasionally suicidal or homicidal thinking .

Failure to intervene in the early stages of cumulative stress will result in severe disability for the person involved. Often he or she will suffer destruction of close family relationships, divorce and inability to work in his or her chosen profession. The best treatment for the cumulative stress reaction is prevention. Emergency Medical Services (EMS) systems should have programs that routinely screen their personnel for signs and symptoms of cumulative stress. Such programs will allow for early interventions, thus, avoiding the condition's devastating consequences. Also stressful assignments such as busy **stations or** those with a higher incidence of critical patients, should be sharedamong personnel**.**

3.1.4 Effect of Stress on the Immune System

Stress is the body's reaction to any stimuli that disturbs its equilibrium. When the equilibrium of various hormones is altered the effect of these changes can be detrimental to the immune system. Much research has shown a negative effect stress has on the immune system, mostly through studies where participants were subjected to a variety of viruses. In one study individuals caring for a spouse with dementia, representing the stress group, saw a significant decrease in immune response when given an influenza-virus vaccine compared to a non-stressed control group. A similar study was conducted using a respiratory virus. Participants were infected with the virus and given a stress index. Results showed that an increase in score on the stress index correlated with greater severity of cold symptoms. Studies with HIV have also

shown stress to speed up viral progression. Men with HIV were 2–3 times more likely to develop AIDS when under above average stress.

Stress affects the immune system in many ways. The immune system protects the body from viruses, bacteria, and anything that is different or that the body does not recognize. The immune system sees these as intruders and it sends messages to attack. The white blood cells, leukocytes, are very important to the immune system. White blood cells have several types including B cells, T cells, and natural killer cells. B cells secrete antibodies. T cells attack intruders and natural killer cells attack cells that have been infected by viruses. These leukocytes produce cytokines which fight infections. But they also are the immune systems communicator in telling the brain that the body is ill. When an individual is stressed or going through a stressful experience the immune system starts to produce natural killer cells and cytokines.

When levels of cytokines are higher they combat infections and therefore the brain gets communicated the body is ill and it produces symptoms as if the individual was ill. These symptoms include fever, sleepiness, and lack of energy, no appetite, and basically flu like symptoms. These symptoms mean the body is fighting the illness or virus. This is useful for when the body goes through the stress from an injury. But the body has now evolved to do this process during stressful events such as taking exams, or even going through a life changing event such as a death of a family member or a divorce. That is why many times when individuals are stressed because of life changing events or situations such as those, they get these symptoms and believe they are sick when in reality it can be because the body is under stress.

3.1.5Effects of Chronic Stress

Chronic stress is defined as a "state of prolonged tension from internal or external stressors, which may cause various physical manifestations – e.g., asthma, back pain, arrhythmias, fatigue, headaches, HTN, irritable bowel syndrome, ulcers, and suppress the immune system". Chronic stress takes a more significant toll on the body than acute stress does. It can raise blood pressure, increase the risk of heart attack and stroke, increase vulnerability to anxiety and depression, contribute to infertility, and hasten the aging process.

For example, results of one study demonstrated that individuals who reported relationship conflict lasting one month or longer have a greater risk of developing illness and show slower wound healing. Similarly, the effects that acute stressors have on the immune system may be increased when there is perceived stress and/or anxiety due to other events. For example, students who are taking exams show weaker immune

responses if they also report stress due to daily hassles. While responses to acute stressors typically do not impose a health burden on young, healthy individuals, chronic stress in older or unhealthy individuals may have long-term effects that are detrimental to health.

3.1.6 Mechanisms of Chronic Stress

Studies revealing the relationship between the immune system and the central nervous system indicate that stress can alter the function of the white blood cells involved in immune function known as lymphocytes and macrophages. People undergoing stressful life events, such as marital turmoil or bereavement, have a weaker lymph proliferative response. People in distressed marriages have also been shown to have greater decreases in cellular immunity functioning over time when compared to those in happier marriages. After antigens initiate an immune response, these white blood cells send signals, composed of cytokines and other hormonal proteins, to the brain and neuroendocrine system. Cytokines are molecules involved with cell signaling.

Cortisol, a hormone released during stressful situations, affects the immune system greatly by preventing the production of cytokines. During chronic stress, cortisol is over produced, causing fewer receptors to be produced on immune cells so that inflammation cannot be ended. A study involving cancer patient's parents confirmed this finding. Blood samples were taken from the participants. Researchers treated the samples of the parents of cancer patients with a cortisol-like substance and stimulated cytokine production. Cancer patient parents' blood was significantly less effective at stopping cytokine from being produced.

3.1.7 Chronic Stress and Wound Healing

The immune system also plays a role in stress and the early stages of wound healing. It is responsible for preparing the tissue for repair and promoting recruitment of certain cells to the wound area. Consistent with the fact that stress alters the production of cytokines; Graham et al. found that chronic stress associated with care giving for a person with Alzheimer's disease leads to delayed wound healing. Results indicated that biopsy wounds healed 25% more slowly in the chronically stressed group or those caring for a person with Alzheimer's disease.

3.1.8 Chronic Stress and Development

Chronic stress has also been shown to impair developmental growth in children by lowering the pituitary gland's production of growth hormone, as in children associated with a home environment involving serious marital discord, alcoholism, or child abuse.

3.1.9 Chronic Stress and Memory

Chronic stress is seen to affect the parts of the brain where memories are processed through and stored. When people feel stressed, stress hormones get over-secreted, which affects the brain. This secretion is made up of glucocorticoids, including cortisol, which are steroid hormones that the adrenal gland releases, although this can increase storage of flashbulb memories it decreases long-term potentiation (LTP). Prolonged Stress can also be harmful to our body. That is because stress releases cortisol, and cortisol causes metabolic activity throughout the body. Metabolic activity is raised in the hippocampus. Overstimulation and toxins then are more likely to kill or damage neurons in the hippocampus. The hippocampus is important in the brain for storing certain kinds of memories and damage to the hippocampus can cause trouble in storing new memories but old memories, memories stored before the damage, are not lost. Also high cortisol levels can be tied to the deterioration of the hippocampus and decline of memory that many older adults start to experience with age.

3.1.10 Clinical Symptoms and Disorders

Signs of stress may be cognitive, emotional, physical, or behavioral.

Cognitive symptoms:

(i)Memory problems

(ii)Inability to concentrate

(iii)Poor judgment

(iv)Pessimistic approach or thoughts

(v)Anxious or racing thoughts

(vi)Constant worrying

Emotional symptoms

- Moodiness
- Irritability or short temper
- Agitation, inability to relax
- Feeling overwhelmed
- Sense of loneliness and isolation
- Depression or general unhappiness

Physical symptoms

- Aches and pains
- Diarrhea or constipation
- Increased frequency of urination
- Indigestion
- Changes in blood glucose
- Nausea, dizziness
- Chest pain, rapid heartbeat
- Loss of sex drive
- Frequent colds
- Irregular periods.

Behavioral symptoms

- Eating more or less
- Sleeping too much or too little
- Isolating oneself from others
- Procrastinating or neglecting responsibilities
- Using alcohol, cigarettes, or drugs to relax
- Nervous habits (eg nail biting, pacing)

Coping/stress management

Responses to stress include adaptation, psychological coping such as stress management, anxiety, and depression. Over the long term, distress can lead to diminished health and/or increased propensity to illness; to avoid this, stress must be managed.

- Stress management encompasses techniques intended to equip a person with effective coping mechanisms for dealing with psychological stress, with stress defined as a person's physiological response to an internal or external stimulus that triggers the fight-or-flight response. Stress management is effective when a person uses strategies to cope with or alter stressful situations.
- There are several ways of coping with stress, such as controlling the source of stress or learning to set limits and to say "no" to some of the demands that bosses or family members may make.
- A person's capacity to tolerate the source of stress may be increased by thinking about another topic such as a hobby, listening to music, or spending time in a wilderness.
- A way to control stress is first dealing with what is causing the stress if it is something the individual has control over. Other methods to control stress and reduce it can be: to not procrastinate and leave tasks for last minute, do things you like, exercise, do breathing routines, go out with friends, and take a

break. Having support from a loved one also helps a lot in reducing stress.

- A study was done and it showed that the power of having support from a loved one or just social support, lowered stress in the individuals. They gave painful shocks to married women's ankles. On some trials women were able to hold their husbands hand, on other trials they held a strangers hand, and then held no one's hand.

When the women were holding their husbands hand, the response reduced in many brain areas. When holding the strangers hand the response reduced a little but not as much as when they were holding their husbands hand. Social support helps reduce stress but even more if the support is from a loved one.

4.0 CONCLUSION

Stress is a part of emergency medical services. You should recognise that a certain level of stress is important. You have learnt various aspects of stress, the management, symptoms and signs, different types of stress reactions and physiology of stress. You can now explain them in your own words.

5.0 SUMMARY

This unit has focused on the details of Stress, its definition, physiology, types and management of stress. Generally, you should remember that stress is an organism's response to a stressor such as environmental conditions or stimulus.

6.0 TUTOR-MARKED ASSIGNMENT

(I) Define stress and point by point discuss the management of stress.

(ii) Mention three types of stress reaction and discuss any one of your choice.

(iii) Discuss fully the physiology of stress.

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UNIT 4 PHYSIOLOGY OF STRESS

CONTENTS

1.0 INTRODUCTION

Stress is an inherent aspect of emergency medical services. Only in recent years has stress been recognised as a bona fide hazard of emergency work. Prehospital personnel must learn to manage stress , as well as the conditions that cause it. Dealing with stress in a positive manner promotes emotional and physical health and prevents burnout.

2.0 OBJECTIVES

At the end of this unit, you should be able to:

- define the term stress
- describe the stress reaction, including the various psychological and physiological components
- describe the three stages of the body's response to stress.
- define the term critical incident, and describe critical incident stress
- describe the three types of stress reaction
- name common causes of job stress for the paramedic
- list the techniques the paramedic can use to deal with stress
- describe the purpose of critical incident stress debriefing.

3.0 MAIN CONTENT

3.1 Physiology of Sress

The word stress literally means a hardship , force, or strain. From a psychological standpoint , stress can be defined as a state of physical

and psychological arousal. It exists to some degree in everyone. And a stressor is any agent or situation that causes stress. Often stress results from a perceived imbalance between the demands of the job ana our ability to meet those demands. In addition, stress may be increased by the demands imposed by the people and events around us.

The body's response to stress is termed a stress reaction. The stress reaction is complex and involves many body systems. After detection of the stressor , information received from the various senses is processed and interpreted in the cerebral cortex. The cerebral cortex is the part of the brain that controls higher mental functions and intellect. The limbic system , located in the midbrain , is subsequently activated and emotional reaction (fear, anger, rage, hostility etc) occurs. The response then proceeds to lower brain centers, including the hypothalamus .

The hypothalamus controls many bodily functions, including temperature and sleep. It also regulates the endocrine system. Once stimulated by the hypothalamus , the endocrine releases hormones such as epinephrine(adrenalin) and norepinephrine(noradrenalin) in response to the stress. The release of epinephrine further stimulates the brain. The body quickly enters a hyperalert state commonly called the fight-or-flight response or alarm reaction. The fight-or-flight response is characterized by an increase in heart rate and blood pressure, pupillary dilation, excessive perspiration, increased muscle tension, increased blood glucose level , and a sense of anxiety. This response prepares the body to deal with any threats, real or perceived.

3.2 The Body's Response to Stress

The body is under various levels of stress at all times. However, after repeated exposure to the same stressor, the body will adapt by suppressing the typical emotional and physical responses. The body's response to stress generally goes through the following stages:

(i) Stage 1: Alarm. An alarm reaction occurs at the first exposure to the stressor. The signs include include increased pulse rate, pupillary dilation and other responses of the sympathetic nervous system. If resistance to stress is diminished, the physiological and emotional response can be overwhelming .

(ii) Stage 11: Resistance. This stage starts when the individual begins to adapt to the stress. Resistance is often brought about by use of various coping mechanisms. Physiological parameters , such as pulse and blood pressure, may return to normal. As adaptation develops, resistance increases above normal level.

(iii) Stage 111: Exhaustion. Prolonged exposure to the same stressors leads to exhaustion of an individual 's adaptation energy. The

signs of the alarm reaction reappear, but they are now much more difficult to reverse.

3.3 TYPES OF STRESS REACTIONS

A stress reaction has both physical and psychological components. There are three types of stress reactions that can occur: acute, delayed and cumulative stress.

Acute stress reaction The acute stress reaction usually occurs after a catastrophic event. The reaction has a powerful emotional impact on the rescuer. Catastrophic events capable of evoking such a response have been labeled critical incidents. The acute stress reaction that follows is called critical incident stress. The acute stress reaction may begin ate the scene or shortly after the event. Several warning signs or symptoms characterize its onset. Some signs and symptoms require immediate intervention while others do not. Early intervention will help minimize the long-term effects of stress. Critical Incident Debriefing(CISD) helps to effectively deal with stress and prevent post-traumatic stress disorder.

Delayed stress reaction Delayed stress reaction, also called post-traumatic stress disorder, occurs days, weeks, months, or even years after a critical incident. Not all personnel who experience a critical incident will suffer a delayed stress reaction-but some will. The following signs or symptoms characterize post-traumatic stress disorder:

(i) Reexperiencing of the traumatic event:
(a) Recurrent and intrusive recollections of the event
(b) Recurrent dreams and nightmares related to the event
(c) Flashbacks, or sudden feelings that the event is recurring, usually after exposure to some triggering stimulus.
(ii) Diminshed responsiveness to the external world:
(a) Decreased interest in life
(b) Feeling detached or enstranged from others.
(c) Suppression of normal emotional responses such as love, anger, or fear.
(iii) Physical and Cognitive symptoms
(a) Hyperalertness
(b) Difficulty sleeping
(c) Survivor guilt
(d) Memory impairment and difficulty concentrating.
(e) Avoidance of any activities that may cause recall of the event
(f) Avoidance of thoughts or feelings associated with the incident
(g) Problems with interpersonal relationships

Post-traumatic stress disorder can interfere with your life. It can provoke marital problems , alcohol and drug abuse, personality changes, and even suicide. Because of this, persons suffering from a post-traumatic stress disorder should seek expert professional help.

Cumulative stress reaction: The third type of stress reaction is called a Cumulative stress reaction, or Burnout. Cumulative stress, unlike the types of stress discussed earlier, does not result from a single critical incident. Instead, it seems from recurring minor stressors, both work and non-work related. A cumulative stress reaction typically takes years to develop. Initially, the person will experience depression, boredom, apathy, and emotional fatique. As the reaction progresses, the person will experience sleep disturbances, increasing loss of emotional control, frequent physical complaints, physical and emotional fatique, irritability, and worsening depression. Ultimately, the person will develop extreme fatique , severe depressio, feelings of paranoia, crying spells, loss of sexual drive, inability to perform a job, significant problems managing his or her personal life, and occasionally suicidal or homicidal thinking.

Failure to intervene in the early stages of cumulative stress will result in severe disability for the person involved. Often he or she will suffer destruction of close family relationships, divorce, and inability to work in his or her chosen profession. The best treatment for the cumulative stress reaction is prevention. EMS systems should have programs that routinly screen their personnel for signs and symptoms of cumulative stress, such programs will allow for intervention, thus avoiding the condition's devastating consequences. Also, stressful assignments, such as busy stations or those with a higher incidence of critical patients, should be shared among personnel.

3.3 Paramedic Job Stress

EMS work involves a lot of stress. Some of the occupational stressors include:

(i) Multiple Role Responsibilities: The paramedic is often a jack of all trades. The various responsibilities may become overwhelming. This is particularly true of systems in which paramedics also function as fire-fighters, police officers,or safety officers.

(ii) Unfinished Tasks. The requirements of the job often leave personnel and work tasks incomplete. For example once a patient is delivered to the hospital , you often lose track of the person with whom you spent a great deal of time and energy treating. On a continuous basis, this can result in stress.

(iii)Angry or confused Citizens. Paramedics do not society at its best. The very people who require our help can be a source of stress, as a

result of either their physical condition or their emotional response to stress.

Critical incident stress debriefing

Critical incident stress Debriefings are structured group meetings that allow emergency and rescue personnel to discuss their feelings and other reactions after a critical incident. They are not psychotherapy or psychological treatment. They are however, designed to reduce the impact of a critical event and to accelerate the normal recovery of normal people. Remember that it is normal to suffer painful reactions to an abnormal event. Abnormal reactions occur when such feelings are not shared. Therefore, every system should offer Critical Incident Stress Debriefing or similar programs to personnel who encounter a critical incident.

4.0 CONCLUSION

In this unit you have learnt about stress, physiology of stress, body's response to stress and others. You now must learn to manage stress in order to survive in emergency medical services. You must recognise the early warning signs of anxiety. Remeber that some stress is valuable.

5.0 SUMMARY

Stress is a part of emergency medical services. You should recognise that a certain level of stress is important. You should also recognise that increasing stress can seriously affect personal health and job performance. As a paramedic you must learn to adapt to stress and to deal with it positively. You must also learn to deal with stress in other people.

6.0 TUTOR-MARKED ASSIGNMENT

(a) Define the term stress.
(b) Describe the stress reaction,including the various psychological and physiological components.
(c) Describe the three stages of the body's response to stress.
(d) Describe the term critical incident , and describe critical incident stress.
(e) Describe the types of stress reactions.

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UNIT 5 ANXIETY, DEATH AND DYING

CONTENTS

1.0 INTRODUCTION

Anxiety is an emotional state caused by stress. It is a major cause for the development of defense mechanism. This unit will help you to acquire a general overview of anxiety and further assist you to understand more of what the condition is all about. Secondly, you will also learn about death and dying which is part of emergency medical services.

2.0 OBJECTIVES

At the end of this unit, you should be able to:

- define anxiety
- know different types of management
- define death and dying
- know how to manage them.

3.0 MAIN CONTENT

3.1 Anxiety

This is a state of intense apprehension, uncertainty, and fear resulting from the anticipation of a threatening event or situation, often to a degree that normal physical and psychological functioning is disrupted.

It is also an emotioncharacterised by an unpleasant state of inner turmoil, often accompanied by nervous behavior, such as pacing back and forth, somatic complaints and rumination. It is the subjectively unpleasant feelings of dread over anticipated events, such as the feeling of imminent death. Anxiety is not the same as fear, which is a response to a real or perceived immediate threat; whereas anxiety is the expectation of future threat. Anxiety is a feeling of fear, worry, and uneasiness, usually generalized and unfocused as an overreaction to a situation that is only subjectively seen as menacing. It is often accompanied by muscular tension, restlessness, fatigue and problems in concentration. Anxiety can be appropriate, but when experienced regularly the individual may suffer from an anxiety disorder.

People facing anxiety may withdraw from situations which have provoked anxiety in the past. There are different types of anxiety. Existential anxiety can occur when a person faces angst, an existential crisis, or nihilistic feelings. People can also face test anxiety, mathematical anxiety, stage fright or somatic anxiety. Another type of anxiety, stranger anxiety and social anxiety are caused when people are apprehensive around strangers or other people in general. Anxiety can be either a short term 'state' or a long term "trait". Anxiety disorders are a group of mental disorderscharacterised by feelings of anxiety and fear, whereas trait anxiety is a worry about future events, close to the concept of neuroticism. Anxiety disorders are partly genetic but may also be due to drug use including alcohol and caffeine, as well as withdrawal from certain drugs. They often occur with other mental disorders, particularly major depressive disorder, bipolar disorder, certain personality disorders, and eating disorders. Common treatment options include lifestyle changes, therapy, and medications.

3.1.1 Descriptions

Anxiety is distinguished from fear, which is an appropriate cognitive and emotional response to a perceived threat and is related to the specific behaviors of fight-or-flight responses, defensive behavior or escape. It occurs in situations only perceived as uncontrollable or unavoidable, but not realistically so. David Barlow defines anxiety as "a future-oriented mood state in which one is ready or prepared to attempt to cope with upcoming negative events," and that it is a distinction between future and present dangers which divides anxiety and fear. Another description of anxiety is agony, dread, terror, or even apprehension. In positive psychology, anxiety is described as the mental state that results from a difficult challenge for which the subject has insufficient coping skills.

Fear and anxiety can be differentiated in four domains: duration of emotional experience, temporal focus, specificity of the threat, and motivated direction. Fear is defined as short lived, present focused, geared towards a specific threat, and facilitating escape from threat; while anxiety is defined as long acting, future focused, broadly focused towards a diffuse threat, and promoting excessive caution while approaching a potential threat and interferes with constructive coping. Anxiety can be experienced with long, drawn out daily symptoms that reduce quality of life, known as chronic (or generalised) anxiety, or it can be experienced in short spurts with sporadic, stressful panic attacks, known as acute anxiety.

Symptoms of anxiety can range in number, intensity, and frequency, depending on the person. While almost everyone has experienced anxiety at some point in their lives, most do not develop long-term problems with anxiety. The behavioral effects of anxiety may include withdrawal from situations which have provoked anxiety in the past. Anxiety can also be experienced in ways which include changes in sleeping patterns, nervous habits, and increased motor tension like foot tapping.The emotional effects of anxiety may include "feelings of apprehension or dread, trouble concentrating, feeling tense or jumpy, anticipating the worst, irritability, restlessness, watching (and waiting) for signs (and occurrences) of danger, and, feeling like your mind's gone blank" as well as "nightmares/bad dreams, obsessions about sensations, , and feeling like everything is scary."

The cognitive effects of anxiety may include thoughts about suspected dangers, such as fear of dying. "You may ... fear that the chest pains are a deadly heart attack or that the shooting pains in your head are the result of a tumor or aneurysm. You feel an intense fear when you think of dying, or you may think of it more often than normal, or can't get it out of your mind."As has been explained above, anxiety alerts a person to impending or perceived danger facilitated through the sympathetic nervous system. Anxiety maintains all potential resources, emotional and physical, in readiness for emergencies. Anxiety is related to each individual’s perception of the environment around him or her. It is based upon a person’s psychological processes and personal history. The factors that lead to anxiety can be divided into two general categories: normal and detrimental.

3.1.2 Normal Anxiety Levels

A normal level of anxiety varies from individual to individual. It acts as a warning system to put us on guard, so we won’t be overwhelmed by a sudden stimulation or immobilized in a critical situation. Normal anxiety may be considered adaptive, because it has evolved to help us cope with

stressors by focusing our attention. It helps us increase our tolerance for stress by developing coping and/ or defense mechanisms.

This process is evident in emergency medical services. The first emergency response that an emergency medical team or paramedic makes is a very stressful event. The person experiences a high pulse rate, dilated pupils, poorly organised thought process, and a feeling of tension. However, over time the body adapts to stresseach response more and more routine. However, as long as the emergency medical team (EMT) or paramedic is, on call, for emergency response, his or her level of anxiety never returns to a non-work state. Keeping anxiety at an, on-alert, is a coping mechanism. Because it is impossible to predict and prepare for the next problem, anxiety helps the paramedic maintain a level of readiness.

3.1.3 Detrimental Anxiety Levels

Although many reactions to anxiety and stress are positive, there are also detrimental ones .Detrimental reactions include the failure of anxiety to stimulate the appropriate coping mechanisms. Conversely, an increase in anxiety that is disproportionate to the actual danger would also be detrimental. These reactions may interfere with a rational thought process, disrupt performance, or cause physical problems. Symptoms of anxiety include:

(i) Heart palpitations

(ii) Difficult or rapid breathing

(iii) Dry mouth

(iv) Chest tightness or pain

(v) Anorexia, nausea, vomiting, abdominal cramps, flatulence, or the classic butterflies in the stomach.

(vi) Flushing, diaphoresis or fluctuation in the body temperature

(vii) Urgency or frequency of urination

(viii) Dysmenorrhea, or decreased sexual drive or performance

(ix) Aching muscles or joints

(x) Backache or headache.

Effects that are not felt include:

(i) Increased blood pressure and heart rate

(ii) Blood shunting to muscles

(iii) Increased blood glucose levels

(iv) Increased catecholamine production by the adrenal glands

(v) Reduced peristalsis in the digestive tract

(vi) Pupillary dilation.

People react differently to stress. The patient and family may react with anger, guilt, or indecisiveness. As a paramedic, you may react with impatience, fear, or anger. It is important to remember that the patient and family are not as adept at dealing with stress as you the professional. Because of indecisiveness, the patient and family members should not be given too many alternatives. Despite your emotions, you must maintain a professional attitude and non- judgmental. Common treatment options include lifestyle changes, therapy, and medications. Medications are typically recommended only if other measures are not effective. Early warning signs of anxiety must be recognised.

3.2 Death and Dying

Death is the end of life while dying is the process of approaching death, including the choices and actions involved in that process , it can also be defined as the cessation of all vital functions of the body including the heartbeat, brain activity(including the brain stem), and breathing. Phenomena which commonly bring about death include biological aging (senescence), predation, malnutrition, disease, suicide, homicide, starvation, dehydration, and accidents or trauma resulting in terminal injury. Bodies of living organisms begin to decompose shortly after death. Death has commonly been considered a sad or unpleasant occasion, due to the termination of bonds with or affection for the being that has died, or having fear of death, necrophobia, anxiety, sorrow, grief, emotional pain, depression, sympathy, compassion, solitude, or saudade.

3.2.1 Signs of Biological Death

Signs of death or strong indications that a warm-blooded animal is no longer alive are:

- Cessation of breathing
- Cardiac arrest (no pulse)
- *Pallor mortis*, paleness which happens in the 15–120 minutes after death
- *Livor mortis*, a settling of the blood in the lower (dependent) portion of the body
- *Algor mortis*, the reduction in body temperature following death. This is generally a steady decline until matching ambient temperature
- *Rigor mortis*, the limbs of the corpse become stiff (Latin *rigor*) and difficult to move or manipulate
- Decomposition, the reduction into simpler forms of matter, accompanied by a strong, unpleasant odor.

3.2.2 Causes of Death

The leading cause of human death in developing countries is infectious disease. The leading causes in developed countries are atherosclerosis (heart disease and stroke), cancer, and other diseases related to obesity and aging.

By extremely wide margin, the largest unifying cause of death in the developed world is biological aging, leading to various complications known as aging-associated diseases. These conditions cause loss of homeostasis, leading to cardiac arrest, causing loss of oxygen and nutrient supply, causing irreversible deterioration of the brain and other tissues. Of the roughly 150,000 people who die each day across the globe, about two thirds die of age-related causes. In industrialized nations, the proportion is much higher, approaching 90%.With improved medical capability, dying has become a condition to be managed. Home deaths, once commonplace, are now rare in the developed world.

- In developing nations, inferior sanitary conditions and lack of access to modern medical technology makes death from infectious diseases more common than in developed countries. One such disease is tuberculosis, a bacterial disease which killed 1.7M people in 2004. Malaria causes about 400–900M cases of fever and 1–3M deaths annually. AIDS death toll in Africa may reach 90–100M by 2025.
- According to Jean Ziegler (United Nations Special Reporter on the Right to Food, 2000—Mar 2008), mortality due to malnutrition accounted for 58% of the total mortality rate in 2006. Ziegler says worldwide approximately 62M people died from all causes and of those deaths more than 36M died of hunger or diseases due to deficiencies in micronutrients.

- Tobacco smoking killed 100 million people worldwide in the 20th century and could kill 1 billion people around the world in the 21st century, a WHO Report warned.
- Many leading developed world causes of death can be postponed by diet and physical activity, but the accelerating incidence of disease with age still imposes limits on human longevity. The evolutionary cause of aging is, at best, only just beginning to be understood. It has been suggested that direct intervention in the aging process may now be the most effective intervention against major causes of death.
- In 2012, suicide overtook car crashes for leading causes of human injury deaths in America, followed by poisoning, falls and murder. Causes of death are different in different parts of the world. In high-income and middle income countries nearly half up to more than two thirds of all people live beyond the age of 70 and predominantly die of chronic diseases. In low-income countries, where less than one in five of all people reach the age of 70, and more than a third of all deaths are among children under 15, people predominantly die of infectious diseases.

Autopsy

An autopsy, also known as a *postmortem examination* or an *obduction*, is a medical procedure that consists of a thorough examination of a human corpse to determine the cause and manner of a person's death and to evaluate any disease or injury that may be present. It is usually performed by a specialisedmedical doctor called a pathologist.Autopsies are either performed for legal or medical purposes. A forensic autopsy is carried out when the cause of death may be a criminal matter, while a clinical or academic autopsy is performed to find the medical cause of death and is used in cases of unknown or uncertain death, or for research purposes. Autopsies can be further classified into cases where external examination suffices, and those where the body is dissected and an internal examination is conducted. Permission from next of kin may be required for internal autopsy in some cases. Once an internal autopsy is complete the body is generally reconstituted by sewing it back together. Autopsy is important in a medical environment and may shed light on mistakes and help improve practices.

- A "necropsy" is an older term for a postmortem examination, unregulated, and not always a medical procedure. In modern times the term is more often used in the postmortem examination of the corpses of animals.

3.2.3 Grief Process

The family of a dying patient, as well as the patient, goes through a grief process initially. The grief process has several identifiable stages;

(i) Denial and Isolation; this stage is used by most dying patients. It is healthy and acts as a mental buffer between the shock of dying and dealing with it. It happens throughout the illness. It is temporary stage, often giving way to acceptance.

(ii) Anger: In the anger phase, the patient and the family ask, Why me? People are angered at the loss and may project their anger to anything and anyone. It is important to remember that this anger has little to do with the people or things present; they are often simply, targets. The anger can be difficult for you to deal with. Try not to take the patients or the family's anger personally. Be tolerant and be don't be afraid of anger. Don't become defensive. Listen to the patient and family.

(iii) Bargaining: Bargaining is a defensive mechanism use by the dying patient to formulate some sort of agreement, which in the patient'smind, postpones the inevitable.

(iv) Depression: Depression is common and expected. It is a normal response to the greatest loss. In reactive depression, the dying patient reacts to the need of a life situation. For example, who will care for the children or take care of the funeral arrangements? There is also preparatory depression. In this state, the patient is often silent and reassurance is not meaningful.

(v) Acceptance: Acceptance may not be a happy stage. At this point, the patient is without fear and despair. He or she is devoid of feelings. The patient becomes less involved with people as he or she prepares face death alone. At this stage, the family needs help, understanding and support more than the patient.

It is important to recognise the needs of individuals when dealing with the dead or dying. The dying patient needs dignity and respect, sharing, communication, hope, privacy, and control. The family has needs, too. They often go through a grief process similar to the patient's. They may need to express their feelings of rage, anger and despair. In addition they need to reduce their feelings of guilt. You may also go through some grief stages. This coping requires a lot of energy to cover feelings. It should be followed by adequate time for reflection and discussion.

3.2.4 Treatment/Management of the Dead or Dying Patient

How you react to death and the dying patient reflects your own thoughts and beliefs. It is natural to feel uncomfortable. Don't bring up the subject of death. Let the patient do so. Don't falsely reassure the patient or the family. Do not be afraid to tell the patient that he or she is dying, if asked. Use non-verbal communications, such as a gentle tone of voice, appropriate facial expression, and a reassuring touch .If the patient is already dead, the family becomes, the patient. Comfort the family with kind deeds, such as calling neighbours , family members, or a minister . The family needs to hear the word dead.

Avoid, Euphemisms like expired, passed away, or moved on. Always refer to the deceased patient by their name. Recognise that the family will cope with death in much the same manner as they deal with everyday stresses. Only recently has there been concerted public effort to address the care of the dying in an effort to improve their comfort and lessen their alienation from those still living. Hospice care represents one of the greatest advances made in this direction. There has also been a liberalization of the use of narcotics and other drugs for symptomatic relief and improvement in the quality of life for the dying.

Living will

One of the most difficult issues surrounding death in the era of technology is that there is now a choice, not of the event itself, but of its timing. When to die, and more often, when to let a loved one die, is coming within people's power to determine. This is both a blessing and a dilemma. Insofar as the decision can be made ahead of time, a living will is an attempt to address this dilemma. By outlining the conditions under which one would rather be allowed to die, a person can contribute significantly to that final decision, even if not competent to do so at the time of actual death. The problem is that there are uncertainties surrounding every severely ill person. Each instance presents a greater or lesser chance of survival. The chance is often greater than zero. The best living will follows an intimate discussion with decision makers covering the many possible scenarios surrounding the end of life. This discussion is difficult, for few people like to contemplate their own demise. However, the benefits of a living will are substantial, both to physicians and to loved ones who are faced with making final decisions. Most states have passed living will laws, honoring instructions on

artificial life support that were made while a person was still mentally competent.

Euthanasia

Another issue that has received much attention is assisted suicide (euthanasia). In 1997, the State of Oregon placed the issue on the ballot, amid much consternation and dispute. Perhaps the main reason euthanasia has become front page news is because Dr. Jack Kevorkian, a pathologist from Michigan, is one of its most vocal advocates. The issue highlights the many new problems generated by increasing ability to intervene effectively in the final moments of life and unnaturally prolong the process of dying. The public appearance of euthanasia has also stimulated discussion about more compassionate care of the dying.

Prevention

Autopsy after death is a way to precisely determine a cause of death. The word autopsy is derived from Greek meaning to see with one's own eyes. A pathologist extensively examines a body and submits a detailed report to an attending physician. Although an autopsy can do nothing for an individual after death, it can benefit the family and, in some cases, medical science. Hereditary disorders and disease may be found. This knowledge could be used to prevent illness in other family members. Information culled from an autopsy can be used to further medical research. The link between smoking and lung cancer was confirmed from data gathered through autopsy. Early information about AIDS was also compiled through autopsy reports.

4.0 CONCLUSION

Anxiety,Death and Dying are all part of emergency medical services. It is important to develop an appropriate personal attitude about them. As public health practioner you must learn to adapt and deal with these situations positively.

5.0 SUMMARY

Anxiety, death and dying are all part of emergency situations. This unit has focused on descriptions of Anxiety, Death and Dying, their signs, management and preventions. You should now be able to explain them in your own words.

6.0 TUTOR-MARKED ASSIGNMENT

(I) Describe the normal anxiety levels. What are its merits?

(ii) Define death and dying. In your own words discuss the causes of death.

(iii) Discuss in details the Grief process and the management of death or dying patient.

7.0 REFERENCES/FURTHER READING

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MODULE 3

UNIT 1 VIOLENT INJURIES

CONTENTS

1.0 INTRODUCTION

An estimated 815,000 people killed themselves in year 2000, making suicide the 13th leading cause of death worldwide. This unit will help you understand what violence is and its basic components.

2.0 OBJECTIVES

At the end of this unit, you should be able to:

- define the term violence
- differentiate types of violence
- discuss risk factors in violence
- define suicide
- mention the prevention of violence.

3.0 MAIN CONTENT

3.1 Definition of Violence

The World Health Organisation (WHO) defines violence as “the intentional use of physical force or power, threatened or actual, against one self, another person, or against a group or community that either results in or has a high likelihood of resulting in physical, sexual or

psychological injury or death."

Types of violence
Three broad categories of violence are recognised; depending upon who commits the violent act.

Self-directed violence
This is the type of violence a person inflicts upon himself or herself. This includes suicidal behaviour and self -abuse. Suicidal behaviour ranges from merely thinking about ending one's life, to planning it, finding the means to do so, attempting to kill self and completing theact.

Interpersonal violence
This **v**iolence is the type inflicted by another individual. This category includes family and intimate partner violence, child and elderly abuse, youth violence, rape or sexual assault, and violence between unrelated individuals.

Collective violence
The violence is between two groups of people. It includes armed conflict within or between states, genocide, repression, human rights abuses, terrorism; organised crime etc. Collective violence is inflicted by groups.

3.2 Risk Factors

Violence is often predictable and preventable. It occurs due to an interaction of multiple factors that could be biological, social, cultural, economic, political and religious. There is no single factor to explain why one person and not another behaves in a violent manner.

Risk factors levels
The "WHO Report on Violence and Health" uses an ecological model to understand the multifaceted nature of violence. These are:

Individuals: Their personal characteristics i.e. age, education, income, personality disorders, substance abuse and a history of behaving aggressively or experiencing abuse.

Relationship: With friends, family, intimate partners and peers.

Community: In schools, workplaces and neighborhoods in which these relationships are taking place.

Society: Violence is more common in societies which encourage such behaviours.

Religion: Wanting to dominate one another, owners of religions or worship places, inciting sermons, etc. Factors at each level are strengthened or modified by factors at another. This is prevented only by planned programmes after understanding thecontext of violence and its specific risk factors in the targeted population.

3.3 Suicide

This is the act or attempt to kill one's self intentionally, ruin one's own interest. Suicide rates vary within countries, between urban and rural populations, and between different racial and ethnic groups.

Risk factors: Stressful events predisposing to self -harm include: poverty, unemployment, and loss of loved one, argument with family or friends, breakdown in relationship andlegal or work-related problems.

Personalities at risk: Alcohol and drug abusers, those with psychiatric problems, history of physical or sexual abuse, and social isolation have a high rate of violence. A generalsense of hopelessness also plays a role. Chronic physical illness, particularly those that are painful or disabling, can also predispose one to self -harm.

Means to kill: Most typically, guns, medicines, and agricultural poisons are important determinants of success. History of previous suicide attempt also predisposes one tosubsequent fatal suicidal behaviour.

Protective Factors: Good relationship with family and friends, high self -esteem, social connectedness, happy marriage, and commitment to a religion appear to protect peopleagainst the desire to commit suicide.

3.4 Prevention of Violence

Violence is not inevitable, much can be done to address and prevent it, and our main emphasis will be on primary prevention of violence, i.e. aiming to prevent violence before it occurs.

General population: Universal Intervention.

Selected intervention: Individuals or groups having one or more risk factors for violence.

Indicated intervention: Those who have demonstrated violent behaviours.

4.0 CONCLUSION

This unit has exposed you to violence and the types of violence which are very common in our society. Also, suicide and suicidal attempts have been discussed. You should be able to explain in your own words what violence and suicide mean and also state ways of preventing them.

5.0 SUMMARY

The focus of this unit is to define the term violence and suicide which are both intentional in nature. Risk factors associated with each exposes the person to injury and death.

6.0 TUTOR-MARKED ASSIGNMENT

1. Briefly, discuss the three categories of violence that you know.
2. Define suicide and explain its preventive measures.

7.0 REFERENCES/FURTHER READING

"The Injury Chart Book: WHO 2002"
www.who.int/violence_injury_prevention.

World Health Organisation, Department of Injury and Violence Prevention www.who.int/violence_injury_prevention/en.

UNIT 2 POISONING

CONTENTS

1.0 INTRODUCTION

In year 2000, 315,000 persons died worldwide due to unintentional poisoning. More than 94% of total reported cases of poisoning occurred in low and middle income countries.This unit will help you acquire basic understanding of what poisoning is.

2.0 OBJECTIVES

At the end of this unit, you should be able to:

- define poisoning , terminology involved, decontamination
- discuss types of poisoning and state the diagnosis of poisoning
- explain the management of poisoning and list the preventive measures of poisoning.

3.0 MAIN CONTENT

3.1 Definition of Poisoning

Poisons are substances that cause disturbances to organisms including human, produces ill-health, disease or death, usually by chemical reactions or other activity on the molecular scale, when a sufficient quantity is absorbed by an organism.

Poisoning refers to all unintentional poisoning related deaths and nonfatal outcomes caused by exposure to noxious substances; those which are intentional, or for which the intent is determined as well as those resulting from reactions of drugs.The field of medicine and zoology often distinguishes a poison from toxin, and from venom. Toxins are poisons produced by some biological functions in nature, and venoms are usually defined as toxins that are injected by a bite or sting to cause their effects, while other poisons are generally defined as substances absorbed through epithelial linings such as skin or gut.

3.1.2 Terminology

The term poison is often used colloquially to describe any harmful substances, particularly corrosive substances, carcinogens, mutagens, teratogens and harmful pollutants, and to exaggerate the dangers of chemicals. Paraceisus(1493-1541), the father of toxicology, once wrote: Everything is POISON, there is poison in everything. Only the dose makes a thing not a poison. The law defines POISON more strictly; substances that are not legally required to carry the label, Poison, can also cause a medical condition of poisoning.

Some poisons are also toxins, usually referring to naturally produced substances, such as the bacterial proteins that cause tetanus and botulism. A distinction between the two terms is not always observed, even among scientist. The derivative forms, TOXIC, and,POISONOUS, are synonymous. Animal poisons that are delivered subcutaneously (eg by sting or bite) are also called venoms. In normal usage, a poisonous organism is one that is harmful to consume, but a venomous organism uses venom to kill its prey or defend itself while still alive. A single organism can be both poisonous and venomous. In nuclear physics, a poison is a substance that obstructs or inhibits a nuclear reaction.

Environmentally hazardous substances are not necessarily poisons and vice versa. For example, food-industry waste-water which may contain potato juice or milk-can be hazardous to the ecosystems of streams and rivers by consuming oxygen and causing eutrophication, but is non-hazardous to humans and classified as a poison. Biologically speaking, any substance, if given in large amounts, is poisonous and cause death. For instance, several kilograms worth of water would constitute a lethal dose. M any substances used as medications-such as fentanyl- have an LD50 only one order of magnitude greater than the ED50.

Poisoning: Acute poisoning is exposure to a poison on one occasion or during a short period of time. Symptoms develop in close relation to the exposure. Absorption of a poison is necessary for systemic poisoning. In contrast, substances that destroy tissue but do not absorb, such as Iye,

are classified as corrosives rather than poisons. Furthermore, many common, household medications are not labelled with skull and crossbones, although, they can cause severe illness or even death. In the medical sense, poisoning can be caused by less dangerous substances than those receiving the legal classification of POISON. Chronic poisoning is long-term repeated or continuos exposure to a poison where symptoms do not occur immediately or after each exposure. The patient gradually becomes ill, or becomes ill after a long latent period. Chronic poisoning most commonly occurs following exposure to poison that bioaccumulate, or are biomagnified, such as mercury, gadolinium and lead. Contact or absorption of poison can cause rapid death or impairment.

Agents that act on the nervous system can paralyse in seconds or less, and include both biologically derived neurotoxins and so-called nerve gases which may synthesised for warfare or industry . Inhaled or ingested cyanide, used as a method of execution in gas chambers, almost instantly starves the body of energy by inhibiting the enzymes in mitochondria that make ATP. Intravenous injections of an unnaturally high concentration of potassium chloride, such as the execution of prisoners in parts of United States, quickly stops the heart by eliminating the cell potential necessary for muscle contraction. Most biiocides, including pesticides, are created to act as poisons to target organisms, although, acute or less observable chronic poisoning can also occur in non- target organisms (secondary poisoning) ,including the human who apply the biocides and other beneficial organisms. For example, the herbicide 2,4-D imitates the action of a plant hormone, to the effect that the lethal toxicity is specific to plants. Indeed, 2,4-D is not a poison but classified as Harmful.

Many substances regarded as poisons are toxic only indirectly by toxication. An example is, Wood Alcohol, or Methanol, which is not poisonous itself, but is chemically converted to toxic formaldehyde and formic acid in the liver. Many drug molecules are made toxic in the liver, and the genetic variability of certain liver enzymes makes the toxicity of many compounds differ between individuals. Toxicology is the study of the symptoms, mechanisms, treatment and diagnosis of biological poisoning. Exposure to radioactive substances can produce radiation poisoning, an unrelated phenomenom.

3.1.2 Types of Poisoning

Poisoning can be classified as due to non-toxic or toxic ingestion.

Non-toxic ingestion
A non-toxic ingestion occurs after an individual consumes a non-edible product, that usually does not produce symptoms, such as abrasives, adhesives, air fresheners, aluminum foils, baby toys, cosmetics, candles, chalk, erasers, ink, lipstick, lubricants, etc.

Toxic ingestion
Household toxic ingestion consist of consumption of any of thefollowing: soaps and detergents, shampoos, bleaches, disinfectants, anddeodorizers, acid and alkalis, boron compounds, cosmetics, nail polishremover (gamma butyrolactone), disc, batteries, button cells,naphthalene moth balls, tobacco products, insecticides, pharmaceuticalsand paints.

3.1.3 Diagnosis of Poisoning

Many poisons and drugs taken in over dosage do not cause rapid loss of consciousness. The diagnosis of self-poisoning may be made almost always on clinical and circumstantial evidence. There are often medicine bottles and containers at the scene and a “suicide” note may be found; certain intoxication may produce a characteristic clinical picture.

3.1.4 Management

(i) Initial management for all poisoning includes ensuring adequate cardiopulmonary function and providing treatment for any symptoms such as seizures, shock, and pain.
(ii) Poisons that have been injected(e.g. from the sting of poisonous animals) can be treated by binding the affected body part with a pressure bandage and by placing the affected body part in hot water (with a temperature of 50c). The pressure bandage makes sure the poison is not pumped throughout the body and the hot water breaks down the poison. This treatment however, only works with poisons that are composed of protein molecules.
(iii) In the majority of poisonings, the mainstay of management is providing supportive care for the patient that is treating the symptoms rather than the poison.

3.1.5 Basic Management

Emergency stabilisation

Transport patient in the head-down semi prone position to minimise the risk of inhalation of gastric contents.

Evaluation

Complete evaluation by detailed history, physical examination and laboratory investigation directed towards identifying the toxic; or possibly, toxicology should be done. It helps in knowing the severity, complication and associated injuries.

Decontamination

To reduce the absorption of poison, the following can be carried out:

Eye decontamination

Immediately irrigate the affected tissue with neutralising solution (e.g. normal saline or water) copiously for at least 15-20 minutes.

Skin decontamination

In case of organ phosphorous poisoning, irrigate the whole body including nails, groin and skin-folds with water or saline as soon as possible; after exposure for 15 minutes, remove all contaminated clothes.

Gut decontamination:

Treatment of a recently ingested poison may involve gastric decontamination to decrease absorption**.**Gastric decontamination can involve activated charcoal, gastric lavage, whole bowel irrigation, or nasogastric aspiration. Routine uses of emetics, cathartics or laxatives are no longer recommended according to WHO.

(i) Activated charcoal is the treatment of choice to prevent poison absorption. It is usually administered when the patient is in the emergency room or by a trained emergency healthcare provider such as a paramedic or emergency medical team. However, charcoal is ineffective against metals such as sodium, potassium and lithium, and alcohols and glycols. It is also not recommended for ingestion of corrosive chemicals such as acids and alkalis.

(ii) Cathartics were postulated to decrease absorption by increasing the expulsion of the poison from the gastrointestinal tract. There are two types of Cathartics used in poisoned patients, saline cathartics(sodium sulphate, magnesium citrate, magnesium sulphate) and saccharide cathartics(Sorbitol). They do not appear to improve patient outcome and are no longer recommended.

(iii) Emesis (induced by Ipecac) is no longer recommended in poisoning situation because vomiting is ineffective of removing poisons.

(iv) Gastric lavage commonly known as stomach pump is the insertion of a tube into stomach, followed by administration of water or saline down the tube. The liquid is then removed along with the contents of the stomach. Lavage has been used for many years as a common treatment for poisoned patients. However, a recent review of the procedure in poisoning suggests no benefit. It is still sometimes used if it can be performed within 1 hour of ingestion and the exposure is potentially life-threatening.

(v) Nasogastric aspiration : Involves the placement of a tube via the nose down into the stomach, the stomach contents are then removed by suction. This procedure is mainly used for liquid ingestions where activated charcoal is ineffective e.g. ethylene glycol poisoning.

(vi) Whole bowel irrigation cleanses the bowel. This is achieved by giving the patient large amounts of polyethylene glycol solution. The osmotically balanced polyethylene glycol solution is not absorbed into the body, having the effect of flushing out the entire gastrointestinal tract. Its major use are following ingestion of sustained release drugs ,toxins that are absorbed by activated charcoal (lithium, iron) and for the removal of ingested packets of drugs(body packing/ smuggling).

Enhanced Excretion: In some situations, elimination of the poison can be enhanced using diuresis, hemodialysis, hemoperfusion, hyperbaric medicine, peritoneal dialysis, exchange transfusion, or chelation. However, this may actually worsen the poisoning in some cases, so it should always be verified based on what substances are involved.

3.1.6 Prevention of Poisoning

“Prevention is better than cure.” A large majority of poison does not have a specific antidote.

Parents should be educated regarding the dangers associated with medicine, household substances and agrochemicals. Parents must teach their children the danger of touching, eating or playing with unknown objects including medicine, pesticides and insecticides, household chemicals or plants berries.Medicines,pesticides and insecticides and other poisonoussubstances should be stored in locked cabinets.
Dyes, polishes, kerosene and other chemicals should never be lefton a low shelf or on the floor; do not store in kitchen orbathroom.Combustion services should be adequately ventilated.Inhalation of spray or fumes should be prevented during painting,or application of insecticides. Wear protective clothing,

goggles, gloves and masks. Dispense medicine and dangerous chemicals in childproof, tamperproof containers.Education on proper hygiene and storage of food in order to avoid food poisoning and training of workers on safe use of chemicals, etc.

3.2 Burns

A **burn** is a type of injury to flesh or skin caused by heat, electricity, chemicals, friction, or radiation. Burns that affect only the superficial skin are known as superficial or first-degree burns. When damage penetrates into some of the underlying layers, it is a partial-thickness or second-degree burn. In a full-thickness or third-degree burn, the injury extends to all layers of the skin. A fourth-degree burn additionally involves injury to deeper tissues, such as muscle or bone.

The treatment required depends on the severity of the burn. Superficial burns may be managed with little more than simple pain relievers, while major burns may require prolonged treatment in specialised burn centers. Cooling with tap water may help relieve pain and decrease damage; however, prolonged exposure may result in low body Temperature.Partial thickness burns may require cleaning with soap and water, followed by dressings. It is not clear how to manage blisters, but it is probably reasonable to leave them intact. Full-thickness burns usually require surgical treatments, such as skin grafting. Extensive burns often require large amounts of intravenous fluid, because the subsequent inflammatory response causes significant capillary fluid leakage and edema. The most common complications of burns involve infection.

While large burns can be fatal, modern treatments developed since 1960 have significantly improved the outcomes, especially in children and young adults. Globally, about 11 million people seek medical treatment, and 300,000 die from burns each year. In the United States, approximately 4% of those admitted to a burn center die from their injuries.The long-term outcome is primarily related to the size of burn and the age of the person affected.

3.2.1 Signs and Symptoms

The characteristics of a burn depend upon its depth. Superficial burns cause pain lasting two or three days, followed by peeling of the skin over the next few days.Individuals suffering from more severe burns may indicate discomfort or complain of feeling pressure rather than pain. Full-thickness burns may be entirely insensitive to light touch or puncture. While superficial burns are typically red in color, severe burns may be pink, white or black. Burns around the mouth or singed

hair inside the nose may indicate that burns to the airways have occurred, but these findings are not definitive.More worrisome signs include: shortness of breath, hoarseness, and stridor or wheezing. Itchiness is common during the healing process, occurring in up to 90% of adults and nearly all children. Numbness or tingling may persist for a prolonged period of time after an electrical injury. Burns may also produce emotional and psychological distress.

3.2.2 Cause

Burns are caused by a variety of external sources classified as thermal (heat-related), chemical, electrical, and radiation. In the United States, the most common causes of burns are: fire or flame (44%), scalds (33%), hot objects (9%), electricity (4%), and chemicals (3%). Most (69%) burn injuries occur at home or at work (9%), and most are accidental, with 2% due to assault by another, and 1-2% resulting from a suicide attempt. These sources can cause inhalation injury to the airway and/or lungs, occurring in about 6%.Burn injuries occur more commonly in the poor. Smoking is a risk factor, although alcohol use is not. Fire-related burns are generally more common in colder climates. Specific risk factors in the developing world include cooking with open fires or on the floor as well as developmental disabilities in children and chronic diseases in adults.

3.2.3 Thermal

In the United States, fire and hot liquids are the most common causes of burns. Of house fires that result in death, smoking causes 25% and heating devices cause 22%. Almost half of injuries are due to efforts to fight a fire. Scalding is caused by hot liquids or gases and most commonly occurs from exposure to hot drinks, high temperature tap water in baths or showers, hot cooking oil, or steam. Scald injuries are most common in children under the age of fiveand, in the United States and Australia, this population makes up about two-thirds of all burns. Contact with hot objects is the cause of about 20-30% of burns in children. Generally, scalds are first- or second-degree burns, but third-degree burns may also result, especially with prolonged contact. Fireworks are a common cause of burns during holiday seasons in many countries. This is a particular risk for adolescent males.

3.2.4 Chemical

Chemicals cause from two to 11% of all burns and contribute to as many as 30% of burns—Chemical burns can be caused by over 25,000 substances, most of which are either a strong base (55%) or a strong acid (26%). Most chemical burn deaths are secondary to ingestion. Common

agents include: sulfuric acid as found in toilet cleaners, sodium hypochlorite as found in bleach, and halogenated hydrocarbons as found in paint remover, among others.Hydrofluoric acid can cause particularly deep burns that may not become symptomatic until sometime after exposureFormic acid may cause the breakdown of significant numbers of red blood cells.

3.2.5 Electrical

Electrical burns or injuries are classified as high voltage (greater than or equal to 1000 volts), low voltage (less than 1000 volts), or as flash burns secondary to an electric arc. The most common causes of electrical burns in children are electrical cords (60%) followed by electrical outlets (14%). Lightning may also result in electrical burns. Risk factors for being struck include involvement in outdoor activities such as mountain climbing, golf and field sports, and working outside. Mortality from a lightning strike is about 10%. While electrical injuries primarily result in burns, they may also cause fractures or dislocations secondary to blunt force trauma or muscle contractions. In high voltage injuries, most damage may occur internally and thus the extent of the injury cannot be judged by examination of the skin alone.Contact with either low voltage or high voltage may produce cardiac arrhythmias or cardiac arrest.

3.2.6 Radiation

Radiation burns may be caused by protracted exposure to ultraviolet light (such as from the sun, tanning booths or arc welding) or from ionizing radiation (such as from radiation therapy, X-rays or radioactive fallout). Sun exposure is the most common cause of radiation burns and the most common cause of superficial burns overall.There is significant variation in how easily people sunburn based on their skin type.[26] Skin effects from ionizing radiation depend on the amount of exposure to the area, with hair loss seen after 3 Gy, redness seen after 10 Gy, wet skin peeling after 20 Gy, and necrosis after 30 Gy. Redness, if it occurs, may not appear until sometime after exposure. Radiation burns are treated the same as other burnsMicrowave burns occur via thermal heating caused by the microwaves. While exposures as short as two seconds may cause injury, overall this is an uncommon occurrence]

3.2.7 Non Accidental

In those hospitalised from scalds or fire burns, 3–10% are from assault. Reasons include: child abuse, personal disputes, spousal abuse, elder

abuse, and business disputes .An immersion injury or immersion scald may indicate child abuse. It is created when an extremity or the lower body (buttock or perineum) is held under the surface of hot water. It typically produces a sharp upper border and is often symmetrical.Other high-risk signs of potential abuse include: circumferential burns, the absence of splash marks, a burn of uniform depth, and association with other signs of neglect or abuse.Bride burning, a form of domestic violence, occurs in some cultures, such as India where women have been burned in revenge for what the husband or his family consider an inadequate dowry In Pakistan, acid burns represent 13% of intentional burns, and are frequently related to domestic violence. Self-immolation (setting oneself on fire) is also used as a form of protest in various parts of the world.

3.2.8 Pathophysiology

At temperatures greater than 44 °C (111 °F), proteins begin losing their three-dimensional shape and start breaking down. This results in cell and tissue damage. Many of the direct health effects of a burn are secondary to disruption in the normal functioning of the skin.They include disruption of the skin's sensation, ability to prevent water loss through evaporation, and ability to control body temperature. Disruption of cell membranes causes cells to lose potassium to the spaces outside the cell and to take up water and sodium.

In large burns (over 30% of the total body surface area), there is a significant inflammatory response. This results in increased leakage of fluid from the capillaries, and subsequent tissue edema. This causes overall blood volume loss, with the remaining blood suffering significant plasma loss, making the blood more concentrated. Poor blood flow to organs such as the kidneys and gastrointestinal tract may result in renal failure and stomach ulcers.Increased levels of catecholamines and cortisol can cause a hyper metabolic state that can last for years. This is associated with increased cardiac output, metabolism, a fast heart rate, and poor immune function.

3.2.9 Diagnosis

Burns can be classified by depth, mechanism of injury, extent, and associated injuries. The most commonly used classification is based on the depth of injury. The depth of a burn is usually determined via examination, although a biopsy may also be used. It may be difficult to accurately determine the depth of a burn on a single examination and repeated examinations over a few days may be necessary. In those who have a headache or are dizzy and have a fire-related burn, carbon

monoxide poisoning should be considered. Cyanide poisoning should also be considered.

3.2.10 Size

Burn severity is determined through, among other things, the size of the skin affected. The image shows the makeup of different body parts, to help assess burn size.The size of a burn is measured as a percentage of total body surface area (TBSA) affected by partial thickness or full thickness burns. First-degree burns that are only red in color and are not blistering are not included in this estimation. Most burns (70%) involve less than 10% of the TBSA.There are a number of methods to determine the TBSA, including the Wallace rule of nines, Lund and Browder chart, and estimations based on a person's palm size. The rule of nines is easy to remember but only accurate in people over 16 years of age. More accurate estimates can be made using Lund and Browder charts, which take into account the different proportions of body parts in adults and children. The size of a person's handprint (including the palm and fingers) is approximately 1% of their TBSA.

Severity To determine the need for referral to a specialised burn unit, the American Burn Association devised a classification system. Under this system, burns can be classified as major, moderate and minor. This is assessed based on a number of factors, including total body surface area affected, the involvement of specific anatomical zones, the age of the person, and associated injuries. Minor burns can typically be managed at home, moderate burns are often managed in hospital, and major burns are managed by a burn center.

3.2.11 Prevention

Historically, about half of all burns were deemed preventable. Burn prevention programs have significantly decreased rates of serious burns. Preventive measures include: limiting hot water temperatures, smoke alarms, sprinkler systems, proper construction of buildings, and fire-resistant clothing. Experts recommend setting water heaters below 48.8 °C (119.8 °F).Other measures to prevent scalds include using a thermometer to measure bath water temperatures, and splash guards on stoves. While the effect of the regulation of fireworks is unclear, there is tentative evidence of benefit with recommendations including the limitation of the sale of fireworks to children. Locally, promoting the use of safe stoves, replacement of pressure cooking stoves with more efficient wick and gas stoves. Introduction of more stable stands for lamps and stoves. Use less hazardous fuels. Fire drills for evacuation from large buildings and public places.Discourage smoking and increase public awareness of fire-related injuries.

3.2.12 Management

Resuscitation begins with the assessment and stabilisation of the person's airway, breathing and circulation. If inhalation injury is suspected, early intubation may be required. This is followed by care of the burn wound itself. People with extensive burns may be wrapped in clean sheets until they arrive at a hospital. As burn wounds are prone to infection, a tetanus booster shot should be given if an individual has not been immunised within the last five years In the United States, 95% of burns that present to the emergency department are treated and discharged; 5% require hospital admission. With major burns, early feeding is important. Hyperbaric oxygenation may be useful in addition to traditional treatments.

Intravenous fluids

In those with poor tissue perfusion, boluses of isotonic crystalloid solution should be givenIn children with more than 10-20% TBSA burns, and adults with more than 15% TBSA burns, formal fluid resuscitation and monitoring should follow.This should be begun pre-hospital if possible in those with burns greater than 25% TBSA. The Parkland formula can help determine the volume of intravenous fluids required over the first 24 hours. The formula is based on the affected individual's TBSA and weight. Half of the fluid is administered over the first 8 hours, and the remainder over the following 16 hours. The time is calculated from when the burn occurred, and not from the time that fluid resuscitation began. Children require additional maintenance fluid that includes glucose. Additionally, those with inhalation injuries require more fluid. While inadequate fluid resuscitation may cause problems, over-resuscitation can also be detrimental. The formulas are only a guide, with infusions ideally tailored to a urinary output of >30 mL/h in adults or >1mL/kg in children and mean arterial pressure greater than 60 mmHg.

While lactated Ringer's solution is often used, there is no evidence that it is superior to normal saline. Crystalloid fluids appear just as good as colloid fluids, and as colloids are more expensive they are not recommended. Blood transfusions are rarely required. They are typically only recommended when the hemoglobin level falls below 60-80 g/L (6-8 g/dL)[due to the associated risk of complications. Intravenous catheters may be placed through burned skin if needed or intraosseous infusions may be used.

Wound care

Early cooling (within 30 minutes of the burn) reduces burn depth and pain, but care must be taken as over-cooling can result in hypothermia Itshould be performed with cool water 10–25 °C (50.0–77.0 °F) and not ice water as the latter can cause further injury. Chemical burns may require extensive irrigation. Cleaning with soap and water, removal of dead tissue, and application of dressings are important aspects of wound care. If intact blisters are present, it is not clear what should be done with them. Some tentative evidence supports leaving them intact. Second-degree burns should be re-evaluated after two days.

In the management of first and second-degree burns, little quality evidence exists to determine which dressing type to use. It is reasonable to manage first-degree burns without dressings. While topical antibiotics are often recommended, there is little evidence to support their use. Silver sulfadiazine (a type of antibiotic) is not recommended as it potentially prolongs healing time. There is insufficient evidence to support the use of dressings containing silver or negative-pressure wound therapy.

Medications

Burns can be very painful and a number of different options may be used for pain management. These include simple analgesics (such as ibuprofen and acetaminophen) and opioids such as morphine. Benzodiazepines may be used in addition to analgesics to help with anxiety. During the healing process, antihistamines, massage, or transcutaneous nerve stimulation may be used to aid with itching. Antihistamines, however, are only effective for this purpose in 20% of people. There is tentative evidence supporting the use of gabapentin and its use may be reasonable in those who do not improve with antihistamines. Intravenous lidocaine requires more study before it can be recommended for pain.

Intravenous antibiotics are recommended before surgery for those with extensive burns .As of 2008, guidelines do not recommend their general use due to concerns regarding antibiotic resistance and the increased risk of fungal infections. Tentative evidence, however, shows that they may improve survival rates in those with large and severe burns. Erythropoietin has not been found effective to prevent or treat anemia in burn cases. In burns caused by hydrofluoric acid, calcium gluconate is a specific antidote and may be used intravenously and/or topically. Recombinant human growth hormone (rhGH) in those with burns that involve more than 40% of their body appears to speed healing without affecting the risk of death.

Alternative medicine

Honey has been used since ancient times to aid wound healing and may be beneficial in first- and second-degree burns. The evidence for Aloe Vera is of poor quality. While it might be beneficial in reducing pain, and a review from 2007 found tentative evidence of improved healing time[,a] subsequent review from 2012 did not find improved healing over silver sulfadiazine. There were only three randomised controlled trials for the use of plants for burns, two for Aloe Vera and one for oatmeal.

There is little evidence that vitamin E helps with keloids or scarring. Butter is not recommended. In low income countries, burns are treated up to one-third of the time with traditional medicine, which may include applications of eggs, mud, leaves or cow dung.Surgical management is limited in some cases due to insufficient financial resources and availability. There are a number of other methods that may be used in addition to medications to reduce procedural pain and anxiety including: virtual reality therapy, hypnosis, and behavioral approaches such as distraction techniques.

3.2.13 Prognosis

The prognosis is worse in those with larger burns, those who are older, and those who are females. The presence of a smoke inhalationinjury, other significant injuries such as long bone fractures and serious co-morbidities (e.g. heart disease, diabetes, psychiatric illness, and suicidal intent) also influence prognosis. On average, of those admitted to United States burn centers, 4% die, with the outcome for individuals dependent on the extent of the burn injury. For example, admitters with burn areas less than 10% TBSA had a mortality rate of less than 1%, while admitters with over 90% TBSA had a mortality rate of 85%.In Afghanistan, people with more than 60% TBSA burns rarely survive. The Beaux score has historically been used to determine prognosis of major burns. However, with improved care, it is no longer very accurate. The score is determined by adding the size of the burn (% TBSA) to the age of the person, and taking that to be more or less equal to the risk of death.

3.2.14 Complications

A number of complications may occur, with infections being the most common. In order of frequency, potential complications include: pneumonia, cellulitis, urinary tract infections and respiratory failure.Risk factors for infection include: burns of more than 30% TBSA, full-thickness burns, extremes of age (young or old), or burns involving the legs or perineum.Pneumonia occurs particularly commonly in those with inhalation injuries.Anemia secondary to full thickness burns of greater than 10% TBSA is common. Electrical burns

may lead to compartment syndrome or rhabdomyolysis due to musclebreakdown.Blood clotting in the veins of the legs is estimated to occur in 6 to 25% of people. The hyper-metabolic state that may persist for years after a major burn can result in a decrease in bone density and a loss of muscle mass. Keloids may form subsequent to a burn, particularly in those who are young and dark skinned. Following a burn, children may have significant psychological trauma and experience post-traumatic stress disorder] Scarring may also result in a disturbance in body image. In the developing world, significant burns may result in social isolation, extreme poverty and child abandonment.

3.2.15 Epidemiology

As of 2004, 11 million burns required medical care worldwide and resulted in 300,000 deaths. This makes it the 4th leading cause of injuries after motor vehicle collisions, falls, and violence. About 90% of burns occur in the developing world. This has been attributed partly to overcrowding and an unsafe cooking situation. Overall, nearly 60% of fatal burns occur in Southeast Asia with a rate of 11.6 per 100,000. The number of fatal burns has increased from 280,000 in 1990 to 338,000 in 2010.In the developed world, adult males have twice the mortality as females from burns. This is most probably due to their higher risk occupations and greater risk-taking activities. In many countries in the developing world, however, females have twice the risk of males. This is often related to accidents in the kitchen or domestic violence. In children, deaths from burns occur at more than ten times the rate in the developing than the developed world. Overall, in children it is one of the top fifteen leading causes of death. From the 1980s to 2004, many countries have seen both a decrease in the rates of fatal burns and in burnsgenerally.

4.0 CONCLUSION

In this unit, you have learned what poisoning is and the types of poisoning. You also learned their diagnosis and prevention measures. You have also learnt about burns, the types of burns, management, pathophysiology, diagnosis, complications and how to prevent their occurrences.

5.0 SUMMARY

This unit has exposed you to the definition of poisoning and burns. The various ways of management and prevention of poisoning and burns have been discussed.

6.0 TUTOR-MARKED ASSIGNMENT

1. Explain the term poisoning and state the types of poisoning that you know.
2a. State the basic management of poison.
b. Define the term burns.
c Discuss different types of burns and their management
d Mention ways of preventing burns at home and at work place.

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A Brief History of Acute Burn Care Management: Total Burn Care.

UNIT 3 FLUID AND ELECTROLYTES

CONTENTS

1.0 INTRODUCTION

The human body is not just an inert structure of bones and cavities and tubes. In fact, the has often been compared to a chemical laboratory or a factory because there is continual internal biochemical and physiological activity necessary to support life. Metabolism is the term for the totality of these ongoing biochemical and physiological processes.When the normal metabolism is disturbed, the body will usually respond in various ways to compensate and restore normal metabolism. If the metabolism is disturbed seriously enough, or when other traumatic or medical conditions prevent it, the body's compensatory mechanisms may not function or may not be sufficient to restore normal metabolism. When metabolic disturbances are not or cannot be corrected, they may result in the death of cells, tissues, organs, and finally, the death of the person.

As a paramedic, you must understand and be able to recognise disturbances in the body's metabolism. When appropriate, you must be able to intervene with treatments aimed at correcting the problem or at sustaining enough function so that the problem can be definitively treated at the hospital. The treatments you initiate may differ from those employed by the Emergency Medical Team(EMT)-Basic and may include advanced procedures,

administration of medications and intravenous fluid therapy.Among the metabolic disturbances your patients may be suffering are: Fluid and electrolyte abnormalities, Acid-base imbalance and Shock.

2.0 OBJECTIVES

At the end of this unit, you should be able to:

- identify the body's major fluid compartments and the proportion of the total body water they contain
- name the abnormal states of hydration, and describe their common causes and effects on the human system
- list the major electrolytes, and discuss the role they play in maintaining a fluid balance within the human body
- define the following terms, and explain the role each process plays in human fluid dynamics: (i) diffusion (ii) osmosis (iii) active transport (iv) facilitated diffusion.

3.0 MAIN CONTENT

3.1 Fluid And Electrolytes

Many medical and traumatic conditions adversely affect the fluid and electrolyte balance of the body. Severe derangements in the body's fluid and electrolyte status can result in death. Certain disease processes, such as diabetic ketoacidosis and heat emergencies, are associated with certain electrolyte abnormalities. Thus, it is prudent for paramedics to have a good understanding of the fluids and electrolytes present in the human body.

Water : Water is the most abundant substance in the human body. In fact, water accounts for approximately 60 percent of the total body weight. The total amount of water in the body at any given time is referred to as the total body water(TBW). I n a person weighing 70 kilograms (154 pounds), the amount of total body water would be approximately 42 liters (11 gallons). Water is usually distributed into various compartments of the body. These compartments are separated by cell membranes. The largest compartment is the intracellular compartment. This compartment contains the intracellular fluid (ICF), which is all of the fluid found inside body cells. Approximately 75 percent of all body water is found within this compartment. The extracellular compartment contains the remaining 25 percent of all body water. It includes all of the fluid found outside the body cells, or extracellular fluid(ECF).

These are two divisions within the extracellular compartment: The first includes the intravascular fluid- the fluid found outside of cells and within the circulatory system. It is essentially the same as the blood plasma. The remaining compartment includes the interstitial fluid- all the fluid found outside of the cell membranes, yet not within the circulatory system.

3.I.I Hydration

Water is the universal solvent. That is, most substances dissolve in water. When they do, various chemical changes take place. For this reason, the water content of the body is crucial to virtually all of the body's biochemical processes. Normally, the total volume of water in the body, as well as the distribution of fluid in the three body compartments, remains relatively constant. This occurs despite wide fluctuations in the amount of water that enters and is excreted from the body on a daily basis. The water coming into the body is referred to as intake. The water excreted from the body is referred to as output. To remain relative homeostasis, or balance, the intake must equal the output, as shown below:

Intake:

Digestive system:

Liquids	1,000 ml,
Food(solids)	1,200 ml
Metabolic sources	300 ml
Total	2,500 ml

Output:

Lungs(water vapor):	400 ml
Kidneys(urine)	1,500 ml
Skin(perspiration)	400 ml
Intestine(faeces)	200 ml
Total	2,500 ml

Several mechanisms work to maintain a relative balance between input and output.As an example, when the fluid volume drops, the pituitory gland at the base of the brain secretes the hormone ADH (Anti-Diuretic Hormone). ADH causes the kidney tubules to reabsorb more water back into the blood and to excrete less urine. This process helps to restore the fluid volume to normal values.Thirst also regulates fluid intake. The sensation of thirst normally occurs when body fluids decrease, stimulating the person to take in more fluids orally. On the other hand, when too many fluids enter the body, the kidneys are activated and more urine is excreted, thus eliminating excess fuid. The body also maintains fluid balance by shifting water from one body space to another.

3.1.2 Dehydration

This is an abnormal decrease in the total body water and can result from several factors. These include:

(a) Gastrointestinal losses result from prolonged vomiting, diarrhoea, or malabsorption disorders.
(b) Increased insensible loss is loss of water through normal mechanisms which are difficult to detect or measure(ie. Perspiration, water vapor from the lungs, saliva). These can be increased in fever states, during hyperventilation, or with high environmental temperatures.
(c) Increased sweating(also called perspiration or diaphoresis) can result in significant fluid loss. This can occur with many medical conditions or in areas of high environmental temperatures.
(d) Internal losses(commonly called third space losses) are losses of fluid into various body fluid compartments. In this situation, fluid is typically lost from the intravascular compartment into the interstitial compartment, effectively taking it out of the circulating volume. This can occur with peritonitis, pancreatitis, or bowel obstruction. It can also occur in poor nutritional states where there is not enough protein in the vascular system to retain water.
(e) Plasma losses occur from burns, surgical drains and fistulas, and open wounds.

Dehydration may involve only the loss of water. However, more commonly, there is also a loss of electrolytes. At the hospital, fluid replacement will be based on both fluid and electrolyte deficits once the patient's electrolyte abnormalities can be determined through laboratory testing.Overhydration can occur as well. The major sign of overhydration is edema. Patients with heart disease may manifest overhydration much earlier than patients without heart disease. In severe cases of overhydration , overt heart failure may be present. Treatment is directed at removing the excessive fluid.

3.1.3 Electrolytes

The various chemical substances present throughout the body can be classified either as electrolytes or non-electrolytes. Electrolytes are substances that dissociate into electrically charged particles when placed into water. The charged particles are referred to as ions. Ions with a positive charge are called cations, while ions with a negative

charge are called anions.An example of this would be the dissociation of the drug sodium bicarbonate when placed into water. Sodium bicarbonate is a neutral salt. When placed into water, it dissociates into two charged particles , as shown below:

$NaHCO_3 = Na^+ + HCO^-_3$

Sodium bicarbonate =Sodium cation + Bicarbonate anion

Neutral salt = cation + anion

Sodium bicarbonate is an example of an electrolyte that is taken into the body as a medication. However, there are many naturally occurring electrolytes present in the body.

The most frequently occurring cations include:

(a) Sodium (Na^+). Sodium is the most prevalent cation in the extracellular fluid. It plays a major role in regulating the distribution of water. In fact, it is often said that water follows sodium. Sodium is also important in the transmission of nervous impulses. An increase in the relative amount of sodium in the body is called hypernatremia, while a decrease is referred as hyponatremia.

(b) Potassium (K^+). Potassium is the most prevalent cation in the intracellular fluid. It is also important in the transmission of electrical impulses. An abnormally level is referred to as hypokalemia, while a high potassium level is referred to as hyperkalemia.

(c) Calcium (Ca^{++}). Calcium has many physiological functions. It plays a major role in muscle contraction as well as nervous impulse transmission. An increased calcium level is called hypercalcemia, while a decreased calcium level is called hypocalcemia.

(d) Magnesium (Mg^{++}). Magnesium is necessary for several biochemical processes that occur in the body and is closely associated with phosphate in many processes.

The most frequently occurring anions include:

(a) Chloride (Cl^-). Chloride is an important anion. Its negative charge balances the positive charge associated with the cations. It also plays a major role in fluid balance and renal (kidney) function. Chloride has a close association with sodium.

(b) Bicarbonate (HCO^-_3). Bicarbonate is the principle buffer of the body. It neutralizes the highly acidic hydrogen ion (H^+) and other organic acids.

(c) Phosphate (HPO^-_4). Phosphate is important in body energy stores. It is closely associated with magnesium in renal function. It also acts as a buffer, primarily in the intracellular space, in much the same manner as bicarbonate.

Many other compounds carr negative charges. Among these are some of the proteins, certain organic acids, and other compounds.

3.1.4 Osmosis And Diffusion

The various fluid compartments, previously discussed, are separated by cell membranes. These membranes are unique, semi-permeable membranes. That is, they allow the passage of certain materials, while restricting the passage of others. Compounds with small molecules, such as water (H_2O), pass readily through the membrane; larger compounds, such as proteins, are restricted. This selective movement of fluids results from the presence of pores(openings) within the membrane. Only compounddddds small enough to pass through the pores can enter or exit the cell. Electrolytes do not pass as readily as water through the membrane. This is not due so much to their size as to their electrical charge. When solutions on opposite sides of a semi-permeable membrane are equal in concentration, the relationship is said to be isotonic. When the concentration of a given solute is greater on one side of the membrane than on theeeee other, it is said to be hypertonic. When the concentration is less on one side of the cell membrane, as compared to the other, it is referred to as hypotonic. This difference in concentration is known as the osmotic gradient.

The natural tendency of the body is to keep the balance of electrolytes and water equal on both sides of the cell membrane. This is an example of homeostasis. If one side of a cell membrane has an increased quantity of a given electrolyte(is hypertonic), there will be a shift of the electrolyte from that side and a shift of water from the other side to restore the balanced state.The tendency of molecules to move from an area of higher concentration to an area of lower concentration is referred to as diffusion and does not require energy. The diffusion of a solute (usually an electrolyte) across a cell membrane from the area of higher concentration to the area of lower concentration continues until the natural balance is again attained. Water also moves across the cell membrane so as to dilute the area of increased electrolyte concentration. The movement of water is more rapid than the movement of electrolytes. This form of diffusion is referred to as osmosis.

3.1.5 Acid-Base Balance

Acid-base balance is a dynamic relationship that reflects the relative concentration of hydrogen ions (H^+) in the body. Hydrogen ions are acidic and the concentration of these within the body must be maintained within fairly strict limits. Any deviation in the hydrogen ion

concentration adversely affects all of the biochemical events that occur in the body. The hydrogen ion concentration is dynamic, changing from second to second.

The pH scale

The total number of hydrogen ions present in the body at any given time is very high. Because of this, the pH system of measurement is utilised. The pH scale is inversely related to hydrogen ion concentration. That is, the greater the hydrogen ion concentration, the lower the pH. The lower the hydrogen ion concentration, the higher the pH..The pH scale is logarithmic, each number represents a value ten times that of its neighboring number, so that pH 6 represents a hydrogen ion concentration 10 times as great as that represented by pH 7.The pH scale ranges from 1 to 14. A pH of 1 means that only hydrogen ions are present. A pH of 14 means that there are virtually no hydrogen ions present.The pH of water is 7.0 , which is neutral pH. The pH of the body is normally 7.35 to 7.45.Because hydrogen ions are acidic , a pH below 7.35 is referred to as acidosis.A substance that produces negatively-charged ions that can neutralize the positively-charged hydrogen ions (or other acids) is called alkali or a base. An excess of alkaline (base) substances or a deficit of acids will produce a pH above 7.45, which is referred to as alkalosis. A variation of only 0.4 of a pH unit in either direction (6.9 or 7.8) can be fatal.

Bodily regulation of acid-base balance

The body is constantly producing hydrogen ions (acids) through metabolism and other biochemical proceses. To maintain the acid-base balance, these hydrogen ions must be constantly eliminated from the body. There are three major mechanisms to remove hydrogen ions from the body. The fastest mechanism is often referred to as the buffer system or the bicarbonate buffer system.

4.0CONCLUSION

In this unit you have learnt about fluid and electrolytes, hydration, dehydration, overhydration, osmosis and difusion as well as acid-base balance. You now can identify the body's major fluid compartments and the proportion of total body water they contain. You can as well name the abnorma states of hydration. You are advised to go through this material for you own sucess.

5.0SUMMARY

This unit has presented fundamental physiology, pathophysiology, and management of fluid and electrolyte disorders, and acid-base balance. All these demand immediate recognition approach.

6.0TUTOR-MARKED ASSIGNMENT

(i) Identify the body's major fluid compartments.
(ii) Name the abnormal states of hydration and describe their common causes and effects on the human system.
(iii)List the major electrolytes, and discuss the role they play in maintaining a fluid balance within the human body.

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MODULE 4

UNIT 1 SHOCKS

CONTENTS

1.0 INTRODUCTION

Shock is an acute medical condition associated with a fall in blood pressure, caused by such events as loss of blood, severe burns, allergic reaction, or sudden emotional stress, and marked by cold, pallid skin, irregular breathing, rapid pulse, and dilated pupils. Having introduced you to this unit, it will help you to acquire the general overview of the whole unit. It is therefore believed that this unit will also help you understand what shock is and its basic components.

2.0 OBJECTIVES

At the end of this unit, you should be able to:

- define the term shock
- state the various types of shock
- mention the clinical features of shock
- state the management of shock.

3.0 MAIN CONTENT

3.1 Acute Conditions

3.1.1 Shock

Circulatory shock, commonly known as shock, is a life-threatening medical condition of low blood perfusion to tissues resulting in cellular injury and inadequate tissue function. The typical signs of shock are low blood pressure, rapid heart rate, and signs of poor end-organ perfusion (i.e. low urine output, confusion, or loss of consciousness). The shock Index (SI), Defined as heart rate, divided by systolic blood pressure, is an accurate diagnostic measure that is more useful than hypotension and tachycardia in isolation. Blood pressure may not be a reliable sign for shock, as there are times when a person is in circulatory shock but has a stable blood pressure. Circulatory shock is not related to the emotional state of shock. Circulatory shock is a life-threatening medical emergency and one of the most common causes of death for critically ill people.

From a medical standpoint, is defined as inadequate tissue perfusion. It can occur for many reasons such as trauma, fluid loss, heart attack, infection, spinal cord injury, and other causes. Although, the causes are different, all forms of shock have the same underlying pathophysiology at the tissue level.

3.1.2 Pathophysiology

All body cells require a constant supply of oxygen and other essential nutrients. At the same time waste products, such as carbon dioxide, must be constantly removed. The circulatory system, in conjunction with the respiratory and gastrointestinal systems, provides the body ‘s cells with these essential nutrients. Inadequate perfusion of body tissues results in shock. Shock occurs first at a cellular level. If allowed to progress, the tissues, organs, and ultimately the entire organism are affected.

Perfusion is dependent on a functioning and intact circulatory system.

The three components of the circulatory system include:

(a) The pump (heart)
(b) The fluid (blood)
(c) The container (blood vessels)

A derangement in any one of these components can adversely affect perfusion.

The pump: The heart is the pump of the cardiovascular system. It receives blood from the venous system, pumps it to the lungs where it is oxygenated, and subsequently pumps it to the peripheral tissues. The amount of blood ejected by the heart in one contraction is referred to as the stroke volume. Several factors affect stroke volume. These include preload, contractile force, and Afterload.

Preload: is the amount of blood delivered to the heart during diastole. It is dependent on venous blood. The venous system is a capacitance, storage, system. That is it can be contracted or expanded as needed to meet the physiological demands of the body. When additional oxygenated blood is required, the venous capacitance is reduced, thus increasing the amount of blood delivered to the heart. The greater the preload, the greater the stroke volume. Preload also affects cardiac contractile force as well as the greater the volume of preload, the more the ventricles are stretched. The greater the stretch, up to a certain point, the greater will be the subsequent cardiac contraction. This is referred to as the Frank Starling Mechanism and can be illustrated through the example of a rubber band. The more the rubber band is stretched, the greater will be its velocity when released. In addition, cardiac contractile strength is affected by circulating Catecholamines (Epinephrine and Norepinephrine) and by sympathetic nervous system tone Catecholamines enhance cardiac contractile strength by action on the beta-adrenergic receptors.

Finally, stroke volume is affected by afterload. The Afterload is the resistance against which the ventricle must contract. This resistance must be overcome before ventricular contraction can result in ejection of blood. Afterload is determined by the degree of peripheral resistance. This in effect, is due to the amount of vasoconstriction present. The arterial system can be expanded and contracted to meet the metabolic demands of the body. The greater the resistance offered by the arterial system, the less the stroke volume. The amount of the blood pumped by the heart in one minute is referred to as the cardiac output. It is a function stroke volume (liters per beat) and heart rate (beats per minute) .Cardiac output is usually expressed in liters per minute . It can be defined by the below equation:

Stroke volume x Heart rate = Cardiac output.

The equation illustrates the factors that can affect cardiac output. An increase in stroke volume or an increase in heart rate can increase cardiac output. Conversely, a decrease in stroke volume or a decrease in heart rate can decrease cardiac output.

The blood pressure is the representative of Cardiac Output

Cardiac output x peripheral vascular resistance = Blood pressure.

Peripheral Vascular Resistance: is the pressure against which the heart must pump. Since the circulatory is a closed system, increasing either cardiac output or peripheral vascular resistance will increase blood pressure. The body strives to keep the blood pressure relatively constant through feed mechanisms. Sensory fibers commonly referred to as baroreceptors are present in the carotid bodies-small structures that contain nerve tissue at the branch of carotid arteries- and the arch of aorta. These baroreceptors centers closely monitor blood pressure.

If the blood pressure increases, the baroreceptors send signals to the brain that cause the blood pressure to return to its normal values. This is accomplished by decreasing the heart rate, decreasing the preload, or decreasing peripheral vascular resistance. If the blood pressure falls, the baroreceptors are stimulated. This results in activation of the sympathetic nervous system. The heart rate is increased, as the strength of the cardiac contractions. In addition, the peripheral blood vessels are also affected. There is arteriolar constriction, venous constriction (which results in decreased container size), and overall increased peripheral vascular resistance. Finally, the adrenal medulla (the inner portion of the adrenal gland) is stimulated. This results in the secretion of epinephrine and norepinephrine, which further enhance the response.

The fluid: Blood is the fluid of the cardiovascular system. Blood is a viscous fluid, that is, it is thicker and more adhesive than water. As a result, it flows more slowly than water. An adequate amount of blood is required for perfusion. The cardiovascular system is a close system, with no major movement of fluid into or out of the system. Because of this, the volume of blood present must be adequate fill the container. Blood which consists of the plasma and the formed elements transports oxygen, carbon dioxide, nutrients, hormones, metabolic waste products and heat.

The container: Blood vessels serve as the container of the cardiovascular system. The blood vessels can be thought of as a continuous, closed, and pressurised pipeline that moves blood throughout the body. Comprised of arteries, arterioles, capillaries, venules, and veins, the blood vessels play a significant role in maintaining blood flow. Although, the heart is considered to be the pump of the circulatory system, the blood vessels-under the control of the autonomic nervous system can regulate blood flow to different areas of the body by adjusting their size as well as by selectively recruiting blood through the large microcirculation.

While the arteries and veins, like the heart, are subject to direct stimulation from the sympathetic portions of the autonomic nervous system, the microcirculation is primarily responsible to local tissue needs. The capability of some vessels in the capillary network to adjust their diameter permits the microcirculation selectively to supply undernourished tissue, while temporarily bypassing tissues with no immediate need. Capillaries have a sphincter at the origin of the capillary, called the pre-capillary sphincter and another at the end of the capillary, called the post-capillary sphincter. The pre-capillary sphincter responds to local tissue demands, such as acidosis, and opens as more arterial blood is needed. The post-capillary sphincter opens when blood is to be emptied into the venous system. Blood flow through the vessels occurs because of two characteristics: Peripheral resistance and pressure within the system. Peripheral resistance, as noted earlier, is the resistance to blood flow. Vessels with large inside diameters create less resistance, while vessels with smaller inside diameters create greater resistance. Peripheral resistance is dependent on three factors –the length of the vessel, the diameter of the vessel, and blood viscosity.

There is very little resistance to blood flow through the aorta and arteries. A significant change in peripheral resistance occurs at the arteriole level. This is because the inside diameter of the arteriole is much smaller, as compared to the aorta and arteries. Additionally, the arteriole has the pronounced ability to change its diameter as much as fivefold. It tends to do this in response to local tissue needs and autonomic nervous signals. Contraction of the venous side of the vascular system results in decreased capacitance and increased cardiac preload. The arterial system, on the other hand, provides systemic vascular resistance. An increase in arterial tone increases resistance, which increases blood pressure.

Tissue perfusion: Tissue perfusion is dependent upon each component of the circulatory system. In addition, the terms of tissue oxygenation, it is dependent upon the respiratory system. If tissue perfusion is compromised, several conditions begin to develop –conditions that ultimately can lead to shock.

3.1.3 Physiological Responses to Shock/Signs and Symptoms

Shock results from many factors. However, regardless of the cause, the underlying problem remains inadequate tissue perfusion. When tissue perfusion declines, the body reacts immediately to restore and maintain blood flow to all body tissues. It accomplishes this through the activation of various compensatory mechanisms. These physiological

mechanisms are often effective in restoring tissue perfusion. However, if the cause is not corrected, the compensatory mechanism will eventually fail. In such cases, shock becomes irreversible, and organism dies. Treatment of shock is dependent upon recognising its presence early. The initial signs and symptoms of shock is subtle. However, to prevent irreversible shock, you will need to detect these subtle signs and symptoms. In order to understand the signs and symptoms of shock, it is essential that you recognise the body's physiological response to inadequate tissue perfusion.

Systemic response to shock

The body's response to shock is complex, involving nearly everybody system. As tissue perfusion declines, several compensatory mechanisms are activated as the body strives to maintain adequate tissue perfusion. Perfusion (The process of a body delivering blood to a capillary bed in its biological tissue). The initial physiological response to shock is progressive vasoconstriction. This causes an increase in peripheral vascular resistance and serves to maintain to maintain blood pressure. Initially, the blood vessels in the skin, digestive organs, and skeletal muscles constrict in order to maintain blood flow to essential organs such as the kidneys, the heart, and brain. In addition, cardiac output increases by increasing either the heart rate, or the stroke volume, or both. Both peripheral vasoconstriction and increased cardiac output aid in restoring tissue perfusion.

Clinically, these early physiological responses may be difficult to detect. One of the earliest detectible changes is an increase in heart rate. As the blood vessel of the skin constrict, the skin losses its color and becomes pale. In addition, it becomes cool to touch, as vasoconstriction continues, capillary refill time will become prolonged. Other body systems also respond to decreased tissue perfusion. The respiratory system reacts by increasing the respiratory rate and tidal volume. This serves to provide the body with increased supplies of oxygen during this period of stress. The urinary system decreases filtration of water in order to maintain intravascular fluid volume. The gastrointestinal system slows because of decreased blood supply. All of these help the cardiovascular system maintain peripheral tissue perfusion. However, when such compensatory mechanisms fail, inadequate tissue perfusion worsens.

3.1.4 Types of Shock

Although, all types of shock result in inadequate tissue perfusion, the causes are different. Medical experts commonly classify shock based on the cause. The frequent types (causes) of shock are discussed below:

Hypovolemic shock
This is shock due to a loss of intravascular fluid volume and is referred as hypovolemic shock. Possible causes of hypovolemic shock include:

(a) Internal or external hemorrhage.
(b) Traumatic injury.
(c) Long bone or open fractures.
(d) Severe dehydration from vomiting or diarrhea.
(e) Plasma loss from burns.
(f) Diabetic ketoacidosis with resultant osmotic diuresis.
(g) Excessive sweating.

Hypovolemic shock can also be due to internal third-space loss. Such a condition can occur with bowel obstruction, peritonitis, pancreatitis, or; liver failure resulting in ascites (accumulation of fluid within the abdominal cavity).

Cardiogenic shock
An inability of the heart to pump enough blood to supply all body parts is referred to as cardiogenic shock. Cardiogenic shock is usually the result of severe left ventricular failure, secondary to acute myocardial infarction or congestive heart failure. The hypotension that accompanies this form of shock aggravates the situation by decreasing coronary perfusion. With decreased coronary perfusion, the heart muscle becomes even more damaged, thus, establishing a vicious cycle that ultimately results in complete pump failure. During cardiogenic shock, the activation of compensatory mechanisms can worsen the situation. When the peripheral resistance increases to maintain the blood pressure the myocardial workload increases. This, in turn, increases the myocardial oxygen demand, further aggravating myocardial ischemia and infarction. Cardiac output is further depressed.

While the most common cause of cardiogenic shock is severe left ventricular failure, several other factors can have the same clinical manifestation. These include chronic progressive heart disease, such as cardiomyopathy, rupture of papillary heart muscles, or interventricular septum, and end-stage valvular disease (mitral stenosis or aortic regurgitation). Most patients who experience cardiogenic shock will have normal blood volume. However, some patients will be hypovolemic from an excessive use of prescribed diuretics or the severe diaphoresis that accompanies some acute cardiac events. Patients may also experience relative hypovolemia (neurogenic shock) from the vasodilatory (blood vessel dilation) effects of drugs such as nitroglycerin.

Neurogenic shock; is an inadequate peripheral resistance. This may be described as widespread vasodilation. With this inappropriate vasodilation, a disproportionate amount of blood collects in the capillary bed. This reduces venous return, cardiac output, and arterial blood pressure. Neurogenic shock is most commonly due to an injury that results in severe spinal cord injury or total transaction of the cord. Other causes of neurogenic shock include; central nervous system injury, septicaemia from bacterial infection, anaphylactic reaction, insulin overdose, and addisonian crisis (a disorder of the adrenal glands) . With neurogenic shock there is an absence of the, sympathetic response.

3.1.5 Evaluation of the Shock Victim

Shock will present itself in a variety of ways. Depending on the degree of compensation, some patients will display few signs and symptoms, while in other indications of shock will be much more obvious. Your initial approach can often yield a great deal of information. Before reaching the patient's side, you can often observe mental status, respiratory effort, and skin color. In situations where the patient is obviously in shock, you must be aggressive with your assessment and treatment.

Primary assessment
As with any patient care situation, assessment should begin the, ABCs:

Airway: First, check the airway. The conscious patient who is able to speak usually has a patent (open) airway, whereas the unconscious patient is often subject to airway obstruction. In unconscious states, the tongue tends to fall back against posterior oropharynx. Also, in trauma related shock, bleeding in the oropharynx is often present, which can further compromise the airway.

Breathing: Once an open airway has been ensured, the adequate of the air exchange should be checked. While compensatory hyperventilation tends to occur in early shock, the unconscious shock patient will often hyperventilate. Hypoventilation occurs when the respiratory center of the brain becomes depressed due to hypoperfusion. When evaluating ventilator status, be sure to check both the rate and depth of respiration s. Watch for patients who exhibit rapid, shallow breathing. This type of breathing is just as ineffective as slow or irregular respirations. Rapid, shallow breathing results in several reduced minute volumes since an inadequate amount of air is being exchanged.

Circulation: After you have assured patency of the airway and ventilator effectiveness, turn your attention to evaluating circulation. First, evaluate the pulse for rate and character. Then examine the patient

for obvious external bleeding. Because of compensatory mechanisms, a normal pulse rate may be seen even in the presence of a 10 to 15 percent volume deficit. A fast weak or thread pulse suggests decreased circulatory volume.

The location of the palpable pulse can also be an important indicator of circulatory status. The presence of a radial pulse indicates that the patient has a systolic blood pressure of at least 80 mmHg. If the radial pulse cannot be palpated, the femoral pulse or carotid pulse should be checked next. A palpable femoral pulse indicates a systolic blood pressure of 70 mmHg, while a palpable carotid pulse indicates a systolic blood pressure of 60 mmHg. Although, these are rough estimates, they can be quite helpful. In the case of profound shock where severe vasoconstriction is present, you might not be able to palpate a pulse at all. Based on your findings of reduced perfusion, it would be appropriate to take immediate measures to restore circulation. In cases of obvious hypotension, as determined by using the, above, rule for checking the location of a palpable pulse , you do not need to take the time to auscultate a blood pressure before initiating immediate shock treatment.

The color, appearance, and temperature of the skin can also serve as useful indicator of circulatory effectiveness, the skin may appear normal in the initial stages of shock. However, compensatory mechanisms soon change the skin's appearance. As vasoconstriction is activated and the blood is routed to the central circulation, the skin becomes pale (decreased perfusion), cyanotic (stagnant pooling of blood with inadequate oxygenation), mottled (combination of both, a late sign of shock), as well as cool to the touch and diaphoretic. Often, the appearance of the skin will indicate shock even before there are any noticeable changes in the blood pressure. Another method of assessing the circulation is to check the capillary refill.

Capillary refill testing is performed by applying pressure to the nail bed of one of the patient's fingers. This pressure should cause a blanching of the nail bed. When the pressure is released, the nail should return to its normal pink color within two seconds. You can approximate this time period by saying, Capillary refill. If the normal pink color does not return to the nail bed, it can be assumed that there is decrease d perfusion to this area, since the nail bed is the most distal part of the circulation, poor capillary refill is an early indicator of decreased perfusion to the whole body. However, use of the capillary refill test has limited value in the field setting due to poor lighting conditions and other environmental factors.

Disability: The level of consciousness should also be assessed throughout the primary survey. In fact, the level of consciousness is

probably a better indicator of decreased tissue perfusion than most other signs, because of the high energy requirements of the brain, any reduction in cerebral blood flow will be manifested by the following conditions:

(i) Agitation
(ii) Disorientation
(iii) Confusion
(iv) An inability to respond to questions or commands appropriately
(v) Unresponsiveness

Any significant alteration in the level of consciousness must be viewed as an indication of critical hypoperfusion or hypoxia. Additionally, with an increased secretion of catecholamines, the patient often becomes anxious or apprehensive.

Secondary assessment

After completion of the primary survey and initiation of the necessary treatment, a secondary survey should be performed. The thoroughness of the secondary survey is dependent upon the severity of the patient's condition. Obvious life-threatening problems cannot be corrected in the prehospital setting warrant rapid transportation of the patient to an appropriate definitive care facility. Ideally, when assessing the seriously injured patient, you should expose and inspect the head, neck, chest, and abdomen. Throughout the secondary survey, and while providing the treatment and transporting the patient to the hospital, you must continually reassess the temperature and moisture of the skin, blood pressure, pulse rate, and respiratory rate.

In shock- due to the presence of hypoxia, acidosis, and increased secretion of catecholamines- cardiac dysrhythmias must be considered a potential complication. Because of this, you should include the continual monitoring of the patient's ECG rhythm in your assessment. Look for rapid or slow heart rates as well as irregular rhythms. These conditions indicate potential life-threatening problems. Additional information can be obtained by asking your patient the appropriate questions. Find out how he or she feels. Is the patient thirsty, weak, nauseous, or dizzy? Does he or she have a history of significant medical conditions or take any medications? Such questions will give you additional information upon which to base your treatment. Evaluation of the trauma victim for shock begins in the primary survey, where the most obvious signs of decreased tissue perfusion may be present. It is continued during the secondary survey, where more subtle clues may be found. The patient is then continually assessed for signs of developing shock until he or she is placed in the hands of emergency department personnel.

3.1.6 Management

The best evidence exists for the treatment of septic shock in adults and as the pathophysiology appears similar in children and other types of shock treatment this has been extrapolated to these areas. Management may include securing the airway via intubation if necessary to decrease the work of breathing and for guarding against respiratory arrest. Oxygen supplementation, intravenous fluids, passive leg raising (not Trendelenburg position) should be started and blood transfusions added if blood loss is severe. It is important to keep the person warm as well as adequately manage pain and anxiety as these can increase oxygen consumption.

Fluids
Aggressive intravenous fluids are recommended in most types of shock (e.g. 1-2 liter normal saline bolus over 10 minutes or 20ml/kg in a child) which is usually instituted as the person is being further evaluated. Which intravenous fluid is superior, colloids or crystalloids, remains undetermined. Thus, as crystalloids are less expensive they are recommended. If the person remains in shock after initial resuscitation packed red blood cells should be administered to keep the hemoglobin greater than 100 gms/l. For those with hemorrhagic shock the current evidence supports limiting the use of fluids for penetrating thorax and abdominal injuries allowing mild hypotension to persist (known as permissive hypotension). Targets include a mean arterial pressure of 60 mmHg, a systolic blood pressure of 70-90 mmHg, or until their adequate mentation and peripheral pulses.

Medications
Vasopressors may be used if blood pressure does not improve with fluids. There is no evidence of superiority of one vasopressor over another. Vasopressors have not been found to improve outcomes when used for hemorrhagic shock from trauma but may be of use in neurogenic shock. Activated protein C (Xigris) while once aggressively promoted for the management of septic shock has been found not to improve survival and is associated with a number of complications. The use of sodium bicarbonate is controversial as it has not been shown to improve outcomes. If used at all it should only be considered if the pH is less than 7.0.

Treatment goals
The goal of treatment is to achieve a urine output of greater than o.5ml/Kg/h, a central venous pressure of 8-12mmHg and a mean arterial pressure of 65-95mmHg. In trauma the goal is to stop the bleeding which in many cases requires surgical interventions.

4.0 CONCLUSION

This unit has focused on the common emergency conditions very vital to health which is shock, the various types and their management. Subsequent unit will also discuss other emergency conditions e.g. Hemorrhage , fractures, etc.

In this unit, you have learned about shock, which occurs as a result the metabolic need of cells not being met due to inadequate blood flow. This unit has also classified the types of shock into three major groups, their clinical features and management process.

5.0 SUMMARY

6.0 TUTOR-MARKED ASSIGNMENT

1. Using your own words, define the term shock.
2. a. Mention the three major types of shock.
   b. Discuss the physiological responses to shock.
3 In your own words discuss the physiology of perfusion.
4 Write short notes on airway, breathing and circulation.

7.0 REFERENCES/FURTHER READING

Basic Life Support: Resuscitation Council, UK. www.resus.org.uk/ pages/bls.htm.

Standard First Aid Course. www.unh.org/standardfirstaid/toc.html.

Silver, Adam (2005). *Shock : A common Pathway For Life-threatening Pediatric Illnesses And injuries Pediatric Emergency Medicine Practice 2.*

Comprehensive Guide to Pre-hospital Skills; Emergency Training.

UNIT 2 CARDIAC ATTACK/ARREST

CONTENTS

1.0 INTRODUCTION

This unit will help you acquire the basic understanding of what cardiac attack is and its main components under the objectives below.

2.0 OBJECTIVES

At the end of this unit, you should be able to:

- define heart attack and its components
- discuss stroke
- explain chronic heart failure
- state the management of stroke and heart attack.

3.0 MAIN CONTENT

3.1 Cardiac Arrest

3.2 Cardiac (Heart) Attack

3.1 Cardiac Arrest

Cardiac arrest, also known as Cardiopulmonary arrest or Circulatory arrest, is a sudden stop in effective blood circulation due to the failure of the heart to contract effectively or at all. A cardiac arrest is different from (but may be caused by) a myocardial infarction (aka heart attack), where blood flow to the muscle of the heart is impaired. It is different from congestive heart failure, where circulation is substandard, but the heart is still pumping sufficient blood to sustain life.

Arrested blood circulation prevents delivery of oxygen and glucose to the body. Lack of oxygen and glucose to the brain causes loss of consciousness, which then results in abnormal or absent breathing. Brain injury is likely to happen if cardiac arrest goes untreated for more than five minutes. For the best chance of survival and neurological recovery immediate treatment is important. Cardiac arrest is a medical emergency that, in certain situations, is potentially reversible if treated early. Unexpected cardiac arrest can lead to death within minutes: this is called sudden cardiac death (SCD). The treatment for cardiac arrest is immediate defibrillation if a "shockable" rhythm is present, while cardiopulmonary resuscitation (CPR) is used to provide circulatory support and/or to induce a "shockable" rhythm.

3.1.1 Classification

Clinicians classify cardiac arrest into "shockable" versus "non–shockable", as determined by the ECG rhythm. This refers to whether a particular class of cardiac dysrhythmia is treatable using defibrillation. The two "shockable" rhythms are ventricular fibrillation and pulseless ventricular tachycardia while the two "non–shockable" rhythms are asystole and pulseless electrical activity.

3.1.2 Signs and Symptoms

Cardiac arrest is sometimes preceded by certain symptoms such as fainting, fatigue, blackouts, dizziness, chest pain, shortness of breath, weakness, and vomiting. The arrest may also occur with no warning.
When the arrest occurs, the most obvious sign of its occurrence will be the lack of a palpable pulse in the person experiencing it (since the heart

has ceased to contract, the usual indications of its contraction such a pulse will no longer be detectable). Certain types of prompt intervention can often reverse a cardiac arrest, but without such intervention the event will almost always lead to death. In certain cases, it is an expected outcome of a serious illness where death is expected.

Also, as a result of inadequate cerebral perfusion, the patient will quickly become unconscious and will have stopped breathing. The main diagnostic criterion to diagnose a cardiac arrest (as opposed to respiratory arrest which shares many of the same features) is lack of circulation; however, there are a number of ways of determining this. Near-death experiences are reported by 10-20% of people who survived cardiac arrest.

3.1.3 Causes

Coronary heart disease is the leading cause of sudden cardiac arrest. Many other cardiac and non-cardiac conditions also increase one's risk.

Coronary artery disease
Approximately 60–70% of SCD is related to coronary heart disease. Among adults, ischemic heart disease is the predominant cause of arrest with 30% of people at autopsy showing signs of recent myocardial infarction.

Non-ischemic heart disease
A number of other cardiac abnormalities can increase the risk of SCD including cardiomyopathy, cardiac rhythm disturbances, hypertensive heart disease, and congestive heart failure. In a group of military recruits aged 18–35, cardiac anomalies accounted for 51% of cases of SCD, while in 35% of cases the cause remained unknown. Underlying pathology included coronary artery abnormalities (61%), myocarditis (20%), and hypertrophic cardiomyopathy (13%). Congestive heart failure increases the risk of SCD fivefold.

Many additional conduction abnormalities exist that place one at higher risk for cardiac arrest. For instance, long QT syndrome, a condition often mentioned in young people's deaths, occurs in one of every 5000 to 7000 newborns and is estimated to be responsible for 3000 deaths each year compared to the approximately 300,000 cardiac arrests seen by emergency services. These conditions are a fraction of the overall deaths related to cardiac arrest but represent conditions which may be detected prior to arrest and may be treatable.

Non-cardiac
About 35% of SCDs are not caused by a heart condition. The most common non-cardiac causes are trauma, bleeding (such as gastrointestinal bleeding, aortic rupture, or intracranial hemorrhage), overdose, drowning and pulmonary embolism. Cardiac arrest can also be caused by poisoning (for example, by the stings of certain jellyfish).

Risk factors
The risk factors for SCD are similar to those of coronary heart disease and include smoking, lack of physical exercise, obesity, and diabetes, as well as family history.

3.1.4 Diagnosis

Cardiac arrest is synonymous with clinical death. A cardiac arrest is usually diagnosed clinically by the absence of a pulse. In many cases lack of carotid pulse is the gold standard for diagnosing cardiac arrest, but lack of a pulse (particularly in the peripheral pulses) may result from other conditions (e.g. shock), or simply an error on the part of the rescuer. Studies have shown that rescuers often make a mistake when checking the carotid pulse in an emergency, whether they are healthcare professionals or lay persons.

Owing to the inaccuracy in this method of diagnosis, some bodies such as the European Resuscitation Council (ERC) have de-emphasised its importance. The Resuscitation Council (UK), in line with the ERC's recommendations and those of the American Heart Association, have suggested that the technique should be used only by healthcare professionals with specific training and expertise, and even then that it should be viewed in conjunction with other indicators such as agonal respiration.

Various other methods for detecting circulation have been proposed. Guidelines following the 2000 International Liaison Committee on Resuscitation (ILCOR) recommendations were for rescuers to look for "signs of circulation", but not specifically the pulse. These signs included coughing, gasping, colour, twitching and movement. However, in face of evidence that these guidelines were ineffective, the current recommendation of ILCOR is that cardiac arrest should be diagnosed in all casualties who are unconscious and not breathing normally.

3.1.5 Prevention

With positive outcomes following cardiac arrest unlikely, an effort has been spent in finding effective strategies to prevent cardiac arrest. With the prime causes of cardiac arrest being ischemic heart disease, efforts to

promote a healthy diet, exercise, and smoking cessation are important. For people at risk of heart disease, measures such as blood pressure control, cholesterol lowering, and other medico-therapeutic interventions are used.

Code teams

In medical parlance, cardiac arrest is referred to as a "code" or a "crash". This typically refers to "code blue" on the hospital emergency codes. A dramatic drop in vital sign measurements is referred to as "coding" or "crashing", though coding is usually used when it results in cardiac arrest, while crashing might not. Treatment for cardiac arrest is sometimes referred to as "calling a code".

Extensive research has shown that patients in general wards often deteriorate for several hours or an even day before a cardiac arrest occurs. This has been attributed to a lack of knowledge and skill amongst ward-based staff, in particular a failure to carry out measurement of the respiratory rate, which is often the major predictor of a deterioration and can often change up to 48 hours prior to a cardiac arrest. In response to this, many hospitals now have increased training for ward-based staff. A number of "early warning" systems also exist which aim to quantify the risk which patients are at of deterioration based on their vital signs and thus provide a guide to staff. In addition, specialist staff are being utilised more effectively to augment the work already being done at ward level. These include:

- Crash teams (or code teams) - These are designated staff members with particular expertise in resuscitation who are called to the scene of all arrests within the hospital. This usually involves a specialised cart of equipment (including defibrillator) and drugs called a "crash cart" or "crash trolley".
- Medical emergency teams - These teams respond to all emergencies, with the aim of treating the patient in the acute phase of their illness in order to prevent a cardiac arrest.
- Critical care outreach - As well as providing the services of the other two types of team, these teams are also responsible for educating non-specialist staff. In addition, they help to facilitate transfers between intensive care/high dependency units and the general hospital wards. This is particularly important, as many studies have shown that a significant percentage of patients discharged from critical care environments quickly deteriorate and are re-admitted; the outreach team offers support to ward staff to prevent this from happening.

In some medical facilities, the resuscitation team may purposely respond slowly to a patient in cardiac arrest, a practice known as "slow code", or

may fake the response altogether for the sake of the patient's family, a practice known as "show code". This is generally done for patients for whom performing CPR will have no medical benefit. Such practices are ethically controversial and are banned in some jurisdictions.

Implantable cardioverter defibrillators

A technologically based intervention to prevent further cardiac arrest episodes is the use of an implantable cardioverter-defibrillator (ICD). This device is implanted in the patient and acts as an instant defibrillator in the event of arrhythmia. Note that standalone ICDs do not have any pacemaker functions, but they can be combined with a pacemaker, and modern versions also have advanced features such as anti-tachycardic pacing as well as synchronized cardioversion. A recent study by Birnie et al. at the University of Ottawa Heart Institute has demonstrated that ICDs are underused in both the United States and Canada. An accompanying editorial by Simpson explores some of the economic, geographic, social and political reasons for this. Patients who are most likely to benefit from the placement of an ICD are those with severe ischemic cardiomyopathy (with systolic ejection fractions less than 30%).

3.1.6 Management

Sudden cardiac arrest may be treated via attempts at resuscitation. This is usually carried out based upon basic life support (BLS)/advanced cardiac life support (ACLS), pediatric advanced life support (PALS) or neonatal resuscitation program (NRP) guidelines.

Cardiopulmonary resuscitation

Cardiopulmonary resuscitation (CPR) is an important part of the management of cardiac arrest. It is recommended that it be started as soon as possible and interrupted as little as possible. The component of CPR which seems to make the greatest difference in most cases is the chest compressions. Correctly performed bystander CPR has been shown to increase survival; however, it is performed in less than 30% of out of hospital arrests as of 2007. If high-quality CPR has not resulted in return of spontaneous circulation and the person's heart rhythm is in asystole, discontinuing CPR and pronouncing the person's death is reasonable after 20 minutes. Exceptions to this include those with hypothermia or who have drowned. Longer durations of CPR may be reasonable in those who have cardiac arrest while in hospital.

Tracheal intubation has not been found to improve survival rates in cardiac arrest and in the prehospital environment may worsen it. A 2009 study found that assisted ventilation may worsen outcomes over placement of an oral airway with passive oxygen delivery.

CPR which involves only chest compressions results in the same outcomes as standard CPR for those who have gone into cardiac arrest due to heart issues. A 2013 review found some evidence that mechanical chest compressions (as performed by a machine) are better than manual chest compressions while a 2011 and 2012 review considered the evidence insufficient. It is unclear if a few minutes of CPR before defibrillation results in different outcomes than immediate defibrillation.

Defibrillation

Shockable and non–shockable causes of cardiac arrest are based on the presence or absence of ventricular fibrillation or pulseless ventricular tachycardia. The shockable rhythms are treated with CPR and defibrillation. In addition, there is increasing use of public access defibrillation. This involves placing automated external defibrillators in public places, and training staff in these areas how to use them. This allows defibrillation to take place prior to the arrival of emergency services and has been shown to lead to increased chances of survival. Some defibrillators even provide feedback on the quality of CPR compressions, encouraging the lay rescuer to press the patient's chest hard enough to circulate blood. In addition, it has been shown that those who have arrests in remote locations have worse outcomes following cardiac arrest.

Medications

Medications, while included in guidelines, have been shown not to improve survival to hospital discharge following out-of-hospital cardiac arrest. This includes the use of epinephrine, atropine, and amiodarone. Vasopressin overall does not improve or worsen outcomes but may be of benefit in those with asystole especially if used early. Epinephrine does appear to improve short-term outcomes such as return of spontaneous circulation some of the lack of long-term benefit may be related to delays in epinephrine use.

The 2010 guidelines from the American Heart Association no longer contain the association's previous recommendation for using atropine in pulseless electrical activity and asystole due to the lack of evidence for its use. Evidence is insufficient for lidocaine, and amiodarone may be considered in those who continue in ventricular tachycardia or ventricular fibrillation despite defibrillation. Thrombolytics when used generally may cause harm but may be of benefit in those with a pulmonary embolism as the cause of arrest.

Targeted temperature management
Cooling a person after cardiac arrest who has a return of spontaneous circulation (ROSC) but no return of consciousness improves outcomes. This procedure is called targeted temperature management (previously known as therapeutic hypothermia). People are typically cooled for a 24-hour period, with a target temperature of 32–34 °C (90–93 °F). Death rates in the hypothermia group are 35% lower than in those with no temperature management. Complications are generally no greater in those who receive this therapy.

A November 2013 trial found that actively cooling to a temperature of 36 °C (97 °F) results in the same outcomes as 33 °C (91 °F). This may be because preventing fever, rather than the hypothermia itself, is more important. Other possible reasons could be the long -time of >8 hours needed to cool in the 33 °C group and the very high rate of bystander of CPR compared to usual international rates. Earlier versus later cooling may result in better outcomes. A trial that cooled in the ambulance, however, found no difference compared to starting cooling in-hospital. A registry database found poor neurological outcome increased by 8% with each five-minute delay in initiating TH and by 17% for every 30-minute delay in time to target temperature.

Do not resuscitate
Some people choose to avoid aggressive measures at the end of life. A do not resuscitate order (DNR) in the form of an advance health care directive makes it clear that in the event of cardiac arrest, the person does not wish to receive cardiopulmonary resuscitation.

3.2 Cardiac (Heart) Attack

A heart attack is the death of, or damage to, part of the heart muscle because the supply of blood to the heart muscle is severely reduced or stopped or an acute attack known as coronary thrombosis which is a blockage of the flow of blood to the heart, caused by a clot in a coronary artery. Though the attack is sudden, it is the result of slowly developing hardening process of the coronary arteries which is known as Artheriosclerosis. Artheriosclerosis is the cause of most heart attacks and the accompanying chest pain known as angina pectoris. The arterial wall becomes rough and narrow due to fatty deposits which harden into patches along the inner lining of the arteries. A blood clot may be formed as a result of the narrowing and block channels, thus leading to less flow of blood to the heart. This results to heart attack otherwise called coronary thrombosis or coronary occlusion.

Most heart attacks are the end result of years of silent but progressive coronary artery disease, which can be prevented in many people. A heart

attack often is the first symptom of coronary artery disease. According to the American Heart Association, 63% of women and 48% of men who died suddenly of coronary artery disease had no previous symptoms. Heart attacks also are called myocardial infarctions (MIs).
A heart attack occurs when one or more of the coronary arteries that supply blood to the heart are completely blocked and blood to the heart muscle is cut off. The blockage usually is caused by atherosclerosis, the build-up of plaque in the artery walls, and/or by a blood clot in a coronary artery. Sometimes, a healthy or atherosclerotic coronary artery has a spasm and the blood flow to part of the heart decreases or stops. Why this happens is unclear, but it can result in a heart attack.

About half of all heart attack victims wait at least two hours before seeking help. This increases their chance of sudden death or being disabled. The longer the artery remains blocked during a heart attack, the more damage will be done to the heart. If the blood supply is cut off severely or for a long time, muscle cells suffer irreversible injury and die. This can cause the patient to die. That is why it is important to recognize the signs of a heart attack and seek prompt medical attention at the nearest hospital with 24-hour emergency cardiac care. About one-fifth of all heart attacks are silent, that is, the victim does not know one has occurred. Although the victim feels no pain, silent heart attacks still can damage the heart. The outcome of a heart attack also depends on where the blockage is, whether the heart rhythm is disturbed, and whether another coronary artery supply blood to that part of the heart. Blockages in the left coronary artery usually are more serious than in the right coronary artery. Blockages that cause an arrhythmia, an irregular heartbeat, can cause sudden death.

3.2.1 Causes and Symptoms

Heart attacks generally are caused by severe coronary artery disease. Most heart attacks are caused by blood clots that form on atherosclerotic plaque. This blocks a coronary artery from supplying oxygen-rich blood to part of the heart. Several major and contributing risk factors increase the risk of developing coronary artery disease. Some of these can be changed and some cannot. People with more risk factors are more likely to develop coronary artery disease.

Major risk factors
Major risk factors significantly increase the risk of coronary artery disease. Those which cannot be changed are:

- Heredity. People whose parents have coronary artery disease are more likely to develop it. African Americans also are at increased risk, due to their higher rate of severe hypertension than whites.

- Sex. Men under the age of 60 years of age are more likely to have heart attacks than women of the same age.
- Age. Men over the age of 45 and women over the age of 55 are considered at risk. Older people (those over 65) are more likely to die of a heart attack. Older women are twice as likely to die within a few weeks of a heart attack as a man. This may be because of other co-existing medical problems.

Major risk factors that can be changed are:

- Smoking. Smoking greatly increases both the chance of developing coronary artery disease and the change of dying from it. Smokers have two to four times the risk of non-smokers of sudden cardiac death and are more than twice as likely to have a heart attack. They also are more likely to die within an hour of a heart attack. Second-hand smoke also may increase risk.
- High cholesterol. Cholesterol is a soft, waxy substance that is produced by the body, as well as obtained from eating foods such as meat, eggs, and other animal products. Cholesterol level is affected by age, sex, heredity, and diet. Risk of developing coronary artery disease increases as blood cholesterol levels increase. When combined with other factors, the risk is even greater. Total cholesterol of 240 mg/dL and over poses a high risk, and 200-239 mg/dL a borderline high risk. In LDL cholesterol, high risk starts at 130-159 mg/dL, depending on other risk factors. HDL (healthy cholesterol) can lower or raise the coronary risk also.
- High blood pressure. High blood pressure makes the heart work harder, and over time, weakens it. It increases the risk of heart attack, stroke, kidney failure, and congestive heart failure. A blood pressure of 140 over 90 or above is considered high. As the numbers increase, high blood pressure goes from Stage 1 (mild) to Stage 4 (very severe). When combined with obesity, smoking, high cholesterol, or diabetes, the risk of heart attack or stroke increases several times.
- Lack of physical activity. This increases the risk of coronary artery disease. Even modest physical activity is beneficial if done regularly.
- Use of certain drugs or supplements. Extreme caution is advised in the use of the herbal supplement ephedra. The supplement, which was marketed for weight loss and to improve athletic performance, was found to contribute to heart attack, seizure, stoke and death.
- In April 2003, the U.S. Food and Drug Administration (FDA) investigating controlling or banning the substance.

- While it was once believed that hormone replacement therapy (HRT) helped prevent heart disease in women, a large clinical trial called the Women's Health Initiative found the opposite to be true. In 2003, the FDA began requiring manufacturers of HRT to place warnings on the box listing adverse effects of estrogen, including increased risk of heart attack, stroke and blood clots. The labels also must mention that HRT should not be used as a preventive medicine for heart disease.

Contributing risk factors

Contributing risk factors have been linked to coronary artery disease, but their significance or prevalence cannot always be demonstrated. Contributing risk factors are:

- Diabetes mellitus. The risk of developing coronary artery disease is seriously increased for diabetics. More than 80% of diabetics die of some type of heart or blood vessel disease.
- Obesity. Excess weight increases the strain on the heart and increases the risk of developing coronary artery disease, even if no other risk factors are present. Obesity increases both blood pressure and blood cholesterol and can lead to diabetes.
- Stress and anger. Some scientists believe that stress and anger can contribute to the development of coronary artery disease. Stress, the mental and physical reaction to life's irritations and challenges, increases the heart rate and blood pressure, and can injure the lining of the arteries. Evidence shows that anger increases the risk of dying from heart disease and more than doubles the risk of having a heart attack right after an episode of anger.
- Rheumatoid arthritis in women. A report released in 2003 noted that women with rheumatoid arthritis have a higher risk of heart attach than those without the condition. The reason is most likely the inflammation arthritis causes.

More than 60% of heart attack victims experience symptoms before the heart attack occurs. These sometimes occur days or weeks before the heart attack. Sometimes, people do not recognise the symptoms of a heart attack or are in denial that they are having one. Symptoms are:

- Uncomfortable pressure, fullness, squeezing, or pain in the center of the chest. This lasts more than a few minutes or may go away and return.
- Pain that spreads to the shoulders, neck, or arms.
- Chest discomfort accompanied by lightheadedness, fainting, sweating, nausea, or shortness of breath.

All these symptoms do not occur with every heart attack. Sometimes, symptoms disappear and then reappear. A person with any of these symptoms should immediately call an emergency rescue service or be driven to the nearest hospital with a 24-hour cardiac care unit, whichever is quicker.

3.2.2 Treatment/Management

Heart attacks are treated with cardiopulmonary resuscitation (CPR) when necessary to start and keep the patient breathing and his heart beating. Additional treatment can include close monitoring, electric shock, drug therapy, re-vascularization procedures, percutaneous transluminal coronary angioplasty and coronary artery bypass surgery.
Upon arrival at the hospital, the patient is closely monitored. An electrical-shock device, a defibrillator, may be used to restore a normal rhythm if the heartbeat is fluttering uncontrollably. Oxygen often is used to ease the heart's workload or to help victims of severe heart attack breathe easier. If oxygen is used within hours of the heart attack, it may help limit damage to the heart.

Drugs to stabilise the patient and limit damage to the heart include thrombolytics, aspirin, anticoagulants, painkillers and tranquilizers, beta-blockers, ace-inhibitors, nitrates, rhythm-stabilizing drugs, and diuretics. Drugs that limit damage to the heart work only if given within a few hours of the heart attack. Thrombolytic drugs that break up blood clots and enable oxygen-rich blood to flow through the blocked artery increase the patient's chance of survival if given as soon as possible after the heart attack. Thrombolytics given within a few hours after a heart attack are the most effective. Injected intravenously, these include anisoylated plasminogen streptokinase activator complex (APSAC) or anistreplase (Eminase), recombinant tissue-type plasminogen activator (r-tPA, Retevase, or Activase), and streptokinase (Streptase, Kabikinase).

To prevent additional heart attacks, aspirin and an anticoagulant drug often follow the thrombolytic drug. These prevent new blood clots from forming and existing blood clots from growing. Anticoagulant drugs help prevent the blood from clotting. The most common anticoagulants are heparin and warfarin. Heparin is given intravenously while the patient is in the hospital. Warfarin, taken orally, often is given later. Aspirin helps to prevent the dissolved blood clots from reforming. To relieve pain, a nitroglycerine tablet taken under the tongue may be given. If the pain continues, morphine sulfate may be prescribed. Tranquilizers such as diazepam (Valium) and alprazolam (Ativan) may be prescribed to lessen the trauma of a heart attack.

To slow down the heart rate and give the heart a chance to heal, beta-blockers often are given intravenously right after the heart attack. These can also help prevent sometimes fatal ventricular fibrillation. Beta-blockers include atenolol (Tenormin), metoprolol (Lopressor), nadolol, pindolol (Visken), propranolol (Inderal), and timolol (Blocadren). Nitrates, a type of vasodilator, also are given right after a heart attack to help improve the delivery of blood to the heart and ease heart failure symptoms. Nitrates include isosorbide mononitrate (Imdur), isosorbide dinitrate (Isordil, Sorbitrate), and nitroglycerin (Nitrostat).

When a heart attack causes an abnormal heart-beat, arrhythmia drugs may be given to restore the heart's normal rhythm. These include: amiodarone (Cordarone), atropine, bretylium, disopyramide (Norpace), lidocaine (Xylocaine), procainamide (Procan), propafenone (Rythmol), propranolol (Inderal), quinidine, and sotalol (Betapace). Angiotensin-converting enzyme (ACE) inhibitors reduce the resistance against which the heart beats and are used to manage and prevent heart failure. They are used to treat heart attack patients whose hearts do not pump well or who have symptoms of heart failure.

Taken orally, they include Altace, Capoten, Lotensin, Monopril, Prinivil, Vasotec, and Zestril. Angiotensin receptor blockers, such as losartan (Cozaar) may substitute. Diuretics can help get rid of excess fluids that sometimes accumulate when the heart is not pumping effectively. Usually taken orally, they cause the body to dispose of fluids through urination. Common diuretics include: bumetanide (Bumex), chlorthalidone (Hygroton), chlorothiazide (Diuril), furosemide (Lasix),
, symptoms recur in only about three or four percent of patients per year. Five years after bypass surgery, survival expectancy is 90%, at 10 years it is about 80%, at 15 years it is about 55%, and at 20 years it is about 40%. hydrochlorothiazide (HydroDIRUIL, Esidrix), spironolactone (Aldactone), and triamterene (Dyrenium).

Percutaneous transluminal coronary angioplasty and coronary artery bypass surgery are invasive revascularisation procedures that open blocked coronary arteries and improve blood flow. They usually are performed only on patients for whom clot-dissolving drugs do not work, or who have poor exercise stress tests, poor left ventricular function, or ischemia. Generally, angioplasty is performed before coronary artery bypass surgery. Percutaneous transluminal coronary angioplasty, usually called coronary angioplasty, is a non-surgical procedure in which a catheter (a tiny plastic tube) tipped with a balloon is threaded from a blood vessel in the thigh or arm into the blocked artery. The balloon is inflated and compresses the plaque to enlarge the blood vessel and open the blocked artery. The balloon is then deflated, and the catheter is removed. Coronary angioplasty is performed in a hospital and generally

requires a two-day stay. It is successful about 90% of the time. For one third of patients, the artery narrows again within six months after the procedure. The procedure can be repeated. It is less invasive and less expensive than coronary artery bypass surgery.

In coronary artery bypass surgery, called bypass surgery, a detour is built around the coronary artery blockage with a healthy leg or chest wall artery or vein. The healthy vein then supplies oxygen-rich blood to the heart. Bypass surgery is major surgery appropriate for patients with blockages in two or three major coronary arteries or severely narrowed left main coronary arteries, as well as those who have not responded to other treatments. It is performed in a hospital under general anesthesia using a heart-lung machine to support the patient while the healthy vein is attached to the coronary artery. About 70% of patients who have bypass surgery experience full relief from angina; about 20% experience partial relief. There are several experimental surgical procedures for unblocking coronary arteries under study including: atherectomy, where the surgeon shaves off and removes strips of plaque from the blocked artery; laser angioplasty, where a catheter with a laser tip is inserted to burn or break down the plaque; and insertion of a metal coil called a stent that can be implanted permanently to keep a blocked artery open.

3.2.3 Prevention

Many heart attacks can be prevented through a healthy lifestyle, which can reduce the risk of developing coronary artery disease. For patients who have already had a heart attack, a healthy lifestyle and carefully following doctor's orders can prevent another heart attack. A heart healthy lifestyle includes eating right, regular exercise, maintaining a healthy weight, no smoking, moderate drinking, no illegal drugs, controlling hypertension, and managing stress.

A healthy diet includes a variety of foods that are low in fat (especially saturated fat), low in cholesterol, and high in fiber; plenty of fruits and vegetables; and limited sodium. Some foods are low in fat but high in cholesterol, and some are low in cholesterol but high in fat. Saturated fat raises cholesterol, and, in excessive amounts, it increases the amount of the proteins in blood that form blood clots. Polyunsaturated and monounsaturated fats are relatively good for the heart. Fat should comprise no more than 30 percent of total daily calories. Cholesterol, a waxy, lipid-like substance, comes from eating foods such as meat, eggs, and other animal products. It also is produced in the liver. Soluble fiber can help lower cholesterol. Cholesterol should be limited to about 300 mg per day. Many popular lipid-lowering drugs can reduce LDL-cholesterol by an average of 25-30% when combined with a low-fat, low-cholesterol diet. Fruits and vegetables are rich in fiber, vitamins,

and minerals. They are also low calorie and nearly fat free. Vitamin C and beta-carotene, found in many fruits and vegetables, keep LDL-cholesterol from turning into a form that damages coronary arteries. Excess sodium can increase the risk of high blood pressure. Many processed foods contain large amounts of sodium, which should be limited to a daily intake of 2,400 mg—about the amount in a teaspoon of salt.

The "Food Guide" Pyramid developed by the U.S. Departments of Agriculture and Health and Human Services provides easy to follow guidelines for daily heart-healthy eating: six to 11 servings of bread, cereal, rice, and pasta; three to five servings of vegetables; two to four servings of fruit; two to three servings of milk, yogurt, and cheese; and two to three servings of meat, poultry, fish, dry beans, eggs, and nuts. Fats, oils, and sweets should be used sparingly. Regular aerobic exercise can lower blood pressure, help control weight, and increase HDL ("good") cholesterol. It may keep the blood vessels more flexible. Moderate intensity aerobic exercise lasting about 30 minutes four or more times per week is recommended for maximum heart health, according to the Centers for Disease Control and Prevention and the American College of Sports Medicine. Three 10-minute exercise periods also are beneficial. Aerobic exercise—activities such as walking, jogging, and cycling—uses the large muscle groups and forces the body to use oxygen more efficiently. It also can include everyday activities such as active gardening, climbing stairs, or brisk housework.

Maintaining a desirable body weight also is important. About one-fourth of all Americans are overweight, and nearly one-tenth are obese, according to the Surgeon General's Report on Nutrition and Health. People who are 20% or more over their ideal body weight have an increased risk of developing coronary artery disease. Losing weight can help reduce total and LDL cholesterol, reduce triglycerides, and boost relative levels of HDL cholesterol. It also may reduce blood pressure.
Smoking has many adverse effects on the heart. It increases the heart rate, constricts major arteries, and can create irregular heartbeats. It also raises blood pressure, contributes to the development of plaque, increases the formation of blood clots, and causes blood platelets to cluster and impede blood flow. Heart damage caused by smoking can be repaired by quitting—even heavy smokers can return to heart health. Several studies have shown that ex-smokers face the same risk of heart disease as non-smokers within five to 10 years of quitting.

Drinking should be done in moderation. Modest consumption of alcohol can actually protect against coronary artery disease. This is believed to be because alcohol raises HDL cholesterol levels. The American Heart Association defines moderate consumption as one ounce of alcohol per

day—roughly one cocktail, one 8-ounce glass of wine, or two 12-ounce glasses of beer. A study released in 2003 reported that risk of heart attack in men was reduced 30% to 35 % if they drank moderate amounts of alcoholic beverages three or four times a week. In some people, however, moderate drinking can increase risk factors for heart disease, such as raising blood pressure. Excessive drinking is always bad for the heart. It usually raises blood pressure and can poison the heart and cause abnormal heart rhythms or even heart failure. Illegal drugs, like cocaine, can seriously harm the heart and should never be used. High blood pressure, one of the most common and serious risk factors for coronary artery disease, can be completely controlled through lifestyle changes and medication. People with moderate hypertension may be able to control it through lifestyle changes such as reducing sodium and fat, exercising regularly, managing stress, quitting smoking, and drinking alcohol in moderation. If these changes do not work, and for people with severe hypertension, there are eight types of drugs that provide effective treatment.

Stress management means controlling mental and physical reactions to life's irritations and challenges. Techniques for controlling stress include: taking life more slowly, spending time with family and friends, thinking positively, getting enough sleep, exercising, and practicing relaxation techniques. Daily aspirin therapy has been proven to help prevent blood clots associated with atherosclerosis. It also can prevent heart attacks from recurring, prevent heart attacks from being fatal, and lower the risk of strokes.

3.3 Stroke (Cerebrovascular Accident)

Cerebrovascular Accident (CVA) or stroke, is a general term that describes injury or death of brain tissue usually due to interruption of cerebral blood flow from either Ischaemic or Haemorrhagic lesions, (when the blood supply to a part of the brain is cut off; this impairs the function s of certain nerves within the brain and also impairs the body functions controlled by the nerve. The effects may be slight or severe). These lesions are commonly secondary to artherosclerotic disease, hypertension, or both. The patient tends to experience a sudden loss of consciousness followed by paralysis.

This may be caused by hemorrhage into the brain tissue, an embolus in the cerebral blood vessels, or thrombus formation that occludes arterial supply to the brain. Additionally, a rupture of an artery causing hemorrhage, usually in the subarachnoid space, can cause stroke. Strokes are the third most common cause of death, and in middle-aged and older patients, are a frequent cause of disability. They are divided into two broad categories:

(i) Infarction (Ischemic or Cerebral Thrombosis or Clot Stroke): An infarction occurs when the blood supply to a limited portion of the brain is inadequate and death of nervous tissue follows. Infarction may be caused by an embolism or by blood vessel occlusion due to atherosclerosis (thrombus). Emboli are usually small clots arising from diseased blood vessels in the neck(carotid) or from clots originating in the heart. Atrial fibrillation often causes atrial dilation, a precursor to the formation of clots. Other types of emboli that may cause occlusion in cerebral blood vessels are air, tumor tissue, and fat.

(ii) Hemorrhage: Hemorrhages are usually categorised as being either intracerebral or subarachnoid. Onset is often sudden and marked by a severe headache and stiff neck. Most intracranial hemorrhages occur in the hypertensive patient when a small vessel deep within the brain tissue ruptures. Subarachnoid hemorrhages most often result from congenital blood vessel abnormalities or from head trauma. The congenital abnormalities are known as aneurysms (weakened vessels) and arteriovenous malformations (collections of abnormal blood vessels). Aneurysms tend to be on the surface and may hemorrhage into the brain tissue or the subarachnoid space. Arteriovenous malformations may be within the brain, subarachnoid space or both.

Hemorrhage inside the brain often tears and separates normal brain tissue. The release of the blood into the cavities containing spinal fluid within the brain may paralyse vital centers. If the blood in the subarachnoid space impairs drainage of cerebrospinal fluid, it may cause a rise in the intracranial pressure. Herniation (protrusion) of brain tissue through a narrow opening in the skull may then occur. In infarction, the tissue that has died will swell, causing further damage to nearby tissue, which only has a marginal blood supply. If swelling is severe, herniation may result.

Clinical presentation of a stroke

Symptoms of a patient who has experienced a stroke will depend upon the area of the brain damaged. Those areas commonly affected are the motor, speech, and sensory centers. The onset of symptoms will be acute, and the patient may experience unconsciousness.

There may be stertorous breathing (laborious breathing accompanied by snoring) due to paralysis of a portion of the soft palate. Respiratory expiration may be puffs of air out of the cheeks and mouth. The patient’s pupils may be unequal with larger pupil on the side of the

hemorrhage. Paralysis will usually involve one side of the face, one arm, and one leg. The eyes often will be turned away from the side of the body paralysis. The patient's skin may be cool and clammy. Speech disturbances, aphasia, also may be noted. Predisposing factors that possibly contribute to the stroke include; hypertension, diabetes, abnormal blood lipid levels, oral contraceptives, sickle disease, and some cardiac dysrhythmias.

Distinguishing transient ischemic attack (TIAs)

Some patients may have small emboli that cause transient stroke-like symptoms known as TIAs, or Transient Ischemic Attack. These emboli temporarily interfere with the blood supply to the brain, producing symptoms of neurological deficit. These symptoms may last for only a few minutes or for several hours. After the attack, the patient will show no evidence of residual brain or neurological damage. The patient who experiences a TIA may, however, be a candidate for an eventual CVA. The onset of a transient ischemic attack is usually abrupt, with the patient experiencing giddiness or a light-headed sensation. The specific signs and symptoms depend upon the area of the brain affected.

Prevention

Given the disease burden of strokes, prevention is an important public health concern.Primary prevention is less effective than secondary prevention (as judged by the number needed to treat to prevent one stroke per year). Recent guidelines detail the evidence for primary prevention in stroke. In those who are otherwise healthy, aspirin does not appear beneficial and thus is not recommended. In people who have had a myocardial infarction or those with a high cardiovascular risk, it provides some protection against a first stroke. In those who have previously had a stroke, treatment with medications such as aspirin, clopidogrel and dipyridamole may be beneficial. The U.S. Preventive Services Task Force (USPSTF) recommends against screening for carotid artery stenosis in those without symptoms.

Risk factors

The most important modifiable risk factors for stroke are high blood pressure and atrial fibrillation (although magnitude of this effect is small: the evidence from the Medical Research Council trials is that 833 patients have to be treated for 1 year to prevent one stroke. Other modifiable risk factors include high blood cholesterol levels, diabetes mellitus, cigarette smoking (active and passive), heavy alcohol consumption and drug use, lack of physical activity, obesity, processed red meat consumption and unhealthy diet. Alcohol use could predispose to ischemic stroke, and intracerebral and subarachnoid hemorrhage via multiple mechanisms (for example via hypertension, atrial fibrillation, rebound thrombocytosis and platelet aggregation and clotting

disturbances). Drugs, most commonly amphetamines and cocaine, can induce stroke through damage to the blood vessels in the brain and/or acute hypertension.

No high-quality studies have shown the effectiveness of interventions aimed at weight reduction, promotion of regular exercise, reducing alcohol consumption or smoking cessation. Nonetheless, given the large body of circumstantial evidence, best medical management for stroke includes advice on diet, exercise, smoking and alcohol use. Medication or drug therapy is the most common method of stroke prevention; carotid endarterectomy can be a useful surgical method of preventing stroke.

Blood pressure

Hypertension (high blood pressure) accounts for 35-50% of stroke risk. Blood pressure reduction of 10 mmHg systolic or 5 mmHg diastolic reduces the risk of stroke by ~40%. Lowering blood pressure has been conclusively shown to prevent both ischemic and hemorrhagic strokes. It is equally important in secondary prevention. Even patients older than 80 years and those with isolated systolic hypertension benefit from antihypertensive therapy. The available evidence does not show large differences in stroke prevention between antihypertensive drugs — therefore, other factors such as protection against other forms of cardiovascular disease and cost should be considered. The routine use of beta-blockers following a stroke or TIA has not been shown to result in benefits.

Blood lipids

High cholesterol levels have been inconsistently associated with (ischemic) stroke. Statins have been shown to reduce the risk of stroke by about 15%. Since earlier meta-analyses of other lipid-lowering drugs did not show a decreased risk, statins might exert their effect through mechanisms other than their lipid-lowering effects.

Diabetes mellitus

Diabetes mellitus increases the risk of stroke by two to three times. While intensive blood sugar control has been shown to reduce small blood vessel complications such as kidney damage and damage to the retina of the eye it has not been shown to reduce large blood vessel complications such as stroke.

Anticoagulation drugs

Oral anticoagulants such as warfarin have been the mainstay of stroke prevention for over 50 years. However, several studies have shown that aspirin and antiplatelet drugs are highly effective in secondary prevention after a stroke or transient ischemic attack. Low doses of

aspirin (for example 75–150 mg) are as effective as high doses but have fewer side effects; the lowest effective dose remains unknown. Thienopyridines (clopidogrel, ticlopidine) "might be slightly more effective" than aspirin and have a decreased risk of gastrointestinal bleeding, but are more expensive. Their exact role remains controversial.

Ticlopidine is associated with higher rates of skin rash, diarrhea, low blood neutrophil count and thrombotic thrombocytopenic purpura. Dipyridamole can be added to aspirin therapy to provide a small additional benefit, even though headache is a common side effect. Low-dose aspirin is also effective for stroke prevention after having a myocardial infarction. Those with atrial fibrillation have a 5% a year risk of stroke, and this risk is higher in those with valvular atrial fibrillation. Depending on the stroke risk, anticoagulation with medications such as warfarin or aspirin is useful for prevention. Except in people with atrial fibrillation, oral anticoagulants are not advised for stroke prevention —any benefit is offset by bleeding risk. In primary prevention however, antiplatelet drugs did not reduce the risk of ischemic stroke but increased the risk of major bleeding. Further studies are needed to investigate a possible protective effect of aspirin against ischemic stroke in women.

Surgery

Carotid endarterectomy or carotid angioplasty can be used to remove atherosclerotic narrowing (stenosis) of the carotid artery. There is evidence supporting this procedure in selected cases. Endarterectomy for a significant stenosis has been shown to be useful in the prevention of further strokes in those who have already had one. Carotid artery stenting has not been shown to be equally useful. Patients are selected for surgery based on age, gender, degree of stenosis, time since symptoms and patients' preferences. Surgery is most efficient when not delayed too long —the risk of recurrent stroke in a patient who has a 50% or greater stenosis is up to 20% after five years, but endarterectomy reduces this risk to around 5%. The number of procedures needed to cure one patient was five for early surgery (within two weeks after the initial stroke), but 125 if delayed longer than 12 weeks.

Screening for carotid artery narrowing has not been shown to be a useful screening test in the general population. Studies of surgical intervention for carotid artery stenosis without symptoms have shown only a small decrease in the risk of stroke. To be beneficial, the complication rate of the surgery should be kept below 4%. Even then, for 100 surgeries, fivepatients will benefit by avoiding stroke, three will develop stroke despite surgery, three will develop stroke or die due to the surgery itself,

and 89 will remain stroke-free but would also have done so without intervention.

Diet
Nutrition, specifically the Mediterranean-style diet, has the potential for decreasing the risk of having a stroke by more than half. It does not appear that lowering levels of homocysteine with folic acid affects the risk of stroke.

Women
A number of specific recommendations have been made for women including taking aspirin after the 11th week of pregnancy if there is a history of previous chronic high blood pressure and taking blood pressure medications during pregnancy if the blood pressure is greater than 150 mmHg systolic or greater than 100 mmHg diastolic. In those who have previously had preeclampsia other risk factors should be treated more aggressively.

Previous stroke or TIA
Keeping blood pressure below 140/90 mmHg is recommended. Anticoagulation can prevent recurrent ischemic strokes. Among people with nonvalvular atrial fibrillation, anticoagulation can reduce stroke by 60% while antiplatelet agents can reduce stroke by 20%. However, a recent meta-analysis suggests harm from anti-coagulation started early after an embolic stroke. Stroke prevention treatment for atrial fibrillation is determined according to the CHADS/CHADS2 system. The most widely used anticoagulant to prevent thromboembolic stroke in patients with nonvalvular atrial fibrillation is the oral agent warfarin while a number of newer agents including dabigatran are alternatives which do not require prothrombin time monitoring. Anticoagulants, when used following stroke, should not be stopped for dental procedures. If studies show carotid stenosis, and the person has residual function in the affected side, carotid endarterectomy (surgical removal of the stenosis) may decrease the risk of recurrence if performed rapidly after stroke.

Management
Ischemic stroke: Definitive therapy is aimed at removing the blockage by breaking the clot down (thrombolysis), or by removing it mechanically (thrombectomy). The philosophical premise underlying the importance of rapid stroke intervention was crystallised as *Time is Brain!* in the early 1990s. Years later, that same idea, that rapid cerebral blood flow restoration results in fewer brain cells dying, has been proved and quantified.

Tight blood sugar control in the first few hours does not improve outcomes and may cause harm. High blood pressure is also not typically lowered as this has not been found to be helpful.[113]

Thrombolysis

Thrombolysis, such as with Recombinant Tissue Plasminogen Activator (RTPA), in acute ischemic stroke, when given within three hours of symptom onset results in an overall benefit of 10% with respect to living without disability. It does not, however, improve chances of survival. Benefit is greater the earlier it is used. Between three and four and a half hours the effects are less clear. A 2014 review found a 5% increase in the number of people living without disability at three to six months; however, there was a 2% increased risk of death in the short term. After four and a half hours thrombolysis worsens outcomes. These benefits or lack of benefits occurred regardless of the age of the person treated. There is no reliable way to determine who will have an intracranial hemorrhage post treatment versus who will not.

Its use is endorsed by the American Heart Association and the American Academy of Neurology as the recommended treatment for acute stroke within three hours of onset of symptoms as long as there are not other contraindications (such as abnormal lab values, high blood pressure, or recent surgery). This position for TPA is based upon the findings of two studies by one group of investigators which showed that TPA improves the chances for a good neurological outcome. When administered within the first three hours thrombolysis improves functional outcome without affecting mortality. 6.4% of people with large strokes developed substantial brain hemorrhage as a complication from being given TPA thus part of the reason for increased short-term mortality. Additionally, the American Academy of Emergency Medicine states that objective evidence regarding the efficacy, safety, and applicability of TPA for acute ischemic stroke is insufficient to warrant its classification as standard of care. Intra-arterial fibrinolysis, where a catheter is passed up an artery into the brain and the medication is injected at the site of thrombosis, has been found to improve outcomes in people with acute ischemic stroke.

Hemicraniectomy

Large territory strokes can cause significant brain swelling with secondary brain injury in surrounding tissue. This phenomenon is mainly encountered in strokes of the middle cerebral artery territory and is also called "malignant cerebral infarction" because it carries a dismal prognosis. Relief of the pressure may be attempted with medication, but some require hemicraniectomy, the temporary surgical removal of the skull on one side of the head. This decreases the risk of death, although

some more people survive with disability who would otherwise have died.

Hemorrhagic stroke

People with intracerebral hemorrhage require neurosurgical evaluation to detect and treat the cause of the bleeding, although many may not need surgery. Anticoagulants and antithrombotics, key in treating ischemic stroke, can make bleeding worse. People are monitored for changes in the level of consciousness, and their blood pressure, blood sugar, and oxygenation are kept at optimum levels.

Stroke unit

Ideally, people who have had a stroke are admitted to a "stroke unit", a ward or dedicated area in hospital staffed by nurses and therapists with experience in stroke treatment. It has been shown that people admitted to a stroke unit have a higher chance of surviving than those admitted elsewhere in hospital, even if they are being cared for by doctors without experience in stroke. When an acute stroke is suspected by history and physical examination, the goal of early assessment is to determine the cause. Treatment varies according to the underlying cause of the stroke, thromboembolic (ischemic) or hemorrhagic.

Rehabilitation

Stroke rehabilitation is the process by which those with disabling strokes undergo treatment to help them return to normal life as much as possible by regaining and relearning the skills of everyday living. It also aims to help the survivor understand and adapt to difficulties, prevent secondary complications and educate family members to play a supporting role.

A rehabilitation team is usually multidisciplinary as it involves staff with different skills working together to help the patient. These include physicians trained in rehabilitation medicine, clinical pharmacists, nursing staff, physiotherapists, occupational therapists, speech and language therapists, and orthotists. Some teams may also include psychologists and social workers, since at least one third of the people manifest post stroke depression. Validated instruments such as the Barthel scale may be used to assess the likelihood of a stroke patient being able to manage at home with or without support subsequent to discharge from hospital.

Good nursing care is fundamental in maintaining skin care, feeding, hydration, positioning, and monitoring vital signs such as temperature, pulse, and blood pressure. Stroke rehabilitation begins almost immediately. For most people with stroke, physical therapy (PT), occupational therapy (OT) and speech-language pathology (SLP) are the cornerstones of the rehabilitation process. Often, assistive technology such as wheelchairs, walkers and canes may be beneficial. Many

mobility problems can be improved by the use of ankle foot orthoses. PT and OT have overlapping areas of expertise; however PT focuses on joint range of motion and strength by performing exercises and re-learning functional tasks such as bed mobility, transferring, walking and other gross motor functions. Physiotherapists can also work with patients to improve awareness and use of the hemiplegic side. Rehabilitation involves working on the ability to produce strong movements or the ability to perform tasks using normal patterns. Emphasis is often concentrated on functional tasks and patient's goals. One example physiotherapists employ to promote motor learning involves constraint-induced movement therapy. Through continuous practice the patient relearns to use and adapt the hemiplegic limb during functional activities to create lasting permanent changes. OT is involved in training to help relearn everyday activities known as the Activities of daily living (ADLs) such as eating, drinking, dressing, bathing, cooking, reading and writing, and toileting. Speech and language therapy is appropriate for patients with the speech production disorders: dysarthria and apraxia of speech, aphasia, cognitive-communication impairments and/or dysphagia (problems with swallowing).

Patients may have particular problems, such as dysphagia, which can cause swallowed material to pass into the lungs and cause aspiration pneumonia. The condition may improve with time, but in the interim, a nasogastric tube may be inserted, enabling liquid food to be given directly into the stomach. If swallowing is still deemed unsafe, then a percutaneous endoscopic gastrostomy (PEG) tube is passed and this can remain indefinitely.

Treatment of spasticity related to stroke often involves early mobilisations, commonly performed by a physiotherapist, combined with elongation of spastic muscles and sustained stretching through various positioning. Gaining initial improvement in range of motion is often achieved through rhythmic rotational patterns associated with the affected limb. After full range has been achieved by the therapist, the limb should be positioned in the lengthened positions to prevent against further contractures, skin breakdown, and disuse of the limb with the use of splints or other tools to stabilise the joint. Cold in the form of ice wraps or ice packs have been proven to briefly reduce spasticity by temporarily dampening neural firing rates. Electrical stimulation to the antagonist muscles or vibrations has also been used with some success.

Stroke rehabilitation should be started as quickly as possible and can last anywhere from a few days to over a year. Most return of function is seen in the first few months, and then improvement falls off with the "window" considered officially by U.S. state rehabilitation units and others to be closed after six months, with little chance of further

improvement. However, patients have been known to continue to improve for years, regaining and strengthening abilities like writing, walking, running, and talking. Daily rehabilitation exercises should continue to be part of the stroke patient's routine. Complete recovery is unusual but not impossible and most patients will improve to some extent: proper diet and exercise are known to help the brain to recover.

Some current and future therapy methods include the use of virtual reality and video games for rehabilitation. These forms of rehabilitation offer potential for motivating patients to perform specific therapy tasks that many other forms do not. Many clinics and hospitals are adopting the use of these off-the-shelf devices for exercise, social interaction and rehabilitation because they are affordable, accessible and can be used within the clinic and home.

Other novel non-invasive rehabilitation methods are currently being developed to augment physical therapy to improve motor function of stroke patients, such as Transcranial Magnetic Stimulation (TMS) and Transcranial Direct-Current Stimulation (TDCS) and robotic therapies. A stroke can also reduce people's general fitness. Reduced fitness can reduce capacity for rehabilitation as well as general health. A systematic review found that there are inadequate long-term data about the effects of exercise and training on death, dependence and disability after a stroke. However, cardiorespiratory training added to walking programs in rehabilitation can improve speed, tolerance and independence during walking.

3.4 Chronic Cardiac (Heart) Failure

Heart Failure (**HF**), often used to mean **Chronic Heart Failure** (**CHF**), occurs when the heart is unable to pump sufficiently to maintain blood flow to meet the body's needs. The terms **Congestive Heart Failure** (**CHF**) or **Congestive Cardiac Failure** (**CCF**) are often used interchangeably with chronic heart failure. Signs and symptoms commonly include shortness of breath, excessive tiredness, and leg swelling.

The shortness of breath is usually worse with exercise, while lying down, and may wake the person at night. A limited ability to exercise is also a common feature. Common causes of heart failure include coronary artery disease including a previous myocardial infarction (heart attack), high blood pressure, atrial fibrillation, valvular heart disease, excess alcohol use, infection, and cardiomyopathy of an unknown cause. These cause heart failure by changing either the structure or the functioning of the heart. There are two main types of heart failure: *heart failure due to left ventricular dysfunction* and *heart failure with normal*

ejection fraction depending on if the ability of the left ventricle to contract is affected, or the heart's ability to relax. The severity of disease is usually graded by the degree of problems with exercise. [

Heart failure is not the same as myocardial infarction (in which part of cardiac arrest (in which blood flow stops altogether). Other diseases similar to heart failure include obesity, kidney failure, liver problems, an The condition is diagnosed based on the history of the symptoms and a confirmation by echocardiography . Blood tests, electrocardiography, a be useful to determine the underlying cause.Treatment depends on the disease. In people with chronic stable mild heart failure, treatment com modifications such as stopping smoking, physical exercise, and die medications. In those with heart failure due to left ventricular dysfunctio enzyme inhibitors or angiotensin receptor blockers along with beta bl For those with severe disease, aldosterone antagonists, or hydralazine p Diuretics are useful for preventing fluid retention. Sometimes, dep implanted device such as a pacemaker or an implantable cardi recommended. In some moderate or severe cases cardiac resynchronisati suggested or cardiac contractility modulation may be of benefit. A v occasionally a heart transplant may be recommended in those with seve measures. Heart Failure is a common, costly, and potentially fata countries, around 2% of adults have heart failure and in those over the a 6–10%. In the year after diagnosis the risk of death is about 35% after w 10% each year. This is similar to the risks with a number of types Kingdom the disease is the reason for 5% of emergency hospital adm been known since ancient times with the Eberspapyrus commenting on i

3.4.1 Signs and Symptoms

Heart failure symptoms are traditionally and somewhat arbitrarily divi sided, recognising that the left and right ventricles of the heart supply circulation. However, heart failure is not exclusively *backward fai* circulation which drains to the ventricle). There are several other excep division of heart failure symptoms. Additionally, the most common failure is left-sided heart failure. The result is that patients commonly signs and symptoms.

Left-sided failure

Common respiratory signs are increased rate of breathing and increase specific signs of respiratory distress). Rales or crackles, heard initially in severe, throughout the lung fields suggest the development of pulmo alveoli). Cyanosis which suggests severe hypoxemia, is a late sign of ex edema. Additional signs indicating left ventricular failure include a lat (which occurs if the heart is enlarged) and a gallop rhythm (additional h as a marker of increased blood flow, or increased intra-cardiac pressure. Heart murmurs may indicate the presence of valvular heart disease, eit

stenosis) or as a result (e.g. mitral regurgitation) of the heart failure. *Ba* ventricle causes congestion of the lungs' blood vessels, and so the sym respiratory in nature Backward failure can be subdivided into failure o ventricle or both within the left circuit. The patient will have dyspnea exertion and in severe cases, dyspnea at rest. Increasing breathlessn orthopnea, occurs. It is often measured in the number of pillows requir in orthopnea, the patient may resort to sleeping while sitting up. Anothe is paroxysmal nocturnal dyspnea: a sudden nighttime attack of seve several hours after going to sleep. Easy fatigability and exercise into complaints related to respiratory compromise."Cardiac asthma" o Compromise of left ventricular *forward* function may result in sym circulation such as dizziness, confusion and cool extremities at rest.

Right-sided failure

Physical examination may reveal pitting peripheral edema, ascites, and venous pressure is frequently assessed as a marker of fluid status, whi eliciting hepatojugular reflux. If the right ventricular pressure is incre may be present, signifying the compensatory increase in contraction stre the right ventricle leads to congestion of systemic capillaries. Thi accumulation in the body. This causes swelling under the (termed perip and usually affects the dependent parts of the body first (causing foot a who are standing up, and sacral edema in people who are predominan (frequent nighttime urination) may occur when fluid from the legs is re while lying down at night. In progressively severe cases, ascites (f abdominal cavity causing swelling) and liver enlargement may d congestion may result in impaired liver function, and jaundice and eve of decreased blood clotting) may occur.

Biventricular failure

Dullness of the lung fields to finger percussion and reduced breath soun may suggest the development of a pleural effusion (fluid collection in chest wall). Though it can occur in isolated left- or right-sided heart fail biventricular failure because pleural veins drain into both the systemi systems. When unilateral, effusions are often right sided.

3.4.2 Causes

Congestive heart failure

Heart failure may also occur in situations of "high output," (termed "hig where the ventricular systolic function is normal but the heart cannc augmentation of blood volume. This can occur in overload situation (b renal diseases, chronic severe anemia, beriberi (vitamin B_1/thiamine d Paget's disease, arteriovenous fistulae, or arteriovenous malformations.

The following risk factors occur:

(i) Ischemic heart disease 62%
(ii) Cigarette smoking 16%
(iii) Hypertension (high blood pressure) 10%
(iv) Obesity 8%
(v) Diabetes 3%
(vi) Valvular heart disease 2%(much higher in older populations)

3.4.3 Management

Treatment focuses on improving the symptoms and preventing the progr
Reversible causes of the heart failure also need to be addressed (eg: in
anaemia, Thyrotoxicosis, arrhythmia, hypotension). Treatments
pharmacological modalities, and occasionally various forms of device t
transplantation.

Acute decompensation
In Acute Decompensated Heart Failure (ADHF), the immediate goal is to re-establish adequate perfusion and oxygen delivery to end organs. This entails ensuring that airway, breathing, and circulation are adequate. Immediate treatments usually involve some combination of vasodilators such as nitroglycerin, diuretics such as furosemide, and possibly Non -Invasive Positive Pressure Ventilation (NIPPV).

Chronic management
The goals of treatment for people with chronic heart failure are the prolongation of life, the prevention of acute decompensation and the reduction of symptoms, allowing for greater activity.
Heart failure can result from a variety of conditions. In considering therapeutic options, it is important to first exclude reversible causes, including thyroid disease, anemia, chronic tachycardia, alcohol abuse, hypertension and dysfunction of one or more heart valves.
Treatment of the underlying cause is usually the first approach in treating heart failure. However, in the majority of cases, either no primary cause is found, or treatment of the primary cause does not restore normal heart function. In these cases, behavioral, medical and device treatment strategies exist which can provide significant improvement in outcomes, including the relief of symptoms, exercise tolerance, and a decrease in the likelihood of hospitalisation or death.

Lifestyle
Behavioral modification is a primary consideration in any chronic heart failure management program, with dietary guidelines regarding fluid and salt intake being of particular importance.

Exercise should be encouraged and tailored to suit individual capabilities. The inclusion of regular physical conditioning as part of a cardiac rehabilitation program can significantly improve quality of life and reduce the risk of hospital admission for worsening symptoms however there is no evidence for a reduction in mortality rates as a result of exercise. Furthermore, it is not clear whether this evidence can be extended to people with Heart Failure with Preserved Ejection Fraction (HFPEF) or to those whose exercise regimen takes place entirely at home. Home visits and regular monitoring at heart failure clinics reduce the need for hospitalization and improve life expectancy.

Medication

First-line therapy for people with heart failure due to reduced systolic function should include angiotensin-Converting Enzyme (ACE) inhibitors (ACE-I) or Angiotensin Receptor Blockers (ARBs). Use of medicines from this class are associated with improved survival and quality of life in people with heart failure. Beta-adrenergic blocking agents (beta blockers) also form part of the first line of treatment, adding to the improvement in symptoms and mortality provided by ACE-I/ARB. The mortality benefits of beta blockers in people with systolic dysfunction who also have Atrial Fibrillation (AF) is more limited than in those who do not have AF. If the ejection fraction is not diminished (HFPEF), the benefits of beta blockers is more modest; a decrease in mortality has been observed but reduction in hospital admission for uncontrolled symptoms has not been observed. In people who are intolerant of ACE-I and ARBs or who have significant renal dysfunction, the use of combined hydralazine and a long-acting nitrate, such as isosorbide dinitrate, are an effective alternative. This regimen has been shown to reduce mortality in people with moderate heart failure. It is especially beneficial in African-Americans (AA). In AAs who are symptomatic, hydralazine and isosorbide dinitrate (H+I) can be added to ACE-I or ARBs.

In people with markedly reduced ejection fraction, the use of an aldosterone antagonist, in addition to beta blockers and ACE-I, can improve symptoms and reduce mortality. Second-line drugs for CHF do not confer a mortality benefit. Digitalis is one such drug. Its narrow therapeutic window, high degree of toxicity, and the failure of multiple trials to show a mortality benefit have reduced its role in clinical practice. It is now used in only a small number of people with refractory symptoms, who are in atrial fibrillation and/or who have chronic hypotension. Diuretics have been a mainstay of treatment for treatment of fluid accumulation, and include diuretics classes such as loop diuretics, thiazide-like diuretic, and potassium-sparing diuretic. Although widely used, evidence on their efficacy and safety is limited, except for spironolactone antagonists. A recent Cochrane review found

that in small studies, the use of diuretics appeared to have improved mortality in individuals with heart failure. However, the extent to which these results can be extrapolated to a general population is unclear due to the small number of participants in the cited studies. Anemia is an independent factor in mortality in people with chronic heart failure. The treatment of anemia significantly improves quality of life for those with heart failure, often with a reduction in severity of the NYHA classification, and also improves mortality rates. The latest European guidelines (2012) recommend screening for iron-deficient anemia and treatinventricular tachycardiag with parenteral iron if anemia is found.

Minimally invasive therapies

In people with severe cardiomyopathy (left ventricular ejection fraction below 35%), or in those with recurrent VT or malignant arrhythmias, treatment with an Automatic Implantable Cardioverter Defibrillator (AICD) is indicated to reduce the risk of severe life-threatening arrhythmias. The AICD does not improve symptoms or reduce the incidence of malignant arrhythmias, but does reduce mortality from those arrhythmias, often in conjunction with antiarrhythmic medications. In people with left ventricular ejection (LVEF) below 35%, the incidence of (VT) or sudden cardiac death is high enough to warrant AICD placement. Its use is therefore recommended in AHA/ACC guidelines.

Cardiac Contractility Modulation (CCM) is a treatment for people with moderate to severe left ventricular systolic heart failure (NYHA class II–IV) which enhances both the strength of ventricular contraction and the heart's pumping capacity. The CCM mechanism is based on stimulation of the cardiac muscle by Non-Excitatory Electrical Signals (NES), which are delivered by a pacemaker-like device. CCM is particularly suitable for the treatment of heart failure with normal QRS complex duration (120 ms or less) and has been demonstrated to improve the symptoms, quality of life and exercise tolerance. CCM is approved for use in Europe, but not currently in North America.

About one third of people with LVEF below 35% have markedly altered conduction to the ventricles, resulting in dyssynchronous depolarization of the right and left ventricles. This is especially problematic in people with left bundle branch block (blockage of one of the two primary conducting fiber bundles that originates at the base of the heart and carries depolarising impulses to the left ventricle). Using a special pacing algorithm, biventricular cardiac resynchronisation therapy (CRT) can initiate a normal sequence of ventricular depolarisation.

In people with LVEF below 35% and prolonged QRS duration on ECG (LBBB or QRS of 150 ms or more) there is an improvement in

symptoms and mortality when CRT is added to standard medical therapy. However, in the two thirds of people without prolonged QRS duration, CRT may actually be harmful.

Surgical therapies

People with the most severe heart failure may be candidates for Ventricular Assist Devices (VAD). VADs have commonly been used as a bridge to heart transplantation but have been used more recently as a destination treatment for advanced heart failure. In select cases, heart transplantation can be considered. While this may resolve the problems associated with heart failure, the person must generally remain on an immunosuppressive regimen to prevent rejection, which has its own significant downsides. A major limitation of this treatment option is the scarcity of hearts available for transplantation.

Palliative care

People with CHF often have significant symptoms, such as shortness of breath and chest pain. Both palliative care and cardiology are trying to get palliative care involved earlier in the course of patients with heart failure, and some would argue any patient with NYHA class III CHF should have a palliative care referral. Palliative care can not only provide symptom management, but also assist with advanced care planning, goals of care in the case of a significant decline, and making sure the patient has a medical power of attorney and discussed his or her wishes with this individual. Without transplantation, heart failure may not be reversible and cardiac function typically deteriorates with time. The growing number of patients with Stage IV heart failure (intractable symptoms of fatigue, shortness of breath or chest pain at rest despite optimal medical therapy) should be considered for palliative care or hospice, according to American College of Cardiology/American Heart Association guidelines.

4.0 CONCLUSION

In this unit, you have learned what heart attack, cardiac arrest, stroke and chronic heart failure is and all their clinical features and management. You should at this point be able to define each condition and explain its management.

5.0 SUMMARY

This unit has focused on the definition of heart attack, cardiac arrest, stroke and chronic heart attack. You should also be able to refer to further readings on the unit.

6.0 TUTOR-MARKED ASSIGNMENT

1. Define the term heart attack and cardiac arrest. What is/are the differences?
2. Cerebrovascular accident has two major broad categories. Name them and discuss any one of your choice.
3. Discuss fully the management and treatment of chronic heart failure
4. Give guidelines on how to avoid stroke.

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UNIT 3 HEMORRHAGE

CONTENTS

1.0 INTRODUCTION

This unit will help you acquire basic understanding of hemorrhage and how to apply pressure in bleeding control points. Before we do this, an overview of the objectives below will serve as a guide.

2.0 OBJECTIVES

At the end of this unit, you should be able to:

- define the term hemorrhage
- describe the major three types of hemorrhage
- identify bleeding control points
- management of hemorrhage.

3.0 MAIN CONTENT

3.1 Definition of Hemorrhage

Hemorrhage is the escape of blood from the circulatory system. Bleeding, technically known as haemorrhaging or haemorrhaging , is blood escaping from the circulatory system. Bleeding can occur internally, where blood leaks from blood vessels inside the body, or externally, either through a natural opening such as the mouth, nose, ear, urethra, vagina or anus, or through a break in the skin. Hypovolemia is a

massive decrease in blood volume, and death by excessive loss of blood is referred to as **exsaguination**.

3.1.1 Types of Hemorrhage

Arterial hemorrhage: This is evident as bright red blood coming from the wound which spurts and can be profuse. Arteries most commonly involved are those in the heart, brain, kidney, small intestine and lower limbs.

Venous Hemorrhage: This is a steady flow of dark, brick-red blood, it may also be profuse.

Capillary Hemorrhage: This is the general oozing of blood which is usually not serious and is usually controlled.

3.1.2 Classification

Hemorrhaging is broken down into four classes by the American College of Surgeons' advanced trauma life support (ATLS).

- **Class I hemorrhage** involves up to 15% of blood volume. There is typically no change in vital signs and fluid resuscitation is not usually necessary.
- **Class II hemorrhage** involves 15-30% of total blood volume. A patient is often tachycardic (rapid heart -beat) with a narrowing of the difference between the systolic and diastolic blood pressures. The body attempts to compensate with peripheral vasoconstriction. Skin may start to look pale and be cool to the touch. The patient may exhibit slight changes in behavior. Volume resuscitation with crystalloids (Saline solution or Lactated Ringer's solution) is all that is typically required. Blood transfusion is not typically required.
- **Class III hemorrhage** involves loss of 30-40% of circulating blood volume. The patient's blood pressure drops, the heart rate increases, peripheral hypoperfusion (shock), such as capillary refill worsens, and the mental status worsens. Fluid resuscitation with crystalloid and blood transfusion are usually necessary.
- **Class IV hemorrhage** involves loss of >40% of circulating blood volume. The limit of the body's compensation is reached and aggressive resuscitation is required to prevent death.

  This system is basically the same as used in the staging of hypovolemic shock. Individuals in excellent physical and cardiovascular shape may have more effective compensatory mechanisms before experiencing cardiovascular collapse. These

patients may look deceptively stable, with minimal derangements in vital signs, while having poor peripheral perfusion. Elderly patients or those with chronic medical conditions may have less tolerance to blood loss, less ability to compensate, and may take medications such as betablockers that can potentially blunt the cardiovascular response. Care must be taken in the assessment of these patients.

3.1.3 World Health Organisation

The World Health Organisation made a standardised grading scale to measure the severity of bleeding:

Grade 0	no bleeding
Grade 1	petechial bleeding
Grade 2	mild blood loss (clinically significant)
Grade 3	gross blood loss, requires transfusion (severe)
Grade 4	debilitating blood loss, retinal or cerebral associated with fatality.

Origin

- Mouth
  - Hematemesis – vomiting fresh blood
  - Hemoptysis – coughing up blood from the lungs
- Anus
  - Hematochezia – rectal blood
- Urinary tract
  - Hematuria – blood in the urine from urinary bleeding
- Upper head
  - Intracranial hemorrhage – bleeding in the skull.
  - Cerebral hemorrhage – a type of intracranial hemorrhage, bleeding within the brain tissue itself.
  - Intracerebral hemorrhage – bleeding in the brain caused by the rupture of a blood vessel within the head. See also hemorrhagic stroke.
  - Subarachnoid hemorrhage (SAH) implies the presence of blood within the subarachnoid space from some pathologic process. The common medical use of the term SAH refers to the non-traumatic types of hemorrhages, usually from rupture of a berry aneurysm or arteriovenous malformation (AVM). The scope of this article is limited to these nontraumatic hemorrhages.
- Lungs
  - Pulmonary hemorrhage
- Gynecologic

  - Vaginal bleeding
    - Postpartum hemorrhage
    - Breakthrough bleeding
  - Ovarian *bleeding*. This is a potentially catastrophic and not so rare complication among lean patients with polycystic ovary syndrome undergoing transvaginal oocyte retrieval.
- Gastrointestinal
  - Upper gastrointestinal bleed
  - Lower gastrointestinal bleed
  - Occult gastrointestinal bleed
  -

3.1.4 Causes

Bleeding arises due to a traumatic injury, underlying medical condition, or a combination.

Traumatic injury
Traumatic bleeding is caused by some type of injury. There are different types of wounds which may cause traumatic bleeding. These include:

- Abrasion - Also called a graze, this is caused by transverse action of a foreign object against the skin, and usually does not penetrate below the epidermis
- Excoriation - In common with Abrasion, this is caused by mechanical destruction of the skin, although it usually has an underlying medical cause
- Hematoma - Caused by damage to a blood vessel that in turn causes blood to collect under the skin.
- Laceration - Irregular wound caused by blunt impact to soft tissue overlying hard tissue or tearing such as in childbirth. In some instances, this can also be used to describe an incision.
- Incision - A cut into a body tissue or organ, such as by a scalpel, made during surgery.
- Puncture wound - Caused by an object that penetrated the skin and underlying layers, such as a nail, needle or knife
- Contusion - Also known as a bruise, this is a blunt trauma damaging tissue under the
- Crushing Injuries - Caused by a great or extreme amount of force applied over a period of time. The extent of a crushing injury may not immediately present itself.
- Ballistic trauma - Caused by a projectile weapon such as a firearm. This may include two external wounds (entry and exit) and a contiguous wound between the two

The pattern of injury, evaluation and treatment will vary with the mechanism of the injury. Blunt trauma causes injury via a shock effect; delivering energy over an area. Wounds are often not straight and unbroken skin may hide significant injury surface of the skin. Penetrating trauma follows the course of the injurious device. As the energy is applied in a more focused fashion, it requires less energy to cause significant injury. Any body organ, including bone and brain, can be injured and bleed. Bleeding may not be readily apparent; internal organs such as the liver, kidney and spleen may bleed into the abdominal cavity. The only apparent signs may come with blood loss. Bleeding from a bodily orifice, such as the rectum, nose, or ears may signal internal bleeding, but cannot be relied upon. Bleeding from a medical procedure also falls into this category.

Medical condition

'Medical bleeding' denotes hemorrhage as a result of an underlying medical condition (i.e. causes of bleeding that are not directly due to trauma). Blood can escape from blood vessels as a result of three basic patterns of injury:

Intravascular changes - changes of the blood within vessels (e.g. ↑ blood pressure, ↓ clotting factors)

- Intramural changes - changes arising within the walls of blood vessels (e.g. aneurysms, dissections, AVMs, vasculitides)
- Extravascular changes - changes arising outside blood vessels (e.g. H pylori infection, brain abscess, brain tumor)

The underlying scientific basis for blood clotting and hemostasis is discussed in detail in the articles, Coagulation, hemostasis and related articles. The discussion here is limited to the common practical aspects of blood clot formation which manifest as bleeding. Certain medical conditions can also make patients susceptible to bleeding. These are conditions that affect the normal "hemostatic" functions of the body. Hemostasis involves several components. The main components of the hemostatic system include platelets and the coagulation system.
Platelets are small blood components that form a plug in the blood vessel wall that stops bleeding. Platelets also produce a variety of substances that stimulate the production of a blood clot.

One of the most common causes of increased bleeding risk is exposure to non-steroidal anti-inflammatory drugs (or "NSAIDs"). The prototype for these drugs is aspirin, which inhibits the production of thromboxane. NSAIDs inhibit the activation of platelets, and thereby increase the risk of bleeding. The effect of aspirin is irreversible; therefore, the inhibitory effect of aspirin is present until the platelets have been replaced (about

ten days). Other NSAIDs, such as "ibuprofen" (Motrin) and related drugs, are reversible and therefore, the effect on platelets is not as long-lived.

There are several named coagulation factors that interact in a complex way to form blood clots, as discussed in the article on coagulation. Deficiencies of coagulation factors are associated with clinical bleeding. For instance, deficiency of Factor VIII causes classic Hemophilia A while deficiencies of Factor IX cause "Christmas disease"(hemophilia B). Antibodies to Factor VIII can also inactivate the Factor VII and precipitate bleeding that is very difficult to control. This is a rare condition that is most likely to occur in older patients and in those with autoimmune diseases. von Willebrand disease is another common bleeding disorder. It is caused by a deficiency of or abnormal function of the "von Willebrand" factor, which is involved in platelet activation. Deficiencies in other factors, such as factor XIII or factor VII are occasionally seen, but may not be associated with severe bleeding and are not as commonly diagnosed.

In addition to NSAID-related bleeding, another common cause of bleeding is that related to the medication, warfarin ("Coumadin" and others). This medication needs to be closely monitored as the bleeding risk can be markedly increased by interactions with other medications. Warfarin acts by inhibiting the production of Vitamin K in the gut. Vitamin K is required for the production of the clotting factors, II, VII, IX, and X in the liver. One of the most common causes of warfarin-related bleeding is taking antibiotics. The gut bacteria make vitamin K and are killed by antibiotics. This decreases vitamin K levels and therefore the production of these clotting factors. Deficiencies of platelet function may require platelet transfusion while deficiencies of clotting factors may require transfusion of either fresh frozen plasma or specific clotting factors, such as Factor VIII for patients with hemophilia.

3.1.5 Bleeding Control Points

Temporal artery: This is to control bleeding from the scalp. Common carotid artery: This is for bleeding from the neck and head, but one must be careful not to occlude blood supply to the brain which can cause unconsciousness. Facial artery: This is for bleeding from the front of the face.

Subclavian artery: This is from the armpit and chest wall.
Brachial artery: This is from the arm.
Femoral artery: This is from the leg.

3.1.6 Management of Hemorrhage Pressure Points

Direct manual pressure over the wound with a sterile dressing with clean cloth or handkerchief. This is usually adequate in the control of mild bleeding. Digital pressure can be applied at pressure point. The main artery to the injured extremities which lies near the skin and over the bone is the second avenue of approach in the control of bleeding.

3.1.7 Use of Tourniquet

A tourniquet is device for stopping the flow of blood through an artery, typically by compressing a limb with a cord or tight bandage. The use of tourniquet should only be in case of severe life-threatening hemorrhage. Tourniquet should be applied on the arm, hand or below the elbow, on the leg or below the groin. Tourniquet should consist of a flat band at least 1 inch wide. Do not use a rope or twine as tourniquet. Use neck-tie, handkerchief, towel, scarf or belt. Place a pad over the artery and rope several layers of clothes around the extremities for protection of the soft tissue. Rope the tourniquet around the extremities applying a hall knot. Place a stick or a rod or a stick and complete the knot. The tourniquet must be tight enough to shut off arterial blood flow.

Disadvantages of tourniquet

Compression inefficiency to stop arterial blood flow allows the blood to continue into the extremities, but block the venous return from the extremities causing increased venous bleeding and inducing greater hemorrhage. Tourniquets are unstable. It may be applied too tightly, thus causing cuts on the skin or injury to the nerve or muscles tissue. It is safe to leave tourniquet for 2-3 hours or until the patient is in the emergency care room or with the physician.

4.0 CONCLUSION

This unit has exposed you to know what hemorrhage is, and you can also state the types of hemorrhage and identify the bleeding control points. You should at this point be able to discuss hemorrhage, and classify it.

5.0 SUMMARY

This unit has defined hemorrhage as blood escaping (bleeding) from the circulatory system. The classification has been mentioned; as well the pressure points to control hemorrhage and management have been discussed.

6.0 TUTOR-MARKED ASSIGNMENT

1. Explain in brief, the types of hemorrhage.
2. Mention five important uses of tourniquet in the control of hemorrhage.
3 Define hemorrhage and concisely discuss the causes of hemorrhage.

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UNIT 4 BEHAVIOURAL AND PSYCHIATRIC EMERGENCY

CONTENTS

1.0 INTRODUCTION

The emergency department and the EMS system are now the most common points of entry by disturbed persons into the mental health system. There are several reasons for this. First, over the last years there has been a trend to deinstitutionalize the mentally ill. These patients, formally confined to mental hos[pitals, now receive care through community centers and other facilities. Second, substance abuse, a major problem in our society, is a frequent cause of behavioural and psychiatric crises. These patients are not generally enrolled in the mental health system. They enter the health care system through EMS, either seeking help or because of a drug or alcohol-related accident or illness.

2.0 OBJECTIVES

At the end of this unit, you should be able to:

- define the term behavioural emergency
- discuss the role of drugs and alcohol in behavioural emergencies.
- list physical problems that can be manifestated as psychiatric problems.

3.0 MAIN CONTENT

3.1 Understanding Behavioural Emergencies

A behavioural emergency is an intrapsychic, environmental, situational, or organic alteration that results in behaviour that cannot be tolerated by the patient or other members of society. It usually requires immediate attention.

Intrapsychic causes
Intrapsychic causes of altered behaviour arise from problems within the person. Such behaviour usually results from an acute stage of an underlying psychiatric condition. A wide range of behaviour can be manifested. These include;

(a) Depression
(b) Withdrawal
(c) Catatonia
(d) Violence
(e) Suicidal acts
(f) Homicidal acts
(g) Paranoid reactions
(h) Phobias
(i) Hysterical conversion
(j) Disorientation and disorganisation.

In the field, behavioural emergencies resulting from intrapsychic causes are less common that those resulting from other causes, such as alcohol or drug abuse.

Interpersonal/environmental causes
Interpersonal and environmental causes of behavioural emergencies result from reactions to stimuli outside the person. They often result from overwhelming and stressful incidents, such as the death of a loved one, rape, or a disaster. The change in behaviour can frequently be linked to a specific incident or series of incidents. The range of behaviour manifested is broad, and a patient's specific symptoms often relate to the type of incident that precipitated them.

3.2 Organic Causes

An organic cause of altered behaviour results from a disturbance in the patient's physical or biochemical state. Such disturbances include drugs, alcohol, trauma, illness, and dementia. The area of the brain affected by the disturbance determines the type of behaviour change;

(a) **Alcohol abuse**. Substance abuse is the pathological use of a substance to thepoint that it significantly interferes with a person's normal activities. Alcohol abuse, a common problem, often complicates an underlying medical or behavioural condition. Alcohol is a CNS depressant, and alcohol abuse should be suspected in any patient who has a breath odor of ethanol, slurred speech, or unsteady gait. Also, suspect alcohol in a person who is slow to respond to questions. Evidence or recent alcohol consumption often appears in the form of empty cans or bottles or reports from friends or bystanders.

(b) **Drug abuse**. Drug abuse can result from the frequent use of either street or prescription drugs. Because of the wide variety of drugs abused today and the wide variety of clinical symptoms, the drug abuser is often much more difficult to evaluate than the alcohol abuser. Assessment of the patient suspected of drug abuse should include routine examination of abuse, such as prescription bottless, drug paraphernalia , or needle tracks. The behaviour of the substance-abuse patient can include withdrawal, suicidal or homicidal actions, violent behaviour, or hysteria.

(c) **Trauma**. Trauma can also result in altered behaviour. Causative factors include increased intracranial pressure, decreased circulation to the brain, or hypoxia resulting from poor perfusion.

(d) **Medical illnesses**. Medical illnesses can hjave behavioural manifestations too. The diabetic may exhibit confusion, slurred speech, and unsteady gait, particularly with hypoglycemia. Because of this, diabetics are occasionally thought to be drunk. In such cases, medical care may not be summoned until coma ensues. In addition to diabetics, various electrolyte imbalances can result in a behaviour change such as confusion, violence and extreme anxiety.

(e) **Dementia**. Dementia results from actual damage to brain cells. Signs of dementia often include impairment or loss of memory, impaired judgement, and confusion. These are often complicated by poor eyesight and hearing. The onset of dementia is usually slow and gradual. Because dementia occurs most frequently in the elderly., it is often associated with aging. Dementia can result from organic brain syndrome, Alzheimer's dseas , or similar conditions. Alzheimer's disease is a progressive degenerative disease that attacks the brain and results in impaired memory, thinking, and behaviour. It affects approximately 2.5 million American adults and is present in approximately 25 percent of persons age 85 and older.

3.3 Assessment of Behavioural Emergencies

A feeling of uncertainty often exists at the scene of a behavioural emergency, creating great stress for paramedics and other public safety personnel called to the incident. This uncertainty often stems from an initial inability to determine the cause of the crisis. Also, in the past, paramedic education did not cover behavioural problems thoroughly, leaving paramedics feeling unprepared. Even today, there are few protocols to guide prehospital care of behavioural emergencies, since these situations do not lend themselves to a structured approach.

Scene survey
Emergency medical providers may be injured at the scene of behavioural emergencies . Therefore, before leaving the vehicle, you should evaluate the scene for possible danger. Paramedics cannot render aid if they become victims. Unless you are adequately trained, avoid the following;

A patient armed with a weapon
Riot scenes
Fire scenes
Hostage situations
Radioactive sites

Assessment
Perform primary and secondary assessments in addition to gathering information necessary for immediate management of life-threatening conditions. Sources of information should include observation of the patient, statements volunteered by the patient, information gained from interviewing the patient, and information obtained from family members, bystanderss, and first responders. A systematic approach to assessment is critical. The information obtained should include:

Participating situation or problem
Patient’s current life situation
Patient’s recent medical and psychiatric history
Patient’s past medical andpsychiatric history
Patient’s mental status
Patient’s affect and physical signs
Patient’s behaviour.

From the assessment you should draw conclusions about the possible cause of the behavioural change.

Interviewing techniques

The interview is the most important part of assessing the behavioural emergency patient. The interview should be organised and logical. Use of a normal checklist is not practical, however. Only short interview's scope. Gather only information that is critical to prehospital management and transportation of the patient, unless the patient volunteers more. The interview should be open-ended, although both direct and indirect questions may be asked. Allow the patient to take the lead in the interview, unless you are afraid that essential information will be lost or suspect that the patient is depressed, minimally responsive, or suicidal. If the patient is relunctant to answer certain questions, do not press, as the patient may withdraw completel;y and provide no information. Be prepared to spend whatever time is required to obtain information. Exceptions include the patient whose physical condition requires immediate transport or the patient who may endanger himself, herself, or others. Above all do not make judgements about the patient's behaviour or answers.

The following guidelines will make your interviews more effective:

(a) Remove the patient from the crisis situation, and exclude the disturbing person or objects.
(b) Communicate self-confidence as well as honesty, firmness, and a reasonable attitude about issues important to the patient and the situation.
(c) It is necessary either to agree or disagree if the patient distorts reality, simply understand that these distortions are real for the patient
(d) Encourage the patient to sit down and relax.
(e) Encourage the patient to speak in his or her own words, and appear interested in his or her statements.
(f) Interrupt the patient as little as possible, unless you must redirect a distorganised, rambling communication.
(g) Do not be afraid of long silent periods. Remain relaxed and attentive.
(h) If the begins to cry or laugh, do not interrupt the display of emotion by talking.
(i) Encourage the patient to relate his or her story. Nod your head, and say things such as "I see, tell me more"
(j) If the patient views the situation as chaotic or if the patient's thoughts are disorganozed, use the interview to build a sense of structure.
(k) Do not argue with the patient.

(l) Do not shout at the patient.

3.4 General Management And Intervention Techniques

The paramedic's attitude is the single most important factor in dealing with the disturbed patient.Communicate warmth, sensitivity, and composition. The patient must take you seriously. Intervene only to the extent that you feel competent, and be aware of your own professional limitations.

The following are general guidelines for managing behavioural emergencies:

(a) Before intervening, assess the risk to your own safety
(b) Give first priority to life-threatening injures
(c) Take command of the situation
(d) Assign bystanders to perform some tasks when appropriate
(e) Accept the patient's feelings. Do not tell the patient how to feel
(f) Display a clean, reassuring attitude to relax the patient
(g) Avoid severe anxiety reactions in family members, friends, and bystanders by good management. Have appropriate authorities remove unnecessary persons from the scene.
(h) Have familliar persons provide support to the patient as necessary
(i) To avoid heightening the patient's anxiety, develop some rapport with the patient before carrying out a physical examination. Maintain privacy, professionalism, and efficiency.
(j) If the patient is anxious or confused, explain all procedures carefully.

3.5 Specific Psychiatric Disorders

The psychiatric and behavioural emergencies that paramedics most often encounter fall into several categories. These include;

Depressive disorders
Suicide attempts
Anxiety disorders
Manic disorders
Schizophrenic disorders
Paranoid disorders
Disorders of age (delirium and dementia)
Substance abuse or dependence

Depression
Depression is a common psychiatric disorder. It affects over 20 percent of the population and accounts for the majority of psychiatric referrals. Depression is a mood disorder characterized by feelings of helplessness and hopelessness. Typically, the patient loses interest and pleasure in his or her usual activities. Depressed patients cry easily. They exhibit behavioural and physical changes, such as appetite increases or decreases, weight loss or gain, insomania, low energy level and malaise, feelings of worthlessness or inappropriate guilt, and, in many cases, recurrent thoughts of death or suicide. The patient's home medications may give you additional insight into the patient's problem. Antidepressant medications include amitriptyline(Elavil), imipramine(Tofranil), Phenelzine(Nardil), buproprion(Wellbutrin), and fluoxetine(Prozac).

Management
Depressed patients should receive supportive care. Encourage them to talk, delicately raising questions about suicidal thoughts. Remember that depression often has an organic cause, such as organic brain syndrome, hypothyroidism, or chronic corticosteroid usage.

Suicide
Suicide is a frequent cause of death in the United States and Africa and other continents.
It is quite prevalent in the youths and above.

4.0 CONCLUSION

In this unit, you have learned what Intrapsychic causes,interpersonal/environmental, organic causes of behavioural emergencies are. You have also learn about assessment and management of them. You can now read with understanding. And can explain what behavioural emergencies are and can answer questions correctly.

5.0 SUMMARY

Paramedics do not see society at its best. Although, there is a strong behavioural component to every emergency, many calls are purely behavioural in nature. Most behavioural emergencies encountered in the field require little more than reassurance of the patient. However, violent or suicidal patients require aggressive therapy.

6.0 TUTOR-MARKED ASSIGNMENT

1. Define the term behavioural emergency.
2. Discuss the role of drugs and alcohol in behavioural emergencies.

3. List physical problems that can be manifested as psychiatric problems.

7.0 REFERENCES FURTHER READING

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MODULE 5

UNIT 1 HEADSINJURY

CONTENTS

1.0 INTRODUCTION

Injury involving the head is a critical one which should not be ignored. Having gone through the course guide, you should have acquired an overview of what constitutes a head injury; this unit will further help you know about head injury as indicated in the objectives below.

2.0 OBJECTIVES

At the end of this unit, you should be able to:

- define the term head injury
- describe the types/classification of head injuries
- discuss the pathological/clinical features, mechanism
- explain the causes of head injury
- state the management/treatment of head injury.
-

3.0 MAIN CONTENT

3.1 Definition of Head Injury

A head injury is any trauma to the scalp, skull, or brain. The injury may be only a minor bump on the skull or a serious brain injury. It can either be closed or open (penetrating).

Traumatic brain injury (**TBI**), also known as **intracranial injury**, occurs when an external force traumatically injures the brain. TBI can be classified based on severity, mechanism (closed or penetrating head injury), or other features (e.g., occurring in a specific location or over a widespread area). *Head injury* usually refers to TBI but is a broader category because it can involve damage to structures other than the brain, such as the scalp and skull.TBI is a major cause of death and disability worldwide, especially in children and young adults. Males sustain traumatic brain injuries more frequently than do females. Causes include falls, vehicle accidents, and violence. Prevention measures include use of technology to protect those suffering from automobile accidents, such as seat belts and sports or motorcycle helmets, as well as efforts to reduce the number of automobile accidents, such as safety education programs and enforcement of traffic laws.

Brain trauma can occur a consequence of a focal impact upon the head, by a sudden acceleration/deceleration within the cranium or by a complex combination of both movement and sudden impact. In addition to the damage caused nowby injury, brain trauma causes *secondary injury*, a variety of events that take place in the minutes and days following the injury. These processes, which include alterations in cerebral blood flow and the pressure within the skull, contribute substantially to the damage from the initial injury.TBI can cause a host of physical, cognitive, social, emotional, and behavioral effects, and outcome can range from complete recovery to permanent disability or death. The 20th century saw critical developments in diagnosis and treatment that decreased death rates and improved outcome. Some of the current imaging techniques used for diagnosis and treatment include CT scans computed tomography and MRIs magnetic resonance imaging. Depending on the injury, treatment required may be minimal or may include interventions such as medications, emergency surgery or surgery years later. Physical therapy, speech therapy, recreation therapy, occupational therapy and vision therapy may be employed for rehabilitation.

3.1.1 Classification/Types of Head Injuries

Traumatic brain injury is defined as damage to the brain resulting from external mechanical force, such as rapid acceleration or deceleration, impact, blast waves, or penetration by a projectile. Brain function is temporarily, or permanently impaired and structural damage may or may not be detectable with current technology.TBI is one of two subsets of acquired brain injury (brain damage that occur after birth); the other subset is non-traumatic brain injury, which does not involve external mechanical force (examples include stroke and infection). All traumatic brain injuries are head injuries, but the latter term may also refer to injury to other parts of the head. However, the terms *head injury* and *brain injury* are often used interchangeably. Similarly, brain injuries fall under the classification of central nervous system injuries and neurotrauma. In neuropsychology research literature, in general the term "traumatic brain injury" is used to refer to non-penetrating traumatic brain injuries.TBI is usually classified based on severity, anatomical features of the injury, and the mechanism (the causative forces). Mechanism-related classification divides TBI into closed and penetrating head injury. A closed (also called non-penetrating, or blunt) injury occurs when the brain is not exposed. A penetrating, or open, head injury occurs when an object pierces the skull and breaches the dura mater, the outermost membrane surrounding the brain.

Severity

Severity of traumatic brain injury [

	GCS	PTA	LOC
Mild	13–15	<1 day	0–30 minutes
Moderate	9–12	>1 to <7 days	>30 min to <24 hours
Severe	3–8	>7 days	>24 hours

Brain injuries can be classified into mild, moderate, and severe categories. TheGlasgow Coma Scale (GCS), the most commonly used system for classifying TBI severity, grades a person's level of consciousness on a scale of 3–15 based on verbal, motor, and eye-opening reactions to stimuli. In general, it is agreed that a TBI with a GCS of 13 or above is mild, 9–12 is moderate, and 8 or below is severe.

Similar systems exist for young children. However, the GCS grading system has limited ability to predict outcomes. Because of this, other classification systems such as the one shown in the table are also used to help determine severity. A current model developed by the Department of Defense and Department of Veterans Affairs uses all three criteria of GCS after resuscitation, duration of post-traumatic amnesia (PTA), and loss of consciousness (LOC). It also has been proposed to use changes that are visible on neuroimaging, such as swelling, focal lesions, or diffuse injury as method of classification. Grading scales also exist to classify the severity of mild TBI, commonly called concussion; these use duration of LOC, PTA, and other concussion symptoms

Types of head injuries
Open wound head injury: This means that the skull has been fractured and the durable matter is lacerated. Closed wound head injury: This occurs when there is severe impact to the head area without obvious wound being sustained or laceration occurring. It is important to note that the brain dies when the blood supply to it is interrupted for a few minutes andthere is no regeneration of damaged neurons.Fractured skull: The fracture itself is not as important as the fearof brain injury and as such patients are treated as neurologicalconditions.

3.1.2Pathological Features

Systems also exist to classify TBI by its pathological features. Lesions can be extra-axial, (occurring within the skull but outside of the brain) or intra-axial (occurring within the brain tissue). Damage from TBI can be focal or diffuse, confined to specific areas or distributed in a more general manner, respectively. However, it is common for both types of injury to exist in a given case.Diffuse injury manifests with little apparent damage in neuroimaging studies, but lesions can be seen with microscopy techniques post-mortem, and in the early 2000s, researchers discovered that diffusion tensor imaging (DTI), a way of processing MRI images that shows white matter tracts, was an effective tool for displaying the extent of diffuse axonal injury. Types of injuries considered diffuse include edema (swelling) and diffuse axonal injury, which is widespread damage to axons including white matter tracts and projections to the cortex. Types of injuries considered diffuse include concussion and diffuse axonal injury, widespread damage to axons in areas including white matter and the cerebral hemispheres.

Focal injuries often produce area. Research shows that the most common areas to have focal lesions in non-penetrating traumatic brain injury are the orbitofrontal cortex (the lower surface of the frontal lobes)

and the anterior temporal lobes, areas that are involved in social behavior, emotion regulation, olfaction, and decision-making, hence the common social/emotional and judgment deficits following moderate-severe TBI. Symptoms such as hemiparesis or aphasia can also occur when less commonly affected areas such as motor or language areas are, respectively, damaged.One type of focal injury, cerebral laceration, occurs when the tissue is cut or torn. Such tearing is common in orbitofrontal cortex in particular, because of bony protrusions on the interior skull ridge above the eyes. In a similar injury, cerebral contusion (bruising of brain tissue), blood is mixed among tissue.

In contrast, intracranial hemorrhage involves bleeding that is not mixed with tissue.Hematomas, also focal lesions, are collections of blood in or around the brain that can result from hemorrhage. Intracerebral hemorrhage, with bleeding in the brain tissue itself, is an intra-axial lesion. Extra-axial lesions include epidural hematoma, subdural hematoma, subarachnoidhemorrhage, and intraventricular hemorrhage. Epidural hematoma involves bleeding into the area between the skull and the dura mater, the outermost of the three membranes surrounding the brain. In subdural hematoma, bleeding occurs between the dura and the arachnoid mater. Subarachnoid hemorrhage involves bleeding into the space between the arachnoid membrane and the pia mater. Intraventricular hemorrhage occurs when there is bleeding in the ventricles

3.1.3 Signs and Symptoms

Symptoms are dependent on the type of TBI (diffuse or focal) and the part of the brain that is affected. Unconsciousness tends to last longer for people with injuries on the left side of the brain than for those with injuries on the right. Symptoms are also dependent on the injury's severity. With mild TBI, the patient may remain conscious or may lose consciousness for a few seconds or minutes. Other symptoms of mild TBI include headache, vomiting, nausea, lack of motor coordination, dizziness, difficulty balancing, lightheadedness, blurred vision or tired eyes, ringing in the ears, bad taste in the mouth, fatigue or lethargy, and changes in sleep patterns. Cognitive and emotional symptoms include behavioral or mood changes, confusion, and trouble with memory, concentration, attention, or thinking. Mild TBI symptoms may also be present in moderate and severe injuries.

A person with a moderate or severe TBI may have a headache that does not go away, repeated vomiting or nausea, convulsions, an inability to awaken, dilation of one or both pupils, slurred speech, aphasia (word-

finding difficulties), dysarthria (muscle weakness that causes disordered speech), weakness or numbness in the limbs, loss of coordination, confusion, restlessness, or agitation. Common long-term symptoms of moderate to severe TBI are changes in appropriate social behavior, deficits in social judgment, and cognitive changes, especially problems with sustained attention, processing speed, and executive functioning. Alexithymia, a deficiency in identifying, understanding, processing, and describing emotions occurs in 60.9% of individuals with TBI. Cognitive and social deficits have long-term consequences for the daily lives of people with moderate to severe TBI but can be improved with appropriate rehabilitation.When the pressure within the skull (intracranial pressure, abbreviated ICP) rises too high, it can be deadly.[44] Signs of increased ICP include decreasing level of consciousness, paralysis or weakness on one side of the body, and a blown pupil, one that fails to constrict in response to light or is slow to do so.

Cushing's triad, a slow heart rate with high blood pressure and respiratory depression is a classic manifestation of significantly raised ICP. Anisocoria, unequal pupil size, is another sign of serious TBI. Abnormal posturing, a characteristic positioning of the limbs caused by severe diffuse injury or high ICP, is an ominous sign. Small children with moderate to severe TBI may have some of these symptoms but have difficulty communicating them. Other signs seen in young children include persistent crying, inability to be consoled, listlessness, refusal to nurse or eat, and irritability.

Clinical features

Persistent pain,swelling in the region of fracture, bleeding from nose, ear, and pharynx and below the conjunctiva, this suggests fracture of base of the skull. There may be escape of cerebrospinal fluid from the ear which is regarded as otorrhea and from the nose as rhinorrhea. Bloody cerebrospinal fluid can also suggest brain laceration or confusion.

3.1.4 Causes

The most common causes of TBI in the U.S. include violence, transportation accidents, construction, and sports. Motor bikes are major causes, increasing in significance in developing countries as other causes reduce. The estimates that between 1.6 and 3.8 million traumatic brain injuries each year are a result of sports and recreation activities in the US. In children aged two to four, falls are the most common cause of TBI, while in older children traffic accidents compete with falls for this position. TBI is the third most common injury to result from child abuse. Abuse causes 19% of cases of pediatric brain trauma, and the death rate

is higher among these cases. Although men are twice as likely to have a TBI. Domestic violence is another cause of TBI, as are work-related and industrial accidents. Firearms and blast injuries from explosions are other causes of TBI, which is the leading cause of death and disability in war zones. According to Representative Bill Pascrell (Democrat, NJ), TBI is "the signature injury of the wars in Iraq and Afghanistan." There is a promising technology called activation database-guided EEG biofeedback, which has been documented to return a TBI's auditory memory ability to above the control group's performance.

3.1.5 Mechanism

The type, direction, intensity, and duration of forces all contribute to the characteristics and severity TBI. Forces that may contribute to TBI include angular, rotational, shear, and translational forces.Even in the absence of an impact, significant acceleration or deceleration of the head can cause TBI; however, in most cases a combination of impact and acceleration is probably to blame. Forces involving the head striking or being struck by something, termed *contact* or *impact loading*, are the cause of most focal injuries, and movement of the brain within the skull, termed *noncontact* or *inertial loading*, usually causes diffuse injuries. The violent shaking of an infant that causes shaken baby syndrome commonly manifests as diffuse injury. In impact loading, the force sends shock waves through the skull and brain, resulting in tissue damage. Shock waves caused by penetrating injuries can also destroy tissue along the path of a projectile, compounding the damage caused by the missile itself.Damage may occur directly under the site of impact, or it may occur on the side opposite the impact (coup and contrecoup injury, respectively). When a moving object impacts the stationary head, coup injuries are typical, while contrecoup injuries are usually produced when the moving head strikes a stationary object.[

3.1.6 Primary and Secondary Injury

A large percentage of the people killed by brain trauma do not die right away but rather days to weeks after the event; rather than improving after being hospitalized, some 40% of TBI patients deteriorate. Primary brain injury (the damage that occurs at the moment of trauma when tissues and blood vessels are stretched, compressed, and torn) is not adequate to explain this deterioration; rather, it is caused by secondary injury, a complex set of cellular processes and biochemical cascades that occur in the minutes to days following the trauma. These secondary processes can dramatically worsen the damage caused by primary injury and account for the greatest number of TBI deaths occurring in hospitals.Secondary injury events include damage to the blood–brain

barrier, release of factors that cause inflammation, free radical overload, excessive release of the neurotransmitterglutamate (excitotoxicity), influx of calcium and sodium ions into neurons, and dysfunction of mitochondria. Injured axons in the brain's white matter may separate from their cell bodies as a result of secondary injury, potentially killing those neurons.

Other factors in secondary injury are changes in the blood flow to the brain; ischemia (insufficient blood flow); cerebral hypoxia (insufficient oxygen in the brain); cerebral edema (swelling of the brain); and raised intracranial pressure (the pressure within the skull). Intracranial pressure may rise due to swelling or a mass effect from a lesion, such as a hemorrhage. As a result, cerebral perfusion pressure (the pressure of blood flow in the brain) is reduced; ischemia results. When the pressure within the skull rises too high, it can cause brain death or herniation, in which parts of the brain are squeezed by structures in the skull. A particularly weak part of the skull that is vulnerable to damage causing extradural hematoma is the pterion, deep in which lies the middle meningeal artery, which is easily damaged in fractures of the pterion. Since the pterion is so weak, this type of injury can easily occur and can be secondary due to trauma to other parts of the skull where the impact forces spread to the pterion.

3.1.7 Treatment / Management

It is important to begin emergency treatment within the so-called "golden hour" following the injury. People with moderate to severe injuries are likely to receive treatment in an intensive care unit followed by a neurosurgical ward. Treatment depends on the recovery stage of the patient. In the acute stage the primary aim of the medical personnel is to stabilize the patient and focus on preventing further injury because little can be done to reverse the initial damage caused by trauma. Rehabilitation is the main treatment for the subacute and chronic stages of recovery. International clinical guidelines have been proposed with the aim of guiding decisions in TBI treatment, as defined by an authoritative examination of current evidence.

Acute stage

Certain facilities are equipped to handle TBI better than others; initial measures include transporting patients to an appropriate treatment center. Both during transport and in hospital the primary concerns are ensuring proper oxygen supply, maintaining adequate blood flow to the brain, and controlling raised intracranial pressure (ICP), since high ICP deprives the brain of badly needed blood flow and can cause deadly brain herniation. Other methods to prevent damage include management

of other injuries and prevention of seizures.Neuroimaging is helpful but not flawless in detecting raised ICP. A more accurate way to measure ICP is to place a catheter into a ventricle of the brain, which has the added benefit of allowing cerebrospinal fluid to drain, releasing pressure in the skull. Treatment of raised ICP may be as simple as tilting the patient's bed and straightening the head to promote blood flow through the veins of the neck.

Sedatives, analgesics and paralytic agents are often used. Hypertonic saline can improve ICP by reducing the amount of cerebral water (swelling), though it is used with caution to avoid electrolyte imbalances or heart failure. Mannitol, an osmoticdiuretic, appears to be equally effective at reducing ICP. Some concerns; however, have been raised regarding some of the studies performed. Diuretics, drugs that increase urine output to reduce excessive fluid in the system, may be used to treat high intracranial pressures, but may cause hypovolemia (insufficient blood volume).Hyperventilation (larger and/or faster breaths) reduces carbon dioxide levels and causes blood vessels to constrict; this decreases blood flow to the brain and reduces ICP, but it potentially causes ischemia and is, therefore, used only in the short term. Administration of corticosteroids is associated with an increased risk of death, and so it is recommended that they not be given routinely.

Endotracheal intubation and mechanical ventilation may be used to ensure proper oxygen supply and provide a secure airway. Hypotension (low blood pressure), which has a devastating outcome in TBI, can be prevented by giving intravenous fluids to maintain a normal blood pressure. Failing to maintain blood pressure can result in inadequate blood flow to the brain. Blood pressure may be kept at an artificially high level under controlled conditions by infusion of norepinephrine or similar drugs; this helps maintain cerebral perfusion. Body temperature is carefully regulated because increased temperature raises the brain's metabolic needs, potentially depriving it of nutrients. Seizures are common. While they can be treated with benzodiazepines, these drugs are used carefully because they can depress breathing and lower blood pressure. TBI patients are more susceptible to side effects and may react adversely or be inordinately sensitive to some pharmacological agents. During treatment monitoring continues for signs of deterioration such as a decreasing level of consciousness

Traumatic brain injury may cause a range of serious coincidental complications that include cardiac arrhythmias and neurogenic pulmonary edema. These conditions must be adequately treated and stabilised as part of the core care for these patients. Surgery can be performed on mass lesions or to eliminate objects that have penetrated the brain. Mass lesions such as contusions or hematomas causing a

significant mass effect (shift of intracranial structures) are considered emergencies and are removed surgically.For intracranial hematomas, the collected blood may be removed using suction or forceps or it may be floated off with water. Surgeons look for hemorrhaging blood vessels and seek to control bleeding. In penetrating brain injury, damaged tissue is surgically debrided, and craniotomy may be needed. Craniotomy, in which part of the skull is removed, may be needed to remove pieces of fractured skull or objects embedded in the brain. Decompressive (DC) is performed routinely in the very short period following TBI during operations to treat hematomas; part of the skull is removed temporarily (primary DC). DC performed hours or days after TBI in order to control high intracranial pressures (secondary DC) has not been shown to improve outcome in some trials and may be associated with severe side-effects.

Chronic stage

Physical therapy will commonly include muscle strength exercise. Once medically stable, patients may be transferred to a subacute rehabilitation unit of the medical center or to an independent rehabilitation hospital. Rehabilitation aims to improve independent function at home and in society and to help adapt to disabilities and has demonstrated its general effectiveness, when conducted by a team of health professionals who specialise in head trauma. As for any patient with neurologic deficits, a multidisciplinary approach is key to optimising outcome. Physiatrists or neurologists are likely to be the key medical staff involved, but depending on the patient, doctors of other medical specialties may also be helpful. Allied health professions such as physiotherapy, speech and language therapy, cognitive rehabilitation therapy, and occupational therapy will be essential to assess function and design the rehabilitation activities for each patient. Treatment of neuropsychiatric symptoms such as emotional distress and clinical depression may involve mental health professionals such as therapists, psychologists, and psychiatrists, while neuropsychologists can help to evaluate and manage cognitive deficits.

After discharge from the inpatient rehabilitation treatment unit, care may be given on an outpatient basis. Community-based rehabilitation will be required for a high proportion of patients, including vocational rehabilitation; this supportive employment matches job demands to the worker's abilities. People with TBI who cannot live independently or with family may require care in supported living facilities such as group homes. Respite care, including day centers and leisure facilities for the disabled, offers time off for caregivers, and activities for people with TBI.Pharmacological treatment can help to manage psychiatric or behavioralproblems.Medication is also used to control post-traumatic epilepsy; however the preventive use of anti-epileptics is not recommended. In those cases where the person is bedridden due to a

reduction of consciousness, has to remain in a wheelchair because of mobility problems, or has any other problem heavily impacting self-caring capacities, caregiving and nursing are critical. The most effective research documented intervention approach is the activation database guided EEG biofeedback approach, which has shown significant improvements in memory abilities of the TBI subject that are far superior than traditional approaches (strategies, computers, medication intervention). Gains of 2.61 standard deviations have been documented. The TBI's auditory memory ability was superior to the control group after the treatment.

4.0 CONCLUSION

In this unit, you have learned what head injury is and the types and classification of head injuries that may occur. You should be able to define what head injury isand its treatment/ management.

5.0 SUMMARY

This unit has focused on the definition of head injury, its clinicalfeatures and necessary cares/treatment. This understanding will enableyou to take immediate care should you encounter such situation.

6.0 TUTOR-MARKED ASSIGNMENT

1. Define the term head injury.
2. Enumerate the clinical features of head injury.
3.Discuss the causes and pathological features of head injury.

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UNIT 2 FRACTURE

CONTENTS

1.0 INTRODUCTION

Having acquired a general overview of this unit, you will be helped further to know what is fracture and its components. The objectives below will also enable you acquire basic understanding of fracture.

2.0 OBJECTIVES

At the end of this unit, you should be able to:

- define the term fracture
- describe the various types of fractures
- list the clinical features of fracture
- enumerate fracture management.

3.0 MAIN CONTENT

3.1 Definition of Fracture

This is a break in the continuity of bone and is usually caused by injury either deliberately or accidentally. Apart from the broken bones, other structures may be affected resulting in oedema in soft tissue, haemorrhage into the muscles and joints, rupture of tendons, injured nerves and joint dislocation. A bone fracture can be the result of high force impact or stress or a minimal trauma injury as a result of certain medical conditions that weaken the bones such as Osteoporosis, Bone cancer,orOsteogenesis imperfect, where the fracture is then properly termed pathologic fracture .

3.1.1 Types of Fractures

Complete fracture: This involves a break across the entire cross section of the bone and is frequently displaced.

Incomplete fracture: In this type, break occurs only through a part of the cross section of the bone and is usually not displaced.

Open fracture: An open fracture is any fracture with an associatedopen wound. The wound communicates with the fracture site and provides a route for infectious materials to enter. Injury may occur when a penetrating object, such as bullet, enters the body and then fractures the bone. It may also result when the bone breaks and pushes through the skin.

Closed fracture: This break does not communicate with the outside area. The closed fracture does not have an associated open wound. The prognosis for closed wound fracture is generally better than for the open fracture.

Hairline and impacted fractures: Two types of fractures that remain in-line and stay relatively stable are the hairline and the impacted fractures. The hairline fracture is a small crack in the bone that does not cause the bone ends to displace , The impacted fracture occurs when the ends of the broken bone compress together, providing the fracture with some stability. In either fracture, the only evidence of a problem may be the mechanism of injury and pain. The patient may use the limb normally. But it is noticeably weak and may, with stress, collapse post-accident. The only way to rule out such an injury is with an X-ray in the emergency department.

Green stick fracture: The Greenstick Fracture is a partial break, disrupting only one side of the long bone. It most frequently affects pediatric patients because their bone structure is less mature and more flexible than that of adults. While incomplete as a fracture, it is significant because of the partial nature of the break. During the natural repair , the injured side experiences growth , while the other does not. The probable result will be a bone that increases angulation as it heals. Occasionally, the emergency department staff will break the bone completely to ensure proper repair.

Fractures in the geriatric patient: Skeletal injuries for the other end of the age spectrum, the geriatric patient, are quite different. The strength of the skeletal system diminishes with advancing age. Bones may degenerate to a point where they fracture easily or spontaneously. The patient frequently describes, feeling a snap, before falling. The injury process is relatively attraumatic. Thus, the patient feels less

uncomfortable than someone suffering an equivalent break because of trauma.

Comminuted fracture: Here the bones fractured are more than two; a bone can be broken in more than two places.

Depressed fracture: This is bone driven and is frequent in fracture of the skull of facial bone.

Pathologic fracture: This is a fracture occurring through an area of diseased bones, e.g. tumor, the bone or osteoporosis in the elderly.

3.1.2 Specific Musculoskeletal Considerations

Lower extremities: The lower extremities may suffer a number of types of fractures or dislocations

(a) Fracture of spine and pelvis

This condition is relatively common in the elderly, degenerative changes cause narrowing of intervertebral discs and osteophytes may develop round the margins of joints of the vertebral column, and cervical region (cervical spondylosis). Pelvic fractures most commonly occur across the iliac crests or through the pelvic ring. Either fracture requires a great deal of force, although, the pelvic ring fracture is generally much more severe. The injury usually affects two sites because of the circular anatomy of the pelvis. The ring fracture usually involves the vasculature that runs along its interior and then in the lower extremities. Pelvic fractures frequently cause severe internal haemorrhage often in excess of two (2) litres . They may also result in the loss of circulation to one or both lower extremities. They may cause damage to the nervous system varying from compression of individual spinal nerves to spinal cord injury.

(b) Hip dislocation: The hip joint may dislocate in two ways. The anterior dislocation displaces the femoral head anteriorly. The patient's foot and knee rotate laterally. There may be a noticeable prominence in the inguinal area reflecting the head of the dislocatedfemur. The posterior dislocation displaces the head of the femur into the buttocks. The patient will generally flex the knee and rotate the leg and foot internally.

(c)Femur fractures: Fractures of the femur in the proximity of the hip may be difficult to differentiate from the anterior dislocation. While you may expect the broken leg to be slightly shorter than the unbroken one, the difference may be slight and unnoticeable if the legs are straight and parallel. Fractures involving the femur, either midshaft or otherwise,

may affect the surrounding vasculature or significant soft-tissue. The bleeding that follows can be extensive. Fractures of the femur, thepelvis, and to a limited degree, the humerus can involve significant blood loss, possibly contributing to, or including, hypovolemia.

(d)Knee dislocation/fracture: Knee dislocation will normally present with the angle and firmly fixed in a position. The dislocation carries a high incidence of vascular and nervous injury because of the proximity of blood vessels and nerves to the injury site. Dislocation can also occur in combination with the fracture, since the energy needed to cause one is commonly sufficient to cause the other. Dislocations of the patella are more common than those of the knee. But to the untrained examiner, it may be difficult to distinguish one dislocation from the other. Patellar dislocations carry a lower incidence of vascular injury than do knee dislocations. Treat any fracture within three inches of a joint, especially the knee, the elbow, or the ankle, similar to the treatment for a dislocation. Immobilise the joint as found.

(e) Lower-leg fractures: Fractures of the lower-leg bones, the tibia and the fibula can occur separately or together. Direct trauma suffered during an auto or athletic accident frequently causes these injuries. The tibia will most commonly fracture, since it is the anterior bone and is responsible for weight bearing. If the fibula is intact, the extremity may not angulate, but it will not be able to bear weight. If only the fibula is broken, the limb may be rather stable.

(f) Foot/anklefractures: Foot/ankle fractures most often result from crush injuries or structural fatigue. The injuries are reasonably stable, even though, the extremity cannot bear weight. With any injury to this area, be concerned about the status of the distal circulation and nervous status.

3.1.3 Upper Extremities

A number of sites in the upper extremities seem prone to fractures or dislocations.

(a) Shoulder dislocation; Shoulder dislocation may occur anteriorly, posteriorly, or interiorly. The posterior dislocation presents with a hollow shoulder. The limb angled forward, the elbow internally rotated, and arm away from the chest. The anterior displacement presents with a prominent shoulder and with the arm close to the chest as well as forward of the anterior axillary line. The inferior displacement locks the patient’s extremity above the head.

(b) Humerus fracture: Fracture may occur along the entire length of the humerus. Here the injury is particularly hard to stabilize because of the structure and mobility of the shoulder joint. The axillary artery runs through the axilla, making it difficult to any mechanical traction to the limb. Humerus fracture also presents a potential hazard for circulatory compromise.

(c) Elbow fracture: Fractures in and around the elbow are particularly dangerous, especially in children. The soft-tissue depth is minimal, and the skeletal diameter is large. Any fracture has a good probability of blood vessel or nerve involvement. Such damage may result in the eventual loss of function distal to the injury.

(d) Forearm fracture: The forearm may fracture anywhere along its length and may involve the radius, ulna, or both. Commonly fractures will occur at the distal end of the radius, breaking it just above the articular surface known as Colle's Fracture. It may present with the wrist turned up at an unusual angle. As with most joint fractures, the major concern is for the distal circulation and innervations.

(e) Hand and wrist fractures: Hand and wrist fractures are commonly associated with direct trauma. They present with very noticeable deformity and significant pain. These fractures are of serious concern to the patient. Since the hand and wrist bones are small, any fracture is in the close proximity of a joint. Exercise concern for vascular and nervous involvement.

Effects of fractures and dislocations: Fractures and dislocations affect living tissue that requires a constant and rich supply of oxygenated blood. While the central nervous system, the heart, and the kidneys will dysfunction more rapidly and more acutely than the skeletal system, the long bones may degenerate time and leave the limb unable to carry out its intended function. Musculoskeletal injuries are of relatively low priority in the seriously injured patient. Yet their proper care is important to the patient's overall well -being and recovery.

Clinical Features in summary
Pain which is continuous and increases in severity
until the fractured bone is immobilised
Loss of function
Localised swelling
Discoloration of the skin
Deformity
Tenderness
Penetration of fractured bone in open wound.

3.1.4 Management/ Treatment

Treatment of bone fractures are broadly classified as surgical or conservative, the latter basically referring to any non-surgical procedure, such as pain management, immobilisation or other non-surgical stabilisation. A similar classification is *open* versus *closed treatment*, in which *open treatment* refers to any treatment in which the fracture site is surgically opened, regardless of whether the fracture itself is an open or closed fracture.

Pain management
In arm fractures in children, ibuprofen has been found to be as effective as a combination of acetaminophen and codeine.

Immobilisation

Since bone healing is a natural process which will most often occur, fracture treatment aims to ensure the best possible *function* of the injured part after healing. Bone fractures are typically treated by restoring the fractured pieces of bone to their natural positions (if necessary), and maintaining those positions while the bone heals. Often, aligning the bone, called reduction, in good position and verifying the improved alignment with an X-ray is all that is needed. This process is extremely painful without anesthesia, about as painful as breaking the bone itself. To this end, a fractured limb is usually immobilised with a plaster or fiberglasscast or splint which holds the bones in position and immobilizes the joints above and below the fracture. When the initial post-fracture edema or swelling goes down, the fracture may be placed in a removable brace or orthosis. If being treated with surgery, surgical nails, screws, plates and wires are used to hold the fractured bone together more directly. Alternatively, fractured bones may be treated by the Ilizarov method which is a form of external fixator.

Occasionally smaller bones, such as phalanges of the toes and fingers, may be treated without the cast, by buddy wrapping them, which serves a similar function to making a cast. By allowing only limited movement, fixation helps preserve anatomical alignment while enabling callus formation, towards the target of achieving union.Splinting results in the same outcome as casting in children who have a distal radius fracture with little shifting.

Surgery
Surgical methods of treating fractures have their own risks and benefits, but usually surgery is done only if conservative treatment has failed, is very likely to fail, or likely to result in a poor functional outcome. With some fractures such as hip fractures (usually caused by osteoporosis),

surgery is offered routinely because non-operative treatment results in prolonged immobilization, which commonly results in complications including chest infections, pressure sores, deconditioning, deep vein thrombosis (DVT) and pulmonary embolism, which are more dangerous than surgery. When a joint surface is damaged by a fracture, surgery is also commonly recommended to make an accurate anatomical reduction and restore the smoothness of the joint. Infection is especially dangerous in bones, due to the recrudescent nature of bone infections. Bone tissue is predominantly extracellular matrix, rather than living cells, and the few blood vessels needed to support this low metabolism are only able to bring a limited number of immune cells to an injury to fight infection. For this reason, open fractures and osteotomies call for very careful antiseptic procedures and prophylactic antibiotics.Occasionally bone grafting is used to treat a fracture. Sometimes bones are reinforced with metal.

These implants must be designed and installed with care. *Stress shielding* occurs when plates or screws carry too large of a portion of the bone's load, causing atrophy. This problem is reduced, but not eliminated, by the use of low-modulus materials, including titanium and its alloys. The heat generated by the friction of installing hardware can easily accumulate and damage bone tissue, reducing the strength of the connections. If dissimilar metals are installed in contact with one another (i.e., a titanium plate with cobalt-chromium alloy or stainless steel screws), galvanic corrosion will result. The metal ions produced can damage the bone locally and may cause systemic effects as well. Electrical bone growth stimulation or osteo-stimulation has been attempted to speed or improve bone healing. Results however do not support its effectiveness.

Management in summary
Check for bleeding, paralysis and other conditions. Mobilise people around to help in splinting.Place the injured limb in as natural a position as possible beforepadding and splinting. Do not apply traction when a broken wound is protruding above the skin. To control bleeding, apply pressure gently by applying a steriledressing to cover the site and rope an elastic bandage. Apply the splint properly before attempting to move fractured victim. Give analgesic to relieve pain. Broken neck patients should be moved in support to neck. Do not attempt to align an open fracture immediately.

4.0 CONCLUSION

In this unit, you have learnt that fracture is a crack or break in the continuity of bone and its features. You should be able at this point to describe fracture.

5.0 SUMMARY

This unit has focused on the definition of fracture, its types, musculoskeletal considerations, and management/treatment. Lower and upper extremities fractures.

6.0 TUTOR-MARKED ASSIGNMENT

1. Define the term fracture.
2a. Mention all types of fractures
b. State the clinical features of fracture
c. Enumerate five management plans for fracture.
3 Discuss in your words what you understand by specific musculoskeletal considerations using lower extremities as examples
4 Discuss pain management in fracture.

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UNIT 3 PATHOGENESIS OF INFECTIOUS DISEASES

CONTENTS

1.0 INTRODUCTION

Infectious diseases are illnesses caused by infection or infestation of the human body by various biological agents. These diseases account for the majprity of known human and animal diseases. Although most infectious diseases seldom prove fatal, you must learn to recognize infectious diseases in the prehospital setting so that you can initiate emergency treatment and take steps to protect emergency personnel. Also, two infectious diseases-acquired immune deficiency syndrome(AIDS) and hepatitis B-present serious hazards to health care workers. The spread of these two diseases has made safety procedures even more important in recent years.This unit addresses the fundamental principles of infecttious diseases, including the operation of the immune system. It then examines the various infectious diseases most frequently encountered in the field.

2.0 OBJECTIVES

At the end of this unit, you should be able to:

- define the following terms; bacteria, antibiotics, virus, fungi, antigen
- define toxin, and give an example of endotoxins and exotoxins
- describe the difference between bacterial and viral infections
- briefly discuss the body's immune system.

3.0 MAIN CONTENT

3.1 Pathogenesis of Infectious Disease

Several biological agents can cause human infection. These include bacteria, viruses, fungi, and parasites. Since treatment and management of the different infections vary significantly, you need to recognise the causative agents of the more commonly encountered infectious diseases.

3.2Bacteria

Bacteria is a small, unicellular organisms that live throughout the environment-frequently cause infection. Smaller than human red blood cells, they can only be viewed through the microscope. Bacteria can live without the aid of another organism. They cause many of the common infections in medicine, including middle ear infections in children, many cases of tonsilitis, and meningitis. Most bacterial infections respond to treatment with drugs called antibiotics. Once administered, antibiotics kill or inhibit the growth of invading bacteria by one of several mechanisms.Bacteria can usually be cultured and identified readily in most hospital laboratories. Many bacteria are categorised according to their appearance under the microscope after staining with several dyes referred to as a Gram stain. Some bacteria stain blue, while others stain red. Bacteria that stain blue are referred to as Gram positive bacteria. They are somewhat similar to each other in their composition. Bacteria that stain red are referred to as Gram negative bacteria. They are also somewhat similar to each other in their structure.

Simple infection is not the only consequence of bacterial infection. Many bacteria release poisonous chemicals, or toxins. Their are two general categories of toxins; extoxins and endotoxins. Exotoxins are released from living bacteria during infection. They travel throughout the body via the blood or lymph, ultimately causing problems. The life-threatening consequences of tetanus infection are an example of the effects of an exotoxin. Tetanus is caused by the bacterium Clostridium tetani. The actual infection by the bacteria is mild and may be limited, for example, to a puncture wound in the foot. Yet, on entering the body, the bacteria release their toxin, called tetanospasmin. This toxin then travels through the blood to the skeletal muscles, causing the spastic rigidity classically seen in tetanus.Other bacteria release toxins referred to as exotoxins. Endotoxins are usually released upon the death and destruction of the bacterial cell. Septic shock, for example , is caused by the release of an endotoxin from several Gram-negative bacteria.

3.3 Viruses

Most infections are caused by biological agents called viruses. Viruses are much smaller than bacteria and can only be seen with an electron microscope. In addition, they cannot grow without the assistance of another organism. In fact, viruses are referred to as intracellular parasites, since they must invade the cells of the organism they infect. Once inside a cell, they take over, using the various cellular enzymes to replicate and produce more viruses. Viruses cannot reproduce outside of the host cell, and, unlike bacteria, they are very difficult to treat. Once a virus infects a cell, it can only be killed by destroying the infected cell. Drugs have not yet been developed that can selectively destroy cells infected by viruses, while simultaneously leaving uninfected cells unharmed. This partially explains the dilemma facing researchers trying to find a cure for AIDS. Fortunately, most viral illnesses are mild and fairly self-limited. Even so, at present, they usually cannot be treated with more than symptomatic care.

Fungi

Another biological agent that can cause human infection are thye fungi(the plural of fungus). More like plants than animals, they include yeasts and molds. Fungi rarely cause human disease other than minor skin infections, such as athlete's foot and some of the more common vaginal infections. Fungi infections are commonly called mycoses. Patients with an impaired immune system suffer fungal infection more commonly than healthy people. In such patients, the fungi can invade the lungs, blood, and several organs. Treatment of complicated, deep fungal infections proves difficult even in the hospital setting.

Parasites

Parasites can also cause infection. They range in size from small, unicellular organisms (not much lager than bacteria) to large intestinal worms. Parasites tend to be more common in developing nations than in the Unitd States for example. Treatment depends upon the organism and the location.

3.4 Immunity

The body has a very sophiscated system for fighting disease called the immune system. Most viruses and bacteria have proteins on their surface called antigens. The human immune system detects these antigens as being foreign and responds to suppress or kill them. Our white blood cells, or leukocytes, are primarily responsible for fighting the infection.

Immune response

There are two general parts to the immune response. One is called cell-mediated immunity and tthe other humoral immunity. Cell-mediated immunity is derived from special leukocytes called T lymphocytes. They originate in the thymus, a gland located in the upper part of the chest. T cells are primarily responsible for fighting infections of biological agents living in certain body cells. Examples of such infections include tuberculosis, many viral infections, and most fungal infections. In these cases, white cells move to the site of infection. Here they attack and attempt to eliminate the invading pathogen. In contrast, humoral immunity derives from B lymphocytes and results in the formation of special proteins called antibodies. There are five classes of human antibodies. They include:

(a) IgM. The antibody that responds immediately
(b) IgM. The antibody that has memory and recognizes a repeatedly invading infection
(c) IgA. The antibody present in the mucous membranes
(d) IgE. The antibody contributing to allergic and anaphylactic responses
(e) IgD. The antibody present in the lowest concentration.

As you known antibodies are produced in response to antigens, or proteins that usually appear on the surface of the invading organism. Typically, the antigen is specific for the particular type of invading pathogen. Following exposure to the antigen, the immune system will release antibodies that seek out the invading antigennn. These antibodies combine with the antigen, forming what is commonly called the antigen-antibody complex. This large complex is subsequently removed by scanvenger cells such as macrphages.

The lymphatic system

The lymphatic system acts as a separate circulatory system. It helps transport materials between the tissues and the blood. As water, solutes, and inactivated or dead infectious agents filter out of the capillaries, they enter the interstitial fluid. The fluid, known as lymph, becomes part of the lymphatic system. As the lymph circulates through the system, it enters nodes that filter the fluid and produce additional antibodies. The nodes thus help curb bodily infection. A key organ in the lymphatic system is the spleen. This solid organ lies in the left upper quadrant of the abdomen. It filters red blood cells and participate in the formation of cells that manufacture antibodies. The spleen is quite vulnerable to abdominal trauma. Blood loss from the spleen can be massive and rapidly fatal.

3.5 Transmission of Infectious Disease

Any of the agents causing infectious disease are transmissible. Infectious diseases can be spread in two ways. Direct transmission occurs when the disease is passed from one person to another through contact with infected blood or other body fluids. In addition, it occurs through the transmission of air-borne particles. Indirect transmission occurs without direct person-to-person contact. Instead, the infectious agent is passed indirectly from one person to another through some contamonated object . For example , blood or another body fluid may be left on an object such as an ambulance stretcher. Later,a second person can contact the disease through contact with the contaminated stretcher.

Routes of disease transmission

There are several routes of ex[posure for infectious diseases. Blood borne diseases are those transmitted by contact with the blood or body fluids of an infected person. Blood borne diseases include AIDS, hepatitis B, hepatitis C, hepatitis D, and syphilis. Other infectious diseases are transmitted through the air on droplets expelled during a productive cough or sneeze. Examples of airborne disease include tuberculosis, meningitis, mumps, measles, rubella, and chicken pox(varicella). In addition, infections can be transmitted through food, also called the faecal-oral route. Foodborne diseases include food poisoning, salmonella, staphylococcus infections, hepatitis A, and hepatitis E.

Infection control in prehospital care

There are four phases of infection control in prehospital care . These include;

(a) Preparation for response to emergency incidents
(b) Response to emergency incidents
(c) Actions at emergency incidents
(d) Recovery from emergency incidents

Emergency personnel should have Personal Protective Equipment(PPE) available at all times.

Preparation for response to emergency incidents

Infection control begins long before an emergency call. To ensure proper protection of patients and personnel from exposure to infectious diseases, each EMS service should complete the following precautions prior to responding to an emergency incident.

(a) Establish and maintain written Standard Operating Procedures (SOPs) and assure that all personnel adhere to these procedures.
(b) Provide adequate infection control training to all personnel
(c) Ensure that proper personal protective equipment is provided and stored appropriately. Have all the equioment checked regularly and maintained properly

(d) Ensure that all emergency personnel treat and bandage all personal wounds(open sores, cuts, skin breaks, etc) prior to the emergency response
(e) Assure that all emergency personnel have a high level of personal hygiene.
(f) Do not allow emergency personnel to deliver patient care if they have a communicable disease such as hepatitis B or influenza
(g) Assure that all emergency personnel obtain appropriate vaccination to protect them from contracting communicable diseases.
(h) Do not allow emergency personnel to eat, drink, or have open food in any part of the ambulance.

3.6 Response to Emergency Incidents

While en route to an emergency incident, all prehospital personnel should begin preparation for immediate patient care. This can be facilitated by the following;

(a) Isolate all body substances. Do not come into contact with any of them.
(b) Wear appropriate personal protective equipment.
(c) Allow only necessary personnel to make patient contact. Limit the risk to as few people as possible, thus minimising exposure.
(d) Use airway adjuncts such as a pocket mask or bag-valve-mask unit to minimise exposures.
(e) Properly dispose of biohasardous waste.
(f) Use extreme caution with sharp instruments. Do not bend, recap, or remove needles. Dispose of all contaminated sharps in puncture-resistant, properly labeled containers.
(g) Allow no smoking, eating, or drinking at the scene.
(h) Do not apply costmetics or lip balm or handle contact lenses where a reasonable likelihood of exposure exists.
(i) Wash hands immediately after patient contact. On-scene, hands can be washed with a waterless, hand-cleansing solution. Upon returning to qurters or at the hospital, thorough hand washing with soap and water must be carried out.

Recovery from emergency incidents

Infection control does not end at the scene. Upon arrival at the hospital, or upon return to qurters, infection control procedures continue. The following actions are necessary;

(a) Dispose of all biohazardous wastes in accordance with local laws and regulations.
(b) Transport infectious wastes in leak-proof containers. Bag any soiled linen, and label for laundry personnel

(c) Decontaminate all contaminated clothing and reusable equipment.

Decontamination methods and procedures

All emergency equipment should be decontaminatedfollowing protocols and standard operating procedures established by the EMS service or department. Take equipment to a designated decontamination area, which should be a properly marked, secured, separate room. Ensure that the room has a proper ventilation system and adequate drainage. Be sure to wear gloves, gowns, boots, protective eye-wear, and a facemask during decontamination. Begin decontamination cleaning with soap and water to remove surface dirt and debris. Then , carry out disinfection, and if required, sterilization. Vatious leves of decontamination exist. These include low-level disinfection, Intermediate-level disinfection, high-level disinfection and sterilisation.

4.0 CONCLUSION

In this unit, you been taught what infectious diseases are, including pathogenesis,bacteria, virus , fungi and parasites. Now, you should be able discuss what infection is , you can as well describe toxins, endotoxins and exotoxins.

5.0 SUMMARY

Prehospital personnel generally will not be directly involved in the treatment of infectious disease. Nevertheless, you should suspect infectious disease and take proper precautions. Appropriate protective devices should be immediatel available for every paramedic in your unit, These devices include disposable gloves, eye protection, face masks, and gowns or aprons. Amouth shield or mouth-to-mask ventilation device should also be readily available. Moreover, because the spectrum of infectious disease is changing rapidly, you must keep up to date with the latest recommendation on prevention of infectious disease. The key to infectious disease management is prevention.

6.0 TUTOR-MARKED ASSGNMENT

(a) Define the following terms; bacteria, antibiotics, virus, fungi and antigen.

(b) Define toxin, and give an example of endotoxin and exotoxin.

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UNIT 4 EMERGENCY RESPIRATORY CONDITION

CONTENTS

1.0 INTRODUCTION

Respiratory conditions are of great importance in understanding and dealing with health emergencies. In this unit, you will acquire the basic understanding of respiratory obstructions, asphyxia and cardiopulmonaryresuscitation.

2.0 OBJECTIVES

At the end of this unit, you should be able to:

- define respiratory obstruction
- explain asphyxia and cardio-pulmonary resuscitation.

3.0 MAIN CONTENT

3.1 Respiratory Conditions

Respiration or breathing is a process by which oxygen passes from theair into the lungs while carbon dioxide as waste product is expelled.Respiration involves the production of energy, typically with the intakeof oxygen and the release of carbon dioxide from the oxidation ofcomplex organic substances. This gaseous exchange takes place in thelungs; the air we breathe consists of 20 % oxygen while the air webreathe out consists of 16 % oxygen.This accounts for the

effectiveness of the expired air methods ofartificial resuscitation. When respiration is impaired, there is lack ofoxygen to the lungs and if this continues without relief, death can occur.

3.1.1 Airway (Respiratory) Obstruction

This is a blockage of respiration in the airway. Blockage of the airway is an immediate threat to the patient's life and a true emergency. Upper airway obstruction is an interference with air movement through the upper airway. This interference can come from the tongue, foreign bodies, vomitus, blood, teeth, or something else.Airway obstruction may be either, partial, or complete. Partial obstruction allows for either adequate or poor air exchange. With adequate air exchange, the patient can cough effectively. Patient's suffering poor air exchange can no longer generate an effective cough.

They often emit a high- pitched noise while inhaling and may have increased breathing difficulty and cyanosis. Complete obstruction is suspected when airflow is not felt from the nose and mouth, or when the patient cannot speak, breath, or cough. A patient with complete airway obstruction will quickly become unconscious, and death will occur if the breathing obstruction is not relieved. In the absence of breathing, complete airway obstruction can be recognised by the difficulty encountered when trying to ventilate the patient.This airway obstruction is further divided into two major areas namely;Upper and lower airway obstructions**.**

Upper airway obstruction
Causes of upper airway obstruction, foreign body aspiration, blunt laryngotracheal trauma, penetrating laryngotracheal trauma, tonsillar hypertrophy, paralysis of the vocal cord or vocal fold, acute laryngotrachitis such as viral croup, bacterial tracheitis, epiglottitis, peritonsillar abscess, pertussis, retropharyngeal abscess, spasmodic croup.

Lower airway obstruction
Lower airway obstruction is mainly caused by increased resistance in the bronchioles (usually from a decreased radius of the bronchioles) that reduces the amount of air inhaled in each breath and the oxygen that reaches the pulmonary arteries. It is different from airway restriction (which prevents air from diffusing into the pulmonary arteries because of some kind of blockage in the lungs). Diseases that cause lower airway obstruction are termed obstructive lung diseases.

Summary of the causes of respiratory obstructions

Foreign body blocking the airway occurs when there is e.g. vomitus, mucus, trinkets, food and water that enters the trachea instead of the oesophagus also swollen vocal cords,tongue blocking the airway when it falls backward into the pharynx all occur with an unconscious patient.

3.1.2 Basic Airway Management

The first step in the primary assessment is to determine if the patient has a patent airway. If there is history of trauma, this must be done in conjunction with appropriate cervical spine stabilisation. Any airway compromise must be rapidly corrected. Initially, manual airwaymaneuvers, either, with or without the use of basic mechanical airways should be applied to provide immediate ventilation and oxygenation. Shortly thereafter, advanced airway maneuvers such as endotracheal intubation, should be carried out to maintain the airway effectively. Since airway maneuvers carry a high likelihood of contact with the patient's body fluids, wear protective gear including gloves and eyewear.

Manual airway maneuvers
Cardiopulmonary resuscitation, commonly known as CPR, is an emergency procedure performed in an effort to manually preserve intact brain function until further measures are taken to restore spontaneous blood circulation and breathing in a person who is in cardiac arrest. It is indicated in those who are unresponsive with no breathing or abnormal breathing, for example, atonal respiration**.**

3.3.1 Methods of CPR /Management

In 2010, the American Heart AssociationandInternational Liaison Committee on Resuscitation updated their CPR guidelines. The importance of high quality CPR (sufficient rate and depth without excessively ventilating) was emphasized. The order of interventions was changed for all age groups except newborns from airway, breathing, chest compressions (ABC) to chest compressions, airway, breathing (CAB). An exception to this recommendation is for those believed to be in a respiratory arrest (drowning, etc.). The most important aspect of CPR is: few interruptions of chest compressions, a sufficient speed and depth of compressions, completely relaxing pressure between compressions, and not ventilating too much. It is unclear if a few minutes of CPR before defibrillation results in different outcomes than immediate defibrillation.

A universal compression to ventilation ratio of 30:2 is recommended by the AHA. With children, if at least 2 trained rescuers are present a ratio of 15:2 is preferred. In newborns a rate of 3:1 is recommended unless a

cardiac cause is known in which case a 15:2 ratio is reasonable. If an advanced airway such as an endotracheal tube or laryngeal mask airway is in place, artificial ventilation should occur without pauses in compressions at a rate of 8–10 per minute. The Standard recommended order of interventions is chest compressions, airway, breathing or CAB in most situations, with a compression rate of at least 100 per minute in all groups. Recommended compression depth in adults and children is at least 5 cm (2 inches) and in infants it is 4 centimetres (1.6 in).

As of 2010 the Resuscitation Council (UK) still recommends ABC for children. As it can be difficult to determine the presence or absence pulse the pulse check has been removed for lay providers and should not be performed for more than 10 seconds by healthcare providers. In adults, rescuers should use two hands for the chest compressions, while in children they should use one, and with infants' two fingers (index and middle fingers).

Compression only

These maneuvers are highly effective and require no specialized equipment. The twocommon manual airway maneuvers are the head-tilt/chin-lift and jaw-thrust. If there is a history of trauma, the modified jaw-thrust should be used as this maneuver does not involve tilting the head. HEAD-Tilt/Chin-Lift Maneuver: In the absence of trauma, the preferred technique for opening the airway is the head-tilt/chin-lift maneuver to perform this maneuver;

a. With the patient supine, position yourself at his or side.
b. Place one hand on the patient's forehead and tilt the head back by applying firm downward pressure with your palm.
c. Use your other hand to grasp the chin without applying undue pressure on the jaw. Be particularly careful to keep your fingers on the bony part of the chin. This will avoid compressing the soft tissues underneath, which may cause airway obstruction
d. Lift the jaw anteriorly to open the way.

Jaw-thrust maneuver(non-trauma): the jaw-thrust maneuver is another useful technique for opening the way because it involves some tilting of the head, it should not be used in the trauma patient to perform this maneuver;

(a) With the patient supine, kneel at the top of his or her head.
(b) Place the finger -tips of each hand on the angles of the patient's lower jaw.
(c) Forcefully displace the jaw forward, while gently tilting the patient's head backward.
(d) Retract the patient\s lower lip with your thumbs .

Alternatively, the jaw-lift maneuver can be used. With this maneuver, the jaw can be elevated by placing a gloved hand into the mouth and elevating the mandible anteriorly. This maneuver must be employed with caution as the fingers are placed inside the patient's mouth.Modified Jaw-Thrust Maneuver (Trauma): The jaw-thrust maneuver is modified for patients who possibly have suffered a head or neck injury. The procedure is the same except the head should be firmly supported without tilting it backward or turning it to the side.

Summary of the management of respiratory problems
Determine quickly the cause of obstruction. Make sure that it is removed or relieved at once. Then start immediate resuscitation if breathing has ceased or stopped or is irregular. Do not attempt to remove pieces of food or other objects from the airway with finger unless they are at easy reach, to prevent it being pushed further in. If the patient is a child, turn him down and strike one or more blows on his back, between the shoulder blades, this will dislodge the object. In the case of an adult, lie him face down in a prone position on a table or a bed with the entire body above the waist hanging over the side of the bed or table. If this method fails to dislodge the object, begin mouth-mouth resuscitation with the hope to getting some air pastthe obstruction. Transport victim quickly to the hospital for emergency opening of the trachea. If victim is unconscious, the obstruction could be vomitus or large big mucus.Use suction equipment if available.

3.2 Asphyxia or Asphyxiation

Is a condition of severely deficient supply of oxygen to the body that arises from abnormal breathing, is also a condition caused by lack of oxygen and excess of carbon dioxide inthe lungs, blood and organs of the body? This condition can lead toUnconsciousness or death if not quickly resolved. An example of asphyxia is choking. Asphyxia causes generalized hypoxia, which affects primarily the tissues and organs. There are many circumstances that can induce asphyxia, all of which are characterized by an inability of an individual to acquire sufficient oxygen through breathing for an extended period of time. Asphyxia can cause coma or death.

3.2.1 Circumstances

Situations that can cause asphyxia include but are not limited to: the constriction or obstruction of airways, such as from asthma, laryngospasm, or simple blockage from the presence of foreign materials; from being in environments where oxygen is not readily accessible: such as underwater, in a low oxygen atmosphere, or in a

vacuum; environments where sufficiently oxygenated air is present, but cannot be adequately breathed because of air contamination such as excessive smoke.

Other causes of oxygen deficiency include but are not limited to:

- Acute respiratory distress syndrome
- Carbon monoxide inhalation, such as that from a car exhaust and the smoke's emission from a lighted cigarette: carbon monoxide has a higher affinity than oxygen to the hemoglobin in the blood's red blood corpuscles, bonding with it tenaciously, and, in the process, displacing oxygen and preventing the blood from transporting oxygen around the body
- Contact with certain chemicals, including pulmonary agents (such as phosgene) and blood agents (such as hydrogen cyanide)
- Drowning
- Drug overdose
- Exposure to extreme low pressure or vacuum to the pattern (see space exposure)
- Hanging, specifically suspension or short drop hanging.
- Self-induced hypocapnia by hyperventilation, as in shallow water **or** deep water blackout and the choking game
- Inert gas asphyxiation
- Ondine's curse, central alveolar hypoventilation syndrome, or primary alveolar hypoventilation, a disorder of the autonomic nervous system in which a patient must consciously breathe; although it is often said that persons with this disease will die if they fall asleep, this is not usually the case
- Respiratory diseases
- Sleep apnea
- A seizure which stops breathing activity
- Strangling

More causes of asphyxia include
This is blockage of airway due to foreign body or spasm of the larynx or bronchial muscle as in the case of asthmatic attack. Disease of the lungs in which the air alveoli are been filled by inhaled exudates from pneumonia or water. Inhaled vapour or gas which replaces air, the victims drown in vapour or water. Drowning in which water rushes into the lungs, paralysis of the respiratory system, pneumo-thorax. There is also a condition of congenital or neonatal asphyxia arising from obstruction or paralysis of the airway; in this case the airway fails to expand when the baby is born.

3.2.2 Smothering

Smothering is the mechanical obstruction of the flow of air from the environment into the mouth and/or nostrils, for instance, by covering the mouth and nose with a hand, pillow, or a plastic bag. Smothering can be either partial or complete, where partial indicates that the person being smothered is able to inhale some air, although less than required. In a normal situation, smothering requires at least partial obstruction of both the nasal cavities and the mouth to lead to asphyxia. Smothering with the hands or chest is used in some combat sports to distract the opponent, and create openings for transitions, as the opponent is forced to react to the smothering.

In some cases, when performing certain routines, smothering is combined with simultaneous compressive asphyxia. One example is overlay, in which an adult accidentally rolls over onto an infant during co-sleeping, an accident that often goes unnoticed and is mistakenly thought to be sudden infant death syndrome. Other accidents involving a similar mechanism are cave-ins or when an individual is buried in sand or grain. In homicidal cases, the term burking is often ascribed to a killing method that involves simultaneous smothering and compression of the torso. The term "burking" comes from the method William Burke and William Hare used to kill their victims during the West Port murders. They killed the usually intoxicated victims by sitting on their chests and suffocating them by putting a hand over their nose and mouth, while using the other hand to push the victim's jaw up. The corpses had no visible injuries, and were supplied to medical schools for money.

3.2.3 Compressive Asphyxia

Compressive asphyxia (also called chest compression) is mechanically limiting expansion of the lungs by compressing the torso, hence interfering with breathing. Compressive asphyxia occurs when the chest or abdomen is compressed posteriorly. In accidents, the term traumatic asphyxia or crush asphyxia usually refers to compressive asphyxia resulting from being crushed or pinned under a large weight or force. An example of traumatic asphyxia includes cases where an individual has been using a car-jack to repair a car from below, and is crushed under the weight of the vehicle. Pythons, anacondas, and other constrictor snakes kill through compressive asphyxia. In cases of co-sleeping ("overlay"), the weight of an adult or large child may compress an infant's chest, preventing proper expansion of the chest.

Risk factors include large or obese adults, parental fatigue or impairment (sedation by drugs or alcohol) of the co-sleeping adult and a small shared sleeping space (for example, both adult and infant sharing a

couch). In fatal crowd disasters, compressive asphyxia from being crushed against the crowd causes the large part of the deaths, rather than blunt trauma from trampling. This is what occurred at the Ibrox disaster in 1971, where 66 Rangers fans died; the 1979 The Who concert disaster where 11 died; the Luzhniki disaster in 1982, when 66 FC Spartak Moscow fans died; and at the Hillsborough disaster in 1989, when 96 Liverpool fans were crushed to death in an overcrowded terrace. In confined spaces, people push and lean against each other; evidence from bent steel railings in several fatal crowd accidents have shown horizontal forces over 4500 N (equivalent to a weight of approximately 450 kg, or 1014 lbs). In cases where people have stacked up on each other forming a human pile, estimations have been made of around 380 kg (838 lbs) of compressive weight in the lowest layer.

"Positional" or "restraint" asphyxia is when a person is restrained and left alone prone, such as in a police vehicle, and is unable to reposition himself or herself in order to breathe. The death can be in the vehicle, or following loss of consciousness to be followed by death while in a coma, having presented with anoxic brain damage. The asphyxia can be caused by facial compression, neck compression, or chest compression. This occurs mostly during restraint and handcuffing situations by law enforcement, including psychiatric incidents. The weight of the restraint(s) doing the compression may contribute to what is attributed to positional asphyxia. Therefore, passive deaths following custody restraint that are presumed to be the result of positional asphyxia may actually be examples of asphyxia occurring during the restraint process.
Chest compression is also featured in various grappling combat sports, where it is sometimes called wringing. Such techniques are used either to tire the opponent or as complementary or distractive moves in combination with pinning holds, or sometimes even as submission holds. Examples of chest compression include the knee-on-stomach position; or techniques such as leg scissors (also referred to as body scissors and in budō referred to as *do-jime*;[, "trunk strangle" or "body triangle") where a participant wraps his or her legs around the opponent's midsection and squeezes them together.Pressing is a form of torture or execution that works through asphyxia e.g. Burking**.**

3.2.4 Perinatal asphyxia

Perinatal asphyxia is the medical condition resulting from deprivation of oxygen (hypoxia) to a newborn infant long enough to cause apparent harm. It results most commonly from a drop in maternal blood pressure or interference during delivery with blood flow to the infant's brain. This can occur as a result of inadequate circulation or perfusion, impaired respiratory effort, or inadequate ventilation. There has long been a scientific debate over whether newborn infants with asphyxia should be

resuscitated with 100% oxygen or normal air It has been demonstrated that high concentrations of oxygen lead to generation of oxygen free radicals, which have a role in reperfusion injury after asphyxia. Research by Ola DidrikSaugstad and others led to new international guidelines on newborn resuscitation in 2010, recommending the use of normal air instead of 100% oxygen.

Summary of prevention and management of asphyxia
In unconscious patient, control the tongue byputting it forward and lifting the mandible forward to prevent thetongue from falling back.Put the patient in prone position or semi-prone toensure that no fluid can be collected in the pharynx or beaspirated into the trachea.Suction is to remove mucus, blood or other bacteriawhich has accumulated into the throat.Give artificial respiration in sudden arrest ofrespiration by interference with the vital centers in the brain stemas in electric shock, cerebral concussion and drowning.Tracheotomy is performed if the throat cannot bekept clear.

3.3 Cardio-Pulmonary Resuscitation (CPR)

Compression-only (hands-only or cardiocerebral resuscitation) CPR is a technique that involves chest compressions without artificial respiration. It is recommended as the method of choice for the untrained rescuer or those who are not proficient because it is easier to perform and instructions are easier to give over a phone. In adults with out-of-hospital cardiac arrest, compression-only CPR by the lay public has a higher success rate than standard CPR. The exceptions are cases of drownings, drug overdose and arrest in children. Children who receive compression-only CPR have the same outcomes as those having received no CPR. The method of delivering chest compressions remains the same, as does the rate (at least 100 per minute). It is hoped that the use of compression-only delivery will increase the chances of the lay public delivering CPR. As per the American Heart Association, the beat of the Bee Gees song "Stayin' Alive" provides an ideal rhythm in terms of beats per minute to use for hands-only CPR. One can also hum Queen's "Another One Bites The Dust", which is exactly 100 beats-per-minute and contains a memorable repeating drum pattern For those with non -cardiac arrest and people less than 20 years of age, standard CPR is superior to compression-only CPR.

3.3.2 Pathophysiology

CPR is used on people in cardiac arrest in order to oxygenate the blood and maintain a cardiac output to keep vital organs alive. Blood circulation and oxygenation are required to transport oxygen to the tissues. The physiology of CPR involves generating a pressure gradient

between the arterial and venous vascular beds; CPR achieves this via multiple mechanisms The brain may sustain damage after blood flow has been stopped for about four minutes and irreversible damage after about seven minutes. Typically if blood flow ceases for one to two hours, then body cells die. Therefore, in general CPR is effective only if performed within seven minutes of the stoppage of blood flow. The heart also rapidly loses the ability to maintain a normal rhythm. Low body temperatures, as sometimes seen in near-drownings, prolong the time the brain survives. Following cardiac arrest, effective CPR enables enough oxygen to reach the brain to delay brain stem death, and allows the heart to remain responsive to defibrillation attempts.

3.3.3 Complications

While CPR is a last resort intervention, without which a patient without a pulse will all but certainly die, the physical nature of how CPR is performed does lead to complications that may need to be rectified. Common complications due to CPR are rib fractures, sternal fractures, bleeding in the anterior mediastinum, heart contusion,hemopericardium,upper airway complications, damage to the abdominal viscus - lacerations of the liver and spleen, fat emboli, pulmonary complications - pneumothorax, hemothorax, lung contusions. The most common injuries sustained from CPR are rib fractures, with literature suggesting an incidence between 13% and 97%, and sternal fractures, with an incidence between 1% to 43%. Whilst these iatrogenic injuries can require further intervention (assuming the patient survives the cardiac arrest), only 0.5% of them are life-threatening in their own right.

The type and frequency of injury can be affected by factors such as gender and age. For instance, women have a higher risk of sternal fractures than men, and risk for rib fractures increases significantly with age. Children and infants have a low risk of rib fractures during CPR, with an incidence less than 2%, although, when they do occur, they are usually anterior and multiple. Where CPR is performed in error by a bystander, on a patient not in cardiac arrest, only around 2% suffer injury as a result (although 12% experienced discomfort).

Adjunct devices

While several adjunctive devices are available, none other than defibrillation, as of 2010, have consistently been found to be better than standard CPR for out-of-hospital cardiac arrest. These devices can be split into three broad groups: timing devices' those that assist the rescuer to achieve the correct technique,especially depth and speed of compressions; and those that take over the process completely.

Timing devices
Timing devices can feature a metronome (an item carried by many ambulance crews) in order to assist the rescuer in achieving the correct rate. Some units can also give timing reminders for performing compressions, ventilating and changing operators.

Manual assist devices
Mechanical devices have not been found to have greater benefit than harm and thus are not currently recommended for widespread use.Audible and visual prompting may improve the quality of CPR and prevent the decrease of compression rate and depth that naturally occurs with fatigue, and to address this potential improvement, a number of devices have been developed to help improve CPR technique .These items can be devices to place on top of the chest, with the rescuer's hands going over the device, and a display or audio feedback giving information on depth, force or rate, or in a wearable format such as a glove. Several published evaluations show that these devices can improve the performance of chest compressions.As well as its use during actual CPR on a cardiac arrest victim, which relies on the rescuer carrying the device with them, these devices can also be used as part of training programs to improve basic skills in performing correct chest compressions.

Automatic devices
There are also some automated devices available that take over the chest compressions for the rescuer. These have several advantages: they allow rescuers to focus on performing other interventions; they do not fatigue and begin to perform less effective compressions, ashumans do; and they are able to perform effective compressions in limited-space environments such as air ambulances, where manual compressions are difficult. These devices use either pneumatic (high-pressure gas) or electrical power sources to drive a compressing pad on to the chest of the patient. One such device, known as the LUCAS, was developed at the University Hospital of Lund, is powered by the compressed oxygen supplies already standard in ambulances and hospitals, and has undergone numerous clinical trials, showing a marked improvement in coronary perfusion pressure and return of spontaneous circulation.
In August 2013, a 41-year-old woman living in a town near Melbourne in Australia was treated with the LUCAS device for 53 minutes while a stent was placed in an artery near her heart, clearing a 100% blockage. She was considered to be clinically dead for 40 minutes. She was discharged from the hospital a week later.

Artificial ventilation can be achieved with multiple devices. While manual bag valve mask devices supply oxygen-enriched air through a facial mask (without maintaining an open airway), automatic devices

utilize an oropharyngeal airway (e.g., Bergman or Guedel airways), which ensures airway patency. They also have a nozzle for the rescuer with a protective mask mode, preventing any mouth-to-mouth contact. Another system called the AutoPulse is electrically powered and uses a large band around the patient's chest that contracts rhythmically in order to deliver chest compressions. This is also backed by clinical studies showing increased rates of return of spontaneous circulation.

4.0 CONCLUSION

In this unit, you have learned what Airway obstruction, asphyxia, and Cardiopulmonary resuscitation are, their clinical presentations and management. You should on your own read further on these conditions.

5.0 SUMMARY

This unit has focused on the definition of respiration airway (respiratory) obstruction as one of the most common respiratory emergencies. Asphyxia is lack of oxygen and excess carbon dioxide in the lungs, while cardio-pulmonary resuscitation is the measure taken to restore life or consciousness to a patient.

6.0 TUTOR-MARKED ASSIGNMENT

1. Explain the term cardio-pulmonary resuscitation.
2. Discuss in your in your own words the medical uses/management of CPR.
3. What is Asphyxia? What are the circumstances that can contribute/cause Asphyxia?
4. Define Respiration and explain in detail what you understand by airway obstruction.

7.0 REFERENCES/FURTHER READING

Anderson, R.N. (2001).*Deaths: Leading Causes for 1999*. National Vital Statistics Reports.

"The World Report on Violence and Health." (2002). Geneva: World Health Organisation, *Violence and Injury Prevention.*

MODULE 6

Unit 1 Wound
Unit 2 Diabetes
Unit 3 Peptic Ulcer
Unit 4 Peritonitis

UNIT 1 WOUND

CONTENTS

1.0 INTRODUCTION

The course guide had introduced you to the unit which has helped you acquire understanding of wound and its basic components. The objectives below will also guide you in studying the unit.

2.0 OBJECTIVES

At the end of this unit, you should be able to:

- define the term wound
- classify wound
- list and explain factors influencing the healing of a wound
- define wound suturing and discuss freely complications of wound.

3.0 MAIN CONTENT

3.1 Definition of Wound

It is an injury to living tissue caused by a cut, blow, or other impact, typically one in which the skin is cut or broken. It can also be defined as a type of injury which happens relatively quickly in which skin is torn, cut, or punctured (an open wound), or where blunt force trauma causes a contusion (a closed wound) . In pathology, it specifically refers to a sharp injury which damages the dermis of the skin.

3.2 Clinical Features of Wound

There will be immediate loss of all or parts of the functioning organ. There may behaemorrhage and blood clotting. There will be bacterial contamination leading to sepsis.There will be death of the cells called gangrene.

3.3 Classification of Wound

Incised wound: This is a clean wound cut with a sharp object, e.g. surgical wound during operation.
Contused wound: This occurs through a blunt force. It is characterised by rupture of the small blood vessels; i.e. capillaries, haemorrhage and swelling as well as soft tissue injury.
Lacerated wound: The tissue is torn resulting in a rugged appearance; e.g. wound sustained from glass or barbwire.
Punctured wound: This is a small perforation of the skin usually caused by sharp objects; e.g. bullets, knife stab, stepping on nails, etc.
Cleaned wound: Cleaned uninfected wound be closed by suturing to aid healing.
Septic wound: This contains bacteria and pus which may be extracted from the wound. Some wounds are very small, that they can be adequately taken care of at home by the family but punctured wounds need adequate care and prophylactic immunisation to prevent tetanus.

3.4 Wound Healing

Healing by 1st intention: This occurs in minimal injury or incised clean wound that can be sutured, small amount of connective tissue filled the wound and it heals up within a week.
Healing by 2nd intention: This occurs in cases of wound involving a large tissue area which cannot be sutured, it may also be infected with pus. Healing is delayed until the pus is cleaned out, the connective tissues are laid down and when healing is completed, there is scar formation.

Healing by 3rd intention: This occurs when a previously sutured area breaks down and necessitates re-suturing of the wound. When healing occurs by this method, the scar formation is larger.

3.5 Factors Influencing Healing

Age: Healing of wounds occurs faster in young people than old people.

Nutrition: Adequate nutrition has a preventive effect on wound healing, high protein diet helps in tissue repair and growth, while Vitamin C helps in the formation of collagen which is necessary for wound healing.

Hormonal activity: Certain hormones such as growth, aldosterone, steroids, and sex hormones tend to depress the healing process.

Blood supply: Deficient blood supply and infection has a negative effect on healing. Inadequate blood supply causes inadequate nutrient to the tissue.

3.6 Wound Suturing

Recent clean cut (wound) will heal faster if the edges are brought together so that the cut (wound) stay closed. Suture a deep wound when it is less than 12 hours old and when it is very clean, through the following procedure: Sterilise a suturing needle and a clean threadwash hand properly with clean water set up the requirement using aseptic technique inform the patient of what you want to do clean the wound area with antiseptic solution e.g. methylated spirit give local anesthetic iodine to the area to prevent pain make the first stitch in the middle of the wound and close it tightly make other stitches to close the whole open wound dress the wound with dressing and leave it for 5-7 days before removing the stitches.

3.7 Complications

Bacterial infection of wound can impede the healing process and lead to life-threatening complications. Scientists at Sheffield University have identified a way of using light to rapidly detect the presence of bacteria. They are developing a portable kit in which specially designed molecules emit a light signal when bound to bacteria. Current laboratory-based detection of bacteria can take hours or even days.

Workup
Individuals who have wounds that are not healing should be investigated to find the causes. Many microbiological agents can be responsible for this. The basic workup includes evaluating the wound, its extent and severity. Cultures are usually obtained both from the wound site and blood. X rays are obtained, and a tetanus shot may be administered if there is any doubt about prior vaccination.

Chronic

Non-healing wounds of the diabetic foot are considered one of the most significant complications of diabetes, representing a major worldwide medical, social, and economic burden that greatly affects patient quality of life. Almost 24 million Americans—one in every 12—are diabetic and the disease is causing widespread disability and death at an epidemic pace, according to the Centers for Disease Control and Prevention. Of those with diabetes, 6.5 million are estimated to suffer with chronic or non-healing wounds. Associated with inadequate circulation, poorly functioning veins, and immobility, non-healing wounds occur most frequently in the elderly and in people with diabetes—populations that are sharply rising as the nation ages and chronic diseases increase.

Although diabetes can ravage the body in many ways, non-healing ulcers on the feet and lower legs are common outward manifestations of the disease. Also, diabetics often suffer from nerve damage in their feet and legs, allowing small wounds or irritations to develop without awareness. Given the abnormalities of the microvasculature and other side effects of diabetes, these wounds take a long time to heal and require a specialised treatment approach for proper healing.As many as 25% of diabetic patients will eventually develop foot ulcers, and recurrence within five years is 70%. If not aggressively treated, these wounds can lead to amputations. It is estimated that every 30 seconds a lower limb is amputated somewhere in the world because of a diabetic wound. Amputation often triggers a downward spiral of declining quality of life, frequently leading to disability and death. In fact, only about one third of diabetic amputees will live more than five years, a survival rate equivalent to that of many cancers.Many of these lower extremity amputations can be prevented through an interdisciplinary approach to treatment involving a variety of advanced therapies and techniques, such as debridement, hyperbaric oxygen treatment therapy, dressing selection, special shoes, and patient education. When wounds persist, a specialised approach is required for healing.

4.0 CONCLUSION

In this unit, you must have learnt what wound is, you have also realisedthat wound is classified into six types; the healing process and factor affecting healing. You should at this point be able to master and practice on wound.

5.0 SUMMARY

This unit has focused on critical and common ailments in the society which is wound, its definition and components therein.

6.0 TUTOR-MARKED ASSIGNMENT

1. Define the term wound.
2. Give the six classifications of wound.
3. Discuss in detail, wound complications.

7.0 REFERENCES/FURTHER READING

‘WHO Disease and Injury Country estimates” (http://www.who.int/healthinfo/global_burden_disease/estimates_country/en/index.html: World Health Organisation 2009.
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UNIT 2 METABOLIC EMERGENCY DIABETES

CONTENTS

1.0 INTRODUCTION

This unit discusses metabolic emergency diabetes; it will give you the general overview of diabetes and its management. It will also enable you to understand more about diabetes.

2.0 OBJECTIVES

At the end of this unit, you should be able to:

- define the term diabetes and diabetic coma
- explain the pathophysiology, clinical presentation and emergency interventions
- discuss hypoglycaemia and hyperosmolar nonketotic coma.

3.0 MAIN CONTENT

3.1 Diabetic coma; A diabetic coma is a life-threatening diabetes complication that causes unconsciousness. If you have diabetes, dangerously high blood sugar (hyperglycemia) or dangerously low blood sugar(hypoglycaemia) can lead to a diabetic coma.While diabetic mellitus (DM), commonly referred to as diabetes is a group of metabolic diseases in which there are high blood sugar levels over a prolonged period.Diabetic coma is a reversible form of coma found in people with diabetes mellitus. It is a medical emergency. Three different types of diabetic coma are identified namely;

(i) Diabetic ketoacidosis advanced enough to result in unconsciousness from a combination of severely increased blood sugar level, dehydration and shock, and exhaustion.

(ii) Severe low blood sugar in a diabetic person.

(iii) Hyperosmolar nonketotic coma in which an extremely high blood sugar level and dehydration alone are sufficient to cause unconsciousness.

3.1.1 Diabetic Ketoacidosis (Diabetic Coma)

Diabetic ketoacidosis is a serious complication of diabetes mellitus. It occurs when insulin levels become inadequate to meet the metabolic demands of the body.Pathophysiology: Diabetic ketoacidosis develops as blood glucose levels increase and individual cells become glucose-depleted. The body begins spilling sugar into the urine. This causes a significant osmotic diuresis and serious dehydration, evidenced by dry, warm skin and mucous membranes. As cellular glucose-depletion continues, ketone and acid production occur. Subsequently, the blood becomes acidotic. Deed respiration begin as the body tries to compensate for the metabolic acidosis. If the ketoacidosis is uncorrected, coma will follow. Clinical presentation: The onset of diabetic ketoacidosis is slow, lasting from 12 to 24 hours.

In its early stages, the signs and symptoms include increased thirst, excessive hunger, urination, and malaise. Increased urination results from the osmotic diuresis accompanying glucose spillage into the urine.Intensified thirst is caused by the body's attempt to replace the fluids lost by increased urination. Diabetic ketoacidosis is characterised by nausea, vomiting, marked dehydration, tarchycardia, and weakness. The skin is usually warm and dry. Coma is not uncommon. The breath may have a sweet or action-like character due to the increased ketone in the blood. Very deep, rapid respiration called kussmaul's respirations, also occur. Kussmaul's respiration represent the body's attempt to compensate for the metabolic acidosis produced by the ketones and organic acids present in the blood.Diabetic ketoacidosis is often associated with infection or decreased insulin intake. It may be complicated by several electrolyte imbalances. The most significant is decreased potassium. Decreased potassium (hypokalmia) can lead to serious dysrhythmias or even death. Ketoacidosis can occur in patients who fail to take their insulin or who take an inadequate amount over an extended period. Persons not previously diagnosed as diabetic will occasionally present in ketoacidosis.

Assessment: The approach used with the patient suffering from diabetic ketoacidosis is essentially the same as with any unconscious patient. You should first complete the primary assessment of airway, breathing, and circulation. You will then undertake the secondary assessment. Pay attention to the presence of Medic-Alert bracelets and/or insulin in the refrigerator. Also, take a history from bystanders. The fruity odor of ketones occasionally can be detected on the breath. If available,

complete the rapid test for blood glucose.Emergency intervention: If you can estimate the blood glucose level in the field, a high blood glucose can alert you to the classical signs and symptoms of diabetic ketoacidosis. In such cases, treatment should consist of drawing a red top tube(or the tube specified by local protocols) of blood. Following this, you should administer one to two litres of 0.9 percent sodium chloride. If transport time is lengthy, the medical control physician may request intravenous or subcutanous administration of regular insulin. If the blood glucose level cannot be quickly determined, draw a red top tube of blood for analysis and start an IV of normal saline. Following this, administer 50ml of 50 percent dextrose solution. If the patient is alcoholic, consider administering 100mg of thiamine. This additional glucose load will not adversely affect the ketoacidotic patient because it is negligible compared to the total quantity present in the body.

3.1.2 Hypoglycemia (Insuln Shock)

This occurs when insulin levels are excessive.Hypoglycaemia is an urgent medical emergency as a prolonged hypoglycaemic episode can result in serious brain injury.Pathophysiology: Hypoglycaemia, sometimes called insulin shock, lies at the other end of the spectrum from diabetic ketoacidosis. Hypoglycaemia can occur if a patient accidentally or intentionally takes too much insulin or eats an inadequate amount of food after taking insulin. If the patient is untreated, the insulin will cause the blood glucose level to drop to a very low level.This is atrue medical emergency.If the patient is not treated quickly, he or she, can sustain serious injury to the brain since it receives most of its energy from glucose metabolism.

Clinical presentation: The clinical signs and symptoms of hypoglycaemia are many and varied. An abnormal mental status is the most important. In the earliest stages of hypoglycaemia, the patient may appear restless or impatient or complain of hunger. As the blood sugar falls lower, he or she may display inappropriate anger (even rage) or display a variety of bizarre behaviours. Sometimes, the patient may be placed in a police custody for such behaviours or be involved in an automobile accident.Physical signs may include diaphoresis and tarchycardia. If the blood sugar falls to a critically low level, the patient may sustain a hypoglycaemic seizure or become comatose. In contrast to diabetic ketoacidosis, hypoglycaemia can develop quickly. A change in mental status can occur without warning. When encountering a patient behaving bizarrely, you should always consider hypoglycaemia.

Assessment: In suspected cases of hypoglycaemia, perform the primary assessment quickly. Inspect the patient for Medic-Alert bracelets. If possible determine the blood glucose level. Because of the seriousness

of the emergency, most of the clinic/hospital workers need to have the capability to perform this task or to rush a blood sample to the hospital along with the patient.
Emergency intervention: If the blood glucose level is noted to be less that 60mg/dl, draw a top red tube of blood and start an IV of normal saline. Next administer 50-100milliliters of 50 percent dextrose intravenously. If the patient is unconscious and able to swallow, complete glucose administration with orange juice, sodas, or commercially available glucose pastes. If the blood glucose cannot be obtained and if the patient is unconscious, you should start an IV of normal saline and administer 50-100milliliters 50 percent dextrose. Transport to a medical facility is also indicated. If you suspect alcoholism, administer 100mg of thiamine.

3.1.3 NonketoticHyperosmolar Coma

Nonketotic hyperosmolar coma usually develops more insidiously than DKA because the principal symptom is lethargy progressing to obtundation, rather than vomiting and an obvious illness. Extremely high blood sugar levels are accompanied by dehydration due to inadequate fluid intake. Coma from NKHC occurs most often in patients who develop type 2 or steroid diabetes and have an impaired ability to recognise thirst and drink. It is classically a nursing home condition but can occur in all ages.The diagnosis is usually discovered when a chemistry screen performed because of obtundation reveals an extremely high blood sugar level (often above 1800 mg/dl (100 mM)) and dehydration. The treatment consists of insulin and gradual rehydration with intravenous fluids.

3.1.4 Identifyingthe Cause/Treatment

Diabetic coma was a more significant diagnostic problem before the late 1970s, when glucose meters and rapid blood chemistry analysers became universally available in hospitals. Inmodern medical practice, it rarely takes more than a few questions, a quick look, and a glucose meter to determine the cause of unconsciousness in a patient with diabetes. Laboratory confirmation can usually be obtained in half an hour or less. Other conditions that can cause unconsciousness in a person with diabetes are stroke, uremic encephalopathy, alcohol, drug overdose, head injury, or seizure.Fortunately, most episodes of diabetic hypoglycemia, DKA (Diabetic Ketoacidosis), and extreme hyperosmolarity do not reach unconsciousness before a family member or caretaker seeks medical help.

Summary of causes
Acute infection and gastro-intestinal disorder, Dietary indiscretion, Consumption of high carbohydrates, Lack of insulin therapy, Undiagnosed diabetic mellitus. Diabetic coma occurs in uncontrolled diabetic; it develops more slowly than in hypoglacaemia usually over several days with signs of thirst (polyuria) glycosuria, and weakness.The glucose in the blood cannot be used by the cells and fat is brokendown to provide energy. The fat is metabolised andbroken down rapidly producing ketone bodies in excess of the tissue cells ability tometabolise them. The acids and ketones accumulates in the blood, the patient's urine shows a high concentration of sugar and ketones. The blood sugar is elevated, the sodium and chloride blood level are low, and there is dehydration.

Treatment: Generally, treatment depends upon the underlying cause:

- Hypoglycaemic diabetic coma: administration of the hormone glucagon to reverse the effects of insulin, or glucose given intravenously.

- Ketoacidotic diabetic coma: intravenous fluids, insulin and administration of potassium and sodium.

- Hyperosmolar diabetic coma: plenty of intravenous fluids, insulin, potassium and sodium given as soon as possible.

4.0 CONCLUSION

In this unit, you have learned what diabetes is and the componentstherein; you have also studied the clinical presentations, pathophysiology, and their interventions to enable you to also manage the disease. You should at this point be able to define and dsicuss diabetic coma in details.

5.0 SUMMARY

This unit has focused on the definition of diabetic coma as life-threatening diabetes complications that cause unconsciousness, characterised by hyperglacemia, itspathophysiology, clinical presentation, and intervention/management/treatment.You should also be able to ensure effective management ofdiabetes.

6.0 TUTOR-MARKED ASSIGNMENT

1 Define diabetic coma, diabetes mellitus and state the difference or differences.

2 Discuss in details the clinical presentation of patient with Hypoglycaemia .
3 Analyse the pathophysiology of a patient with Diabetic ketoacidosis .

7.0 REFERENCES/FURTHER READING

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UNIT 3 PEPTIC ULCER

CONTENTS

1.0 INTRODUCTION

Having gone through the course guide, you would have acquired a general overview of what peptic ulcer is. This unit will further help you acquire understanding of peptic ulcer asindicated in the objectives below.

3.0 OBJECTIVES

At the end of this unit, you should be able to:

- define the term peptic ulcer
- explain the types of peptic ulcers
- mention the clinical features of peptic ulcer
- state the management of peptic ulcer.

3.0 MAIN CONTENT

3.1 Definition of Peptic Ulcer

Peptic Ulcer Disease(PUD), also known as a peptic ulcer or stomach ulcer, is a break in the lining of the stomach, first part of the small intestine, or occasionally the lower esophagus.It also can be defined as mucosal erosions equal to or greater than 0.5cm ofan area of the gastrointestinal tract that is usually acidic and thusextremely painful. An ulcer in the stomach is known as a gastric ulcer while that in the first part of the intestines is known as a duodenal ulcer. The most common

symptoms are waking at night with upper abdominal pain or upper abdominal pain that improves with eating. The pain is often described as a burning or dull ache. Other symptoms include belching, vomiting, weight loss, or poor appetite. About a third of older people have no symptoms. Complications may include bleeding, perforation, and blockage of the stomach. Bleeding occurs in as many as 15% of people.

Common causes include the bacteria, *Helicobacter pylori* and non-steroidal anti-inflammatory drugs (NSAIDs). Other less common causes include tobacco smoking, stress due to serious illness, Behcet disease, Zollinger-Ellison syndrome, Crohn disease and liver cirrhosis, among others. Older people are more sensitive to the ulcer causing effects of NSAIDs. The diagnosis is typically suspected due to the presenting symptoms with confirmation by either endoscopy or barium swallow. *H. pylori* can be diagnosed by testing the blood for antibodies, a urea breath test, testing the stool for signs of the bacteria, or a biopsy of the stomach. Other conditions that produce similar symptoms include stomach cancer, coronary heart disease, and inflammation of the stomach lining or gallbladder.

Diet does not play an important role in either causing or preventing ulcers. Treatment includes stopping smoking, stopping NSAIDs, stopping alcohol, and medications to decrease stomach acid. The medication used to decrease acid is usually either a proton pump inhibitor (PPI) or an H2 blocker with four weeks of treatment initially recommended. Ulcers due to *H. pylori* are treated with a combination of medications such as amoxicillin, clarithromycin, and a PPI. Antibiotic resistance is increasing and thus treatment may not always be effective. Bleeding ulcers may be treated by endoscopy, with open surgery typically only used in cases in which it is not successful.

Peptic ulcers are present in around 4% of the population. About 10% of people develop a peptic ulcer at some point in their life. They resulted in 301,000 deaths in 2013 down from 327,000 deaths in 1990. The first description of a perforated peptic ulcer was in 1670 in Princess Henrietta of England. *H. pylori* was first discovered in 1981 by Barry Marshall and Robin Warren**.**

3.1.1 Signs and Symptoms/Clinical Presentations

Signs and symptoms of a peptic ulcer can include one or more of the following:

- abdominal pain, classically epigastric strongly correlated to mealtimes. In case of duodenal ulcers the pain appears about three hours after taking a meal;

- bloating and abdominal fullness;
- waterbrash (rush of saliva after an episode of regurgitation to dilute the acid in esophagus - although this is more associated with gastroesophageal reflux disease) nausea, and copious vomiting;
- loss of appetite and weight loss;
- hematemesis (vomiting of blood); this can occur due to bleeding directly from a gastric ulcer, or from damage to the esophagus from severe/continuing vomiting.
- melena (tarry, foul-smelling feces due to presence of oxidized iron from hemoglobin);
- rarely, an ulcer can lead to a gastric or duodenal perforation, which leads to acute peritonitis, extreme, stabbing pain,[9] and requires immediate surgery.

A history of heartburn, gastro-esophageal reflux disease (GERD) and use of certain forms of medication can raise the suspicion for peptic ulcer. Medicines associated with peptic ulcer include NSAIDs (non-steroid anti-inflammatory drugs) that inhibit cyclooxygenase, and most glucocorticoids (e.g. dexamethasone and prednisolone).In patients over 45 with more than two weeks of the above symptoms, the odds for peptic ulceration are high enough to warrant rapid investigation by esophagogastroduodenoscopy.

The timing of the symptoms in relation to the meal may differentiate between gastric and duodenal ulcers: A gastric ulcer would give epigastric pain during the meal, as**gastric acid**production is increased as food enters the stomach. Symptoms of duodenal ulcers would initially be relieved by a meal, as the pyloric sphincter closes to concentrate the stomach contents, therefore acid is not reaching the duodenum. Duodenal ulcer pain would manifest mostly 2–3 hours after the meal, when the stomach begins to release digested food and acid into the duodenum.Also, the symptoms of peptic ulcers may vary with the location of the ulcer and the patient's age. Furthermore, typical ulcers tend to heal and recur and as a result the pain may occur for few days and weeks and then wane or disappear. Usually, children and the elderly do not develop any symptoms unless complications have arisen.

Burning or gnawing feeling in the stomach area lasting between 30 minutes and three hours commonly accompanies ulcers. This pain can be misinterpreted as hunger, indigestion or heartburn. Pain is usually caused by the ulcer but it may be aggravated by the stomach acid when it comes into contact with the ulcerated area. The pain caused by peptic ulcers can be felt anywhere from the navel up to the sternum, it may last from few minutes to several hours and it may be worse when the stomach is empty. Also, sometimes the pain may flare at night and it can

commonly be temporarily relieved by eating foods that buffer stomach acid or by taking anti-acid medication.However, peptic ulcer disease symptoms may be different for every sufferer**.**

3.1.2 Complications

Gastrointestinal bleeding is the most common complication. Sudden large bleeding can be life-threatening. It occurs when the ulcer erodes one of the blood vessels, such as the gastroduodenalarter y Perforation (a hole in the wall of the gastrointestinal tract) often leads to catastrophic consequences if left untreated. Erosion of the gastro-intestinal wall by the ulcer leads to spillage of stomach or intestinal content into the abdominal cavity. Perforation at the anterior surface of the stomach leads to acute peritonitis, initially chemical and later bacterial peritonitis. The first sign is often sudden intense abdominal pain; an example is Valentino's syndrome, named after the silent-film actor who experienced this pain before his death. Posterior wall perforation leads to bleeding due to involvement of gastroduodenal artery that lies posterior to the 1st part of duodenum.

- Perforation and penetration are when the ulcer continues into adjacent organs such as the liver and pancreas.[10]
- Gastric outlet obstruction is the narrowing of pyloric canal by scarring and swelling of gastric antrum and duodenum due to peptic ulcers. Patient often presents with severe vomiting without bile.
- Cancer is included in the differential diagnosis (elucidated by biopsy), *Helicobacter pylori* as the etiological factor making it three to six times more likely to develop stomach cancer from the ulcer.[10]

3.1.3 Causes

H. pylori

A major causative factor (60% of gastric and up to 50-75% of duodenal ulcers) is chronic inflammation due to *Helicobacter pylori* that colonises the antralmucosa.The immune system is unable to clear the infection, despite the appearance of antibodies. Thus, the bacterium can cause a chronic active gastritis (type B gastritis). Gastrin stimulates the production of gastric acid by parietal cells. In *H. pylori*colonisation responses to increased gastrin, the increase in acid can contribute to the erosion of the mucosa and therefore ulcer formation.

NSAIDs
Another major cause is the use of NSAIDs. The gastric mucosa protects itself from gastric acid with a layer of mucus, the secretion of which is stimulated by certain prostaglandins. NSAIDs block the function of cyclooxygenase 1 (*cox-1*), which is essential for the production of these prostaglandins. COX-2 selective anti-inflammatories (such as celecoxib or the since withdrawn rofecoxib) preferentially inhibit *cox-2*, which is less essential in the gastric mucosa, and roughly halve the risk of NSAID-related gastric ulceration.

Stress
Stress due to serious health problems such as those requiring treatment in an intensive care unit is well described as a cause of peptic ulcers, which are termed stress ulcers. While chronic life stress was once believed to be the main cause of ulcers this is no longer the case. It is, however, still occasionally believed to play a role. This may be by increasing the risk in those with other causes such as *H. pylori* or NSAID use.

Diet
Dietary factors such as spice consumption were hypothesized to cause ulcers until late in the 20th century, but have been shown to be of relatively minor importance. Caffeine and coffee, also commonly thought to cause or exacerbate ulcers, appear to have little effect.Similarly, while studies have found that alcohol consumption increases risk when associated with *H. pylori* infection, it does not seem to independently increase risk. Even when coupled with *H. pylori* infection, the increase is modest in comparison to the primary risk factor.

Others
Although some studies have found correlations between smoking and ulcer formation,others have been more specific in exploring the risks involved and have found that smoking by itself may not be much of a risk factor unless associated with *H. pylori* infection.Gastrinomas (Zollinger Ellison syndrome), rare gastrin-secreting tumors, also cause multiple and difficult-to-heal ulcers.

3.1.4 Diagnosis

The diagnosis is mainly established based on the characteristic symptoms. Stomach pain is usually the first signal of a peptic ulcer. In some cases, doctors may treat ulcers without diagnosing them with specific tests and observe whether the symptoms resolve, thus indicating that their primary diagnosis was accurate.

Confirmation of the diagnosis is made with the help of tests such as endoscopies or barium contrast x-rays. The tests are typically ordered if the symptoms do not resolve after a few weeks of treatment, or when they first appear in a person who is over age 45 or who has other symptoms such as weight loss, because stomach cancer can cause similar symptoms. Also, when severe ulcers resist treatment, particularly if a person has several ulcers or the ulcers are in unusual places, a doctor may suspect an underlying condition that causes the stomach to overproduce acid.

An EsophagoGastroDuodenoscopy (EGD), a form of endoscopy, also known as a gastroscopy, is carried out on patients in whom a peptic ulcer is suspected. By direct visual identification, the location and severity of an ulcer can be described. Moreover, if no ulcer is present, EGD can often provide an alternative diagnosis.One of the reasons that blood tests are not reliable for accurate peptic ulcer diagnosis on their own is their inability to differentiate between past exposure to the bacteria and current infection. Additionally, a false negative result is possible with a blood test if the patient has recently been taking certain drugs, such as antibiotics or proton pump inhibitors.

The diagnosis of *Helicobacter pylori* can be made by:
Urea breath test (noninvasive and does not require EGD);Direct culture from an EGD biopsy specimen; this is difficult to do, and can be expensive. Most labs are not set up to perform *H. pylori* cultures;Direct detection of urease activity in a biopsy specimen by rapid urease test;Measurement of antibody levels in blood (does not require EGD) It is still somewhat controversial whether a positive antibody without EGD is enough to warrant eradication therapy;Stool antigen test;Histological examination and staining of an EGD biopsy.

The breath test uses radioactive carbon to detect H. pylori. To perform this exam the patient will be asked to drink a tasteless liquid which contains the carbon as part of the substance that the bacteria breaks down. After an hour, the patient will be asked to blow into a bag that is sealed. If the patient is infected with H. pylori, the breath sample will contain radioactive carbon dioxide. This test provides the advantage of being able to monitor the response to treatment used to kill the bacteria.
The possibility of other causes of ulcers, notably malignancy (gastric cancer) needs to be kept in mind. This is especially true in ulcers of the *greater (large) curvature* of the stomach; most are also a consequence of chronic *H. pylori* infection.

If a peptic ulcer perforates, air will leak from the inside of the gastrointestinal tract (which always contains some air) to the peritoneal cavity (which normally never contains air). This leads to "free gas"

within the peritoneal cavity. If the patient stands erect, as when having a chest X-ray, the gas will float to a position underneath the diaphragm. Therefore, gas in the peritoneal cavity, shown on an erect chest X-ray or supine lateral abdominal X-ray, is an omen of perforated peptic ulcer disease**.**

3.1.5 Classification

1. Esophagus
2. Stomach
3.Ulcers
4.Duodenum
5.Mucosa
6.Submucosa
7.Muscle

By area

- Duodenum (called duodenal ulcer)
- Esophagus (called esophageal ulcer)
- Stomach (called gastric ulcer)
- Meckel's diverticulum (called Meckel's diverticulum ulcer; is very tender with palpation)

Type I: Ulcer along the body of the stomach, most often along the lesser curve at incisuraangularis along the locus minorisresistantiae. Not associated with acid hypersecretion.

- Type II: Ulcer in the body in combination with duodenal ulcers. Associated with acid oversecretion.
- Type III: In the pyloric channel within 3 cm of pylorus. Associated with acid oversecretion.
- Type IV: Proximal gastroesophageal ulcer
- Type V: Can occur throughout the stomach. Associated with chronic use of NSAIDs(such as ibuprofen**).**

Macroscopic appearance
Gastric ulcers are most often localized on the lesser curvature of the stomach. The ulcer is a round to oval parietal defect ("hole"), 2 to 4 cm diameter, with a smooth base and perpendicular borders. These borders are not elevated or irregular in the acute form of peptic ulcer, regular but with elevated borders and inflammatory surrounding in the chronic form. In the ulcerative form of gastric cancer the borders are irregular. Surrounding mucosa may present radial folds, as a consequence of the parietal scarring.

Microscopic appearance
A gastric peptic ulcer is a mucosal defect which penetrates the muscularis mucosae and lamina propria, produced by acid-pepsin aggression. Ulcer margins are perpendicular and present chronic gastritis. During the active phase, the base of the ulcer shows four zones: inflammatory exudate, fibrinoid necrosis, granulation tissue and fibrous tissue. The fibrous base of the ulcer may contain vessels with thickened wall or with thrombosis.

Differential diagnosis

- Gastritis
- Stomach cancer
- Gastroesophageal reflux disease
- Pancreatitis
- Hepatic congestion
- Cholecystitis
- Biliary colic
- Inferior myocardial infarction
- Referred pain (pleurisy, pericarditis)
- Superior mesenteric artery syndrome.

3.1.6 Management/Treatment

Younger patients with ulcer-like symptoms are often treated with antacids or H2 antagonists before endoscopy is undertaken.People who are taking nonsteroidal anti-inflammatories (NSAIDs) may also be prescribed a prostaglandinanalogue (misoprostol) in order to help prevent peptic ulcers.Acid reducing medication Ranitidine and famotidine, which are both H2 antagonists, provide relief of peptic ulcers, heartburn, and indigestion. They decrease the amount of acid in the stomach helping with healing of ulcers. In the absence of *H. pylori*, 4 weeks of a PPIs are also often used.

H. pylori
When *H. pylori* infection is present, the most effective treatments are combinations of two antibiotics (e.g. clarithromycin, amoxicillin, tetracycline, metronidazole) and a proton pump inhibitor (PPI), sometimes together with a bismuth compound. In complicated, treatment-resistant cases, three antibiotics (e.g. amoxicillin + clarithromycin + metronidazole) may be used together with a PPI and sometimes with bismuth compound. An effective first-line therapy for uncomplicated cases would be amoxicillin + metronidazole + pantoprazole (a PPI).

Treatment of *H. pylori* usually leads to clearing of infection, relief of symptoms and eventual healing of ulcers. Recurrence of infection can occur and retreatment may be required, if necessary with other antibiotics. Since the widespread use of PPI's in the1990s, surgical procedures (like "highly selective vagotomy") for uncomplicated peptic ulcers became obsolete.

Surgery
Perforated peptic ulcer is a surgical emergency and requires surgical repair of the perforation. Most bleeding ulcers require endoscopy urgently to stop bleeding with cautery, injection, or clipping.

4.0 CONCLUSION

In this unit, you have learned that peptic ulcer is also known as stomach ulcer and is a break in the lining of the stomach, first part of small intestine, or occasionally the lower esophagus, it can also be described as mucosal erosions equal to or greater than 0.5cm of an area of the gastrointestinal tract, you have also gone through the signs and symptoms, complications, management/treatment, causes.
You can explain the whole episode in your own words.

5.0 SUMMARY

This unit has focused on the definition of peptic ulcer, its complications and diagnosis and management.

6.0 TUTOR-MARKED ASSIGNMENT

1. Define the term peptic ulcer.
2a. State the clinical features of peptic ulcer.
b. Enumerate complications of peptic ulcer.
3. Outline and discuss different classifications of peptic ulcer.

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UNIT 4 PERITONITIS

CONTENTS

1.0 INTRODUCTION

Peritonitis is the inflammation of the linings of the peritoneum
This unit will give you the general overview of the condition known as peritonitis .

2.0 OBJECTIVES

At the end of this unit, you should be able to:

- define the term peritonitis
- explain the clinical manifestations and complications of peritonitis
- state the causes of peritonitis and discuss the management of peritonitis.

3.0 MAIN CONTENT

3.1 Peritonitis

Is an inflammation of the peritoneum, the serious membrane which lines part of the abdominal cavity and viscera. Peritonitis may be localised or generalised, and may result from infection (often due to rupture of a hollow abdominal organ as may occur in abdominal trauma or inflamed appendix) or from a non-infectious process.The abdomen is lined with a membrane called the peritoneum. Most organs are located within the peritoneum; some however, are located behind it and are referred to as being retroperitoneal. These include the kidneys, portions of the duodenum, and portions of the pancreas. In certain disease states, the

peritoneum can become inflamed- a condition known as peritonitis. These medical conditions is characterised by generalised abdominal pains and rebound tenderness (Blumberg Sign).

3.1.1 Clinical manifestations/features

Abdominal pain and tenderness
The main manifestations of peritonitis are acute abdominal pain, abdominal tenderness and abdominal guarding, which are exacerbated by moving the peritoneum, e.g., coughing (forced cough may be used as a test), flexing one's hips, or eliciting the Blumberg sign (rebound tenderness, meaning that pressing a hand on the abdomen elicits less pain than releasing the hand abruptly, which will aggravate the pain, as the peritoneum snaps back into place). The presence of these signs in a patient is sometimes referred to as peritonism. The localisation of these manifestations depends on whether peritonitis is localised (e.g., appendicitis or diverticulitis before perforation), or generalised to the whole abdomen. In either case, pain typically starts as a generalised abdominal pain (with involvement of poorly localising innervation of the visceral peritoneal layer), and may become localised later (with the involvement of the somatically innervated parietal peritoneal layer). Peritonitis is an example of an acute abdomen.

3.1.2 Complications

Sequestration of fluid and electrolytes, as revealed by decrease venous pressure may cause electrolyte disturbance, as well as significant hypovolemia, possibly leading to shock and acute renal failure. A peritoneal abscess may form e.g. above or below the liver, or inthe lesser sac. Sepsis may develop so blood cultures should be obtained. The fluid may push on the diaphragm causing splinting and subsequent breathing difficulties.

3.1.3 Diagnosis

A diagnosis of peritonitis is based primarily on the clinical manifestations described above. If peritonitis is strongly suspected, then surgery is performed without further delay for other investigations. Leukocytosis, hypokalemia, hypernatremia, and acidosis may be present, but they are not specific findings. Abdominal X-rays may reveal dilated, edematous intestines, although such X-rays are mainly useful to look for pneumoperitoneum, an indicator of gastrointestinal perforation. The role of whole-abdomen ultrasound examination is under study and is likely to expand in the future. Computed tomography (CT or CAT scanning) may be useful in differentiating causes of abdominal pain. If reasonable doubt still persists, an exploratory peritoneal lavage or

laparoscopy may be performed. In patients with ascites, a diagnosis of peritonitis is made via paracentesis (abdominal tap): More than 250 polymorphonucleate cells per μL is considered diagnostic. In addition, Gram stain is almost always negative, whereas culture of the peritoneal fluid can determine the microorganism responsible and determine their sensitivity to antimicrobial agents.

3.1.4Causes of Peritonitis

Infected peritonitis
Perforation of part of the gastrointestinal tract is the most common cause of peritonitis, e.g. oesophagus, peptic ulcer, gastric carcinoma. Disruption of the peritoneum: Even in the absent of perforation of a hollow viscus, may also cause infection by letting microorganisms into the peritoneal cavity, e.g. trauma, surgical wound, continuous ambulatory peritoneal dialysis, intra-peritoneal chemotherapy, candida, etc. Intra-peritoneal dialysis predisposes to peritoneal infection, Spontaneous bacterial peritonitis: Is a peculiar form of peritonitis occurring in the absence of an obvious force of contamination in patients with ascetics. Systemic infections (such as tuberculosis) may rarely have a peritoneal localisation.

Non-infected peritonitis
Leakage of sterile body fluids into the peritoneum such as blood(e.g. endometriosis, blunt abdominal trauma), gastric juice (e.g. peptic ulcer, gastric carcinoma), bile (e.g. liver biopsy), urine (pelvic trauma), menstrum (salpingitis), pancreatic juice (pancreatitis), or even the contents of a ruptured dermoid cyst.
Sterile abdominal surgery: Normally causes localised or minimal generalised peritonitis, which may leave behind a foreign body reaction and/or fibrotic adhesion. Obviously,
peritonitis may also be caused by the rare, unfortunate case of a sterile foreign body inadvertently left in the abdomen after surgery (e.g. gauge, sponge). Much rare non-infectious causes may include familial Mediterranean fever, porphyria and systemic lupus erythematosus.

3.1.5 Management/Treatment of Peritonitis

General supportive measures such as vigorous intravenousrehydration and correction of electrolyte disturbances.Antibiotics are usually administered intravenously, but they may also be infused directly into the peritoneum. The empiric choice of broad-spectrum antibiotics often consist of multiple drugs, and should be targeted against the most likely agents, depending on the cause of peritonitis (see above); once one or

more agents are actually isolated, therapy will of course be targeted on them.

- Gram positive and gram negative organisms must be covered. Out of the cephalosporins, cefoxitin and cefotetan can be used to cover gram positive bacteria, gram negative bacteria, and anaerobic bacteria. Beta-lactams with beta lactamase inhibitors can also be used, examples include ampicillin/sulbactam,
- piperacillin/tazobactam, and ticarcillin/clavulanate.[1]Carbapenems are also an option when treating primary peritonitis as all of the carbapenems cover gram positives, gram negatives, and anaerobes except for ertapenem. The only fluoroquinolone that can be used is moxifloxacin because this is the only fluoroquinolone that covers anaerobes. Finally, tigecycline is a tetracycline that can be used due to its coverage of gram positives and gram negatives. Empiric therapy will often require multiple drugs from different classes.
- Surgery (laparotomy) is needed to perform a full exploration and lavage of the peritoneum, as well as to correct any gross anatomical damage that may have caused peritonitis.[1] The exception is spontaneous bacterial peritonitis, which does not always benefit from surgery and may be treated with antibiotics in the first instance..

4.0 CONCLUSION

In this unit, you have learned what peritonitis is, its clinical manifestations;complications and management. You can now read and understand the discussion and answer correctly the questions.

5.0 SUMMARY

This unit has focused on the description of peritonitis as inflammation of the peritoneum with its clinical presentations, diagnosis and complication. You should be able to describe peritonitis in your words.

6.0 TUTOR-MARKED ASSIGNMENT

1. Define the term peritonitis and discuss in details the causes of peritonitis .

2a. Describe in your own words the clinical manifestations of peritonitis.
b. Briefly discuss the complications of peritonitis.

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