# Chapter One

# Introduction

## 1.1 Background to the Study

Pressure ulcers (PU), also known as decubitus ulcers or bed sores, are skin injuries caused on by prolonged, continuous pressure, shear, friction, or a combination of these on a specific area of skin[1]. This pressure may limit blood flow to the location, which could cause injured tissue to die and become damaged[2]. Due to their susceptibility to pressure and tissue distortion, bony areas of the body typically experience pressure ulcers (PUs)[1]. Friction and shear are potential causes of pressure ulcer[3]. When a person is placed in these positions for some time (often 2 hours or less), tissue damage above the bone may occur just as a result of the person's body weight[4]. This is because protruding bones at the site of injuries apply pressure on the skin and tissue. Pressure, which might also be the beginning of a pressure ulcer, is present here. Pressure ulcers can produce varying degrees of injury to the skin, underlying tissues, muscles, and surface area over bony prominence[5].

In addition, pressure ulcers can be separated into four stages based on how badly the skin and underlying tissue have been damaged, with stage one only displaying non-blanchable erythema. There are no tears or breaches in the skin, even though it could hurt. Instead of blanching, the skin appears reddish. In a person with a dark complexion, the patch could not be red but still appear to be a different colour from the surrounding skin. Usually, skin temperature is higher[6]. The stage 1 sore may also feel different from the surrounding skin in terms of stiffness or softness. The skin rips apart, dries out, or forms an ulcer in stage two, which is often uncomfortable and sensitive. The deeper layers of the skin begin to feel the agony. It could look like a small crater, a blister, or an abrasion on the skin. This stage can occasionally resemble a blister filled with translucent fluid. At this stage, some skin may already be dead or irreparably damaged. In stage 3, the sore starts to degenerate and invades

the tissue beneath the skin, developing a small crater. Stage four implies entire thickness tissue loss with exposed bone, tendon, or muscle [7]. Fat may appear in the sore, but not muscle, tendon, or bone. The pressure injury is severe and causes the significant bone and muscle damage. Because the real depth of some ulcers cannot be established, they cannot be staged[8].

Although pressure ulcers can occur at any age, they are more common in the elderly, the immobile, and people who have severe, acute neurological problems[9].

Pressure ulcers (PUs) are a significant health concern, according to the healthcare industry[10]. Pressure ulcers pose a risk to a patient's health and well-being because they are one of the top five most common causes of patient harm[11]. Its global impact affects millions of people, and the degree and severity of injury to the skin, underlying tissue, and muscle varies[12]. It still poses a major health risk to 3 million adult patients[13]. Three million people are expected to be affected by pressure ulcers, which presents a serious challenge to medical experts [14].

Pressure ulcers rank third in terms of cost behind cancer and cardiovascular diseases[15]. Pressure ulcers are very common in hospitals; in European hospitals, they can occur anywhere between 8.3% to 23%, and from 1990 to 2003, they reached 26% in Canadian healthcare facilities[15]. Pressure ulcers were blamed for 29,000 deaths worldwide in 2013, up from 14,000 in 1990. The mortality rates from this illness are 2 to 6 times greater than those from other diseases[17], with 60,000 fatalities per year as a result of this consequence. This amounts to a considerable amount of morbidity annually in the United States[18].

Patients who have pressure ulcers exhibit severe physical and social dysfunction and are at risk of suffering from a variety of side effects, including depression, pain, skin infections, osteomyelitis, sepsis, and even death[14]. Rising mortality rates, a decline in quality of life, longer hospital stays, and rising healthcare expenses all pose serious threats to patient safety[19].

Adult pressure ulcer prevalence was estimated to be 12.8% globally[20]. Although medical technology has advanced, it still accounts for 14% to 42% of global mortality[21] and is most

common in hospitalized patients, especially those in the intensive care unit (ICU). Additionally, one year after being released from the hospital, pressure ulcers contributed to a 60% rise in mortality among senior patients.

Skincare is a crucial aspect of a patient's treatment. For patients and families, pressure ulcers can be devastating. If the pressure ulcer is severe, it may infect the bones, frequently (osteomyelitis). This kind of infection may necessitate pricey medical care, surgery, protracted hospital stays, extensive rehabilitation, and occasionally even amputation of the affected part or death. A patient's entire life may be impacted.

Financially, pressure ulcers are very expensive[22]. The interest in calculating the cost of preventing and treating pressure ulcers and their impact on patients, healthcare, and society stems from the demand for high-quality treatment when spending is constrained[22]. Stage IV pressure ulcers and their accompanying sequelae cost hospitals on average $129,248 per admission for hospital-acquired ulcers and $124,327 per admission on average for community-acquired ulcers[23].

These expenses alone highlight the significance of treating and preventing pressure ulcers. The significance of prevention is increased when human suffering is considered.

## 1.2 Statement of the Problem

Even though pressure ulcers are usually preventable, their prevalence is rising at an astounding rate[25]. Preventing this problem will not only safeguard patients from harm but also lower healthcare costs[26]. Pressure ulcer morbidity may call for more attention, resources, and a protracted hospital stay. Pressure ulcers in their advanced stages occasionally even lead to deadly infections. Pressure ulcers lead to 60 000 patient fatalities per year in the US.

Pressure ulcers are painfully painful, can lead to other diseases, and are highly expensive to treat. Pressure ulcers can have potentially lethal effects. Although research has shown that

there are variances in nurses' practices and training, pressure ulcer prevention is something that nurses are expected to be trained in. There are better odds of lowering the prevalence of pressure ulcers if healthcare workers are aware of the risk factors, screening procedures, and prevention measures.

### 1.3 Justification of the Study

Although they are common in high- and middle-income countries, less is known about pressure ulcers in low-income countries[27]. Although there is little open data regarding them, the problem of pressure ulcers is more prevalent in developing countries due to a lack of medical resources[28]. There is a lack of knowledge regarding nurses' practices and knowledge of pressure ulcer prevention in Sub-Saharan Africa. Considering the severe consequences of pressure ulcers and the possibility of prevention, there is little knowledge on pressure ulcer prevention among nurses in Sub-Saharan Africa. Assessing the prevalence and identifying the risk variables that affect the occurrence of PUs in developed and underdeveloped nations is therefore essential to filling this knowledge gap to create interventions that would prevent PUs and improve the standard of care.

### 1.4 Significance of the Study

Important information on the prevalence and risk factors of pressure ulcers in children, adolescents, and adults receiving medical care in hospital settings in Sub-Saharan Africa will be provided by the study. The study's findings can be utilized as a guide to help medical practitioners better grasp PUs screening and prevention methods in Sub-Saharan Africa.

### 1.5 Scope of the Study

The research will focus on literature evaluations on the prevalence and risk factors of pressure ulcers in Sub-Saharan Africa to order to design a strategy for pressure ulcer prevention.

Pressure ulcers are one of the top five patient hazards, per studies[29]. Although pressure injuries are frequently avoidable issues, they still have a significant negative impact on the healthcare system[30]. The study will examine the prevalence and risk factors for pressure ulcers, systematically identify the method used to prevent pressure ulcers in Sub-Saharan Africa, assess the results of the pressure ulcer risk assessment and grading scale, and determine the anatomical location of the pressure ulcer.

## 1.6 Aim and Objectives of the Study

Pressure ulcer prevention is one of the quality indicators to reduce the incidence of pressure ulcers in acute care settings[31]. This study aims to systematically assess the prevalence and risk factors of pressure ulcers in Sub-Saharan Africa

## 1.7 Primary Outcomes

**Primary Outcomes:**

1. To systematically assess the prevalence of pressure ulcers in Sub-Saharan Africa
2. To systematically assess the risk factors for pressure ulcers in Sub-Saharan Africa
3. To systematically assess the best-adopted strategy to prevent pressure ulcers in sub-Saharan Africa
4. To systematically assess the risk assessment and grading scores for pressure ulcer
5. To systematically assess the anatomical location of the pressure ulcer

### 1.7.1 Secondary Outcome

1. To systematically assess the pressure ulcer classification and risk assessment scales used in Sub-Saharan Africa

2. To systematically assess the country that has the most prevalent pressure ulcer in Sub-Saharan Africa

## 1.8 Review Question

1. What is the prevalence of pressure ulcers in Sub-Saharan Africa?
2. What are the pressure ulcer classification and risk assessment scales used in Sub-Saharan Africa
3. What are the risk factors for pressure ulcers in Sub-Saharan Africa?
4 Which country has the most prevalent pressure ulcer in Sub-Saharan Africa?
6. What is the best-adopted strategy to prevent pressure ulcers in sub-Saharan Africa?

## 1.9 Definition of Terms

Pressure ulcer: This is skin damage brought on by prolonged, continuous pressure on a particular area of the skin.

Fiction: Friction happens when two forces rub together. Pressure ulcers can occur when friction is combined with other forces, however friction alone typically does not cause skin injury or pressure ulcers.

Shear: This is defined as the force that can cause an object's planes to slide in the opposite direction. The amount of pressure placed on the underlying tissue has an impact on the shear.

Pressure ulcer risk assessment: This is a step in the procedure used to pinpoint those who are at a higher risk of getting a pressure ulcer. Skin assessment: This is based on a detailed examination, palpation, and recording of one's findings to determine the patient's overall physical state.

Risk factor: An element of one's behaviour or way of life, exposure to the environment, or inborn or inherited trait that is known to be connected to a health issue that is crucial to prevent based on epidemiological research.

Prevalence: The overall number of cases of a particular disease in a given population at a given time is known as the prevalence.

## Endnotes


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# Chapter Two

## 2.0 Review of Related Literature

The epidemiology of pressure ulcers will be investigated globally and in sub-Saharan Africa. The knowledge of pressure ulcers will be investigated among different healthcare populations from existing literature. Clinical practice of managing pressure ulcers will be investigated in the literature as well. Existing works of literature will be considered, available theoretical frameworks to use will be considered, and for the sake of this study, a theoretical framework will be developed.

### 2.1 Theoretical Framework

The theoretical framework is the framework for the theoretical presumptions of a research project. The theoretical framework introduces and describes the theory that underlies the existence of the study topic under investigation. Pressure ulcers have been studied by numerous researchers in a variety of contexts and dimensions. A conceptual framework used to identify pressure ulcers is shown in Figure 1 below. This starts with identifying the risk variables and classifying them into two categories: mechanical boundary conditions and the individual's susceptibility and tolerance. It will be possible to tell if there are internal strains and stresses present or a damaged threshold based on these categorizations. This will eventually reveal whether a pressure ulcer is there or not. The elements that operate as risk factors as indicators of a pressure ulcer's potential development are necessary for any study on pressure ulcers. Similar to this, it is important to make sure that the risk factors for pressure ulcers are what we access for and basic diagnostic, while ensuring that the attitude of basic practice guidelines and principles will serve as the guide to the rate their level of adherence to the basic practice of catering for a pressure ulcer patient.

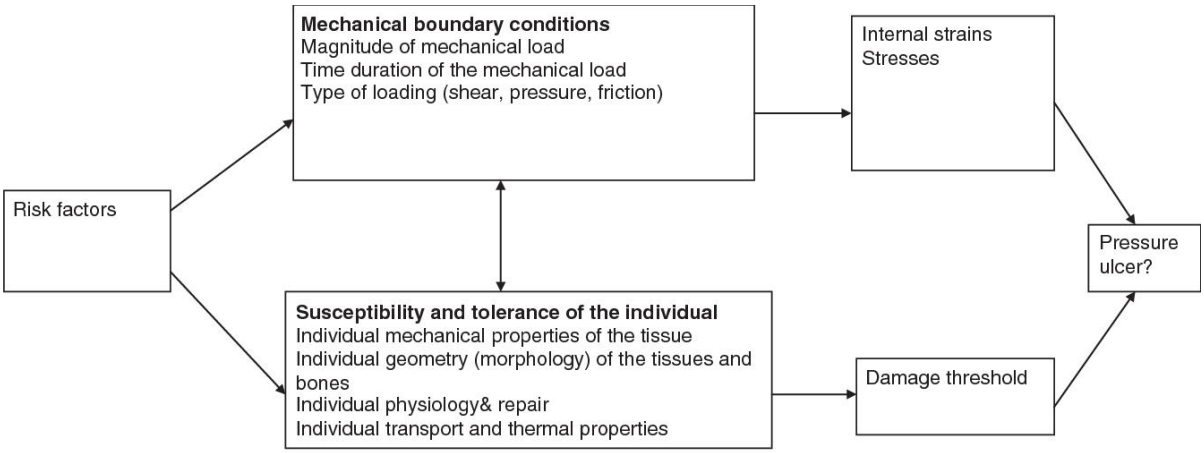


Figure 1: A sample pressure ulcer conceptual framework.

## 2.2 Conceptual Framework

It is best to approach the investigation of nurses' knowledge and practices in the prevention of pressure ulcers in Sub-Saharan Africa for this study from the angles of assessing their knowledge first in light of what they are expected to know and their practices assessed from the angles of what the standard of operation dictates.

Based on Rogers' theories of unitary humans, a conceptual framework for PU prevention and treatment will be applied in this study.

Martha E. Rogers developed her unitary human model based on concepts from systems theory. According to Rogers, a person's environment and self are fundamentally intertwined. She believed that because individuals and their environment are a single unit, they should be analyzed as such[27]. She also believes that once a transition has occurred, neither humans nor their environment can go back to their prior state and that humans and their environment adapt, advance, and evolve together.

In Roger's view of human nature, nursing is characterized as a humanistic and humanitarian art and science. The art of nursing is the use of the science of nursing in novel ways to enhance patient outcomes. The science of nursing is the information specific to nursing that is

acquired from scientific study. She claimed that the patient's environment cannot be separated while discussing health and therapy[28].

The purpose of nursing, in Rogers' view, is to promote harmonious interaction between a person and his environment to strengthen that person's coherence and integrity as well as to direct and reroute patterns of interaction between energy fields to realize that person's full potential for health[26].

By analyzing this unitary being, nurses can use the knowledge, attitude, and practice that they have obtained from nursing science to modify the prevention and treatment of PU in hospitals. Patient outcomes from PU prevention and treatment will be improved by the relationship between the patient and the environment. Nurses can analyze the patient's human variables (mobility, health status, dietary deficit status, age, tissue perfusion) and environmental factors (moisture, inadequate bedding, pressure, shear, and/or skin friction, usage of a wheelchair or lower-limb prosthesis) to help prevent and treat PU. To give patients with holistic nursing care for PU prevention and treatment, it is vital to assess these two human and environmental factors. Following the nurses' KAP review, which enhances the patient's environment and overall wellness, there is quality assurance assuring the patient's outcome.

### 2.3 Operational Definition of conceptions/terms/variables

Pressure ulcer: This is skin damage brought on by prolonged, continuous pressure on a particular area of the skin.

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Shear: This is defined as the force that can cause an object's planes to slide in the opposite direction. The amount of pressure placed on the underlying tissue has an impact on the shear.

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Risk factor: An element of one's behaviour or way of life, exposure to the environment, or inborn or inherited trait that is known to be connected to a health issue that is crucial to prevent based on epidemiological research.

Prevalence: The overall number of cases of a particular disease in a given population at a given time is known as the prevalence.

## 2.4 Pressure ulcer

Pressure ulcers, which are sometimes referred to as pressure sores, bed sores, or pressure injuries, are small regions of skin injury and/or underlying tissue damage that generally appear over a bony prominence as a result of prolonged pressure, especially when shear or friction is present[29]. The skin overlying the sacrum, coccyx, heels, and hips are the sites that are most frequently affected, however other places, such as the elbows, knees, ankles, back of shoulders, or the back of the skull, can also be affected[30].

Pressure ulcers can develop when pressure on soft tissue completely or partially prevents blood flow to the soft tissue. Another explanation is that shear can put stress on the blood vessels that supply the skin. Pressure ulcers are more likely to form in immobile people, such as those who frequently use wheelchairs or are bedridden for extended periods. It is widely acknowledged that other factors may impact the skin's ability to endure pressure and shear, increasing the risk of pressure ulcer development[30]. Under-nutrition in protein and calories, microclimate (sweating or incontinence-related skin moisture), arteriosclerosis, and diseases that impair skin sensitivity such as paralysis or neuropathy are a few of them. Pressure ulcer healing can be hampered by old age, diseases (such as arteriosclerosis, diabetes, or infections),

smoking, or medications like anti-inflammatory drugs[31]. Even though pressure ulcers are generally preventable and treatable if identified early (especially where spinal injury is involved)[31], they can be incredibly difficult to prevent in critically ill patients, elderly elders, and people with limited mobility like wheelchair users. Rotating the individual frequently to spread the pressure is the main preventative strategy. Turning has long been known to aid in preventing new sores, at least since the 19th century. Along with rotating and moving the client in the bed or wheelchair, it's important to provide a balanced diet with appropriate protein and protect the skin from urine and faeces. In European hospitals, the prevalence of pressure ulcers ranged from 8.3per cent to 23per cent, and from 1990 to 2003[32], it was 26 percent in Canadian healthcare facilities. In 2013, there were 29,000 deaths globally attributed to pressure ulcers, up from 14,000 in 1990[33].

Pressure ulcers can cause other illnesses, be excruciatingly painful, and be very expensive to treat. Anaemia, urethral fistula, gangrene, autonomic dysreflexia, bladder distension, bone infection, synarthrosis, sepsis, amyloidosis, autonomic dysreflexia, secondary carcinomas in chronic wounds, and very rarely malignant transformation (Marjolin's ulcer) are a few outcomes. Patients with pressure ulcers risk having their sores come back, and develop seromas, hematomas, infections, or wound dehiscence if their treatment is not adhered to. Paralyzed people are more prone to pressure sore recurrence. The consequences of pressure sores may be potentially fatal. The two leading causes of death are amyloidosis and renal failure. Everyone, regardless of age or pressure ulcer development stage, reports discomfort from them[32,33].

### 2.4.1 Autonomic Dysreflexia

Uncontrolled hypertension and bradycardia are the defining signs of autonomic dysreflexia, a potential medical emergency. Tachycardia is known to occur often. AD typically affects people with spinal cord injuries who have lesions at or above the T6 spinal cord level,

although it has been observed in patients with lesions as low as T10[34]. Guillain-Barré syndrome can potentially result in autonomic dysreflexia.

Minor symptoms including headaches, goosebumps, excessive perspiration above the lesion, and vision impairment might be brought on by uncontrolled hypertension in AD. However, seizures, cerebral bleeding, and retinal detachment are only a few of the potentially fatal effects of high hypertension. AD, which results in sympathetic activation and hyperactivity, can be brought on by both noxious and non-noxious stimuli[35]. Bowel or bladder over-distension brought on by faecal compaction or urine retention, respectively, are the most frequent causes. The ensuing sympathetic surge, which results in systemic vasoconstriction below the level of the spinal cord lesion, is transmitted by intact peripheral nerves. Due to peripheral artery vasoconstriction and hypertension, a parasympathetic surge that originates in the central nervous system prevents the sympathetic outflow, but the parasympathetic signal cannot pass below the level of the spinal cord lesion. Above the spinal lesion, this results in bradycardia, vasodilation, flushing, pupillary constriction, and nasal stuffiness because of the dominating sympathetic outflow, but below the lesion, there is piloerection and pale, cold skin[34,36].

The patient should sit up straight, remove any constricting clothing (such as abdominal binders and support stockings), have their blood pressure checked regularly, and then the doctor should search for and treat the underlying reason, which may need bowel or urine catheterization. Fast-acting, short-duration antihypertensives are an option to consider while the symptoms are being assessed for other contributing variables if the systolic blood pressure is still high (over 150 mm Hg) after the initial few measures have been taken. To stop AD, the patient, family, and caregivers must be made aware of any precipitating factors and how to avoid them, in addition to other triggers. Regular bowel and bladder programs, as well as

urological follow-up for cystoscopy/urodynamic studies, are required for prevention because of the prevalence of bladder and bowel problems[37].

### 2.4.2 **Autonomic Dysreflexia Significance and Symptoms**

These signs may include a "feeling of dread" or terror, extremely high blood pressure, severe headaches, intense sweating, facial erythema, goosebumps, stuffy nose, and blurred eyesight. This syndrome is distinct and frequently ephemeral.

### 2.4.3 Causes of Autonomic Dysreflexia

The current definition of dyslexia comprises an increase of 20 mm Hg over baseline systolic blood pressure with a likely cause below the neurological level of harm[37].

The first episode of autonomic dysreflexia can happen weeks, months, or years after the injury[38], but the majority of patients at risk (80%) suffer their first episode within the first year of spinal cord damage. Early AD blood pressure increases are frequently less severe. Urinary tract infections that have gone undiagnosed could be a common contributing factor. It might be more challenging to identify this if suprapubic or indwelling catheters are being used. Additional concerns include the side effects of prescription medications and different disease processes. CNS depressants and other psychotropic compounds can produce constipation and urine retention when used for an extended period, which can lead to autonomic dysreflexia. Urinary retention can also be brought on by stimulants such as cocaine and amphetamines. Recent scientific studies indicate that unpleasant (painful) stimuli are the primary cause of AD. (Remember that not every unpleasant stimulus will cause AD. Broken bones, for instance, which in unaffected people would be exceedingly uncomfortable stimuli, may not induce AD and may even go unnoticed.) However, other studies have shown that AD was not brought on by spinal cord injury patients activating pain receptors in the muscle and skin below the lesion. Because non-noxious stimuli can also cause AD, these findings show that not all noxious stimuli are reliable AD triggers. As a result, doctors may overlook underlying non-noxious

triggers when attributing an episode of AD to noxious stimuli. As a result, non-toxic trigger factors are overlooked and an AD episode is prolonged. They concluded that when identifying the probable causes of AD, it is important to consider both noxious triggers and non-noxious types of stimulation. Currently, bladder and bowel palpations and bladder scans are used in the evaluation of autonomic dysreflexia in individuals with established causal causes[38].

Supraspinal vasomotor neurons project onto the sympathetic preganglionic neurons (SPN) that make up the intermediolateral cell column (TML) via the T1-L2 segments. The SPN's influence on the SPN and its tonic firing have an impact on the adrenal medulla and the peripheral sympathetic chain ganglia. The sympathetic ganglia directly control vessel width and resistance on the blood arteries they innervate throughout the body, whereas the adrenal medulla indirectly governs the same function by releasing adrenaline and norepinephrine. There is a decrease in sympathetic outflow beneath the site of the damage as a result of the disruption of the descending autonomic pathways, which are in charge of the supraspinal interaction with the SPN. In this instance, spinal effects[39] are the only factors affecting the SPN. During the initial weeks after a spinal injury, decreased sympathetic output causes reduced blood pressure and sympathetic reflex. Afferent inputs like bowel or bladder distension cause the SPN synaptic remodelling and plasticity to become excessively sensitive over time, which results in abnormal reflex activation of the SPN. The spinal cord is impacted by the disruption of systemic vasoconstriction brought on by reflex activation. Peripheral artery vasoconstriction and hypertension activate baroreceptors, causing a parasympathetic surge to arise from the central nervous system and limiting the sympathetic outflow, even though the parasympathetic signal cannot reach below the level of the spinal cord lesion. Due to the dominant sympathetic output, this results in piloerection, pallid, and cold skin beneath the spinal lesion. Above the spinal lesion, it also results in vasodilation, flushing, pupillary constriction, and nasal congestion. The Splanchnic nerves originate at the T5 level and lower, which explains why injuries at or above the T6 level cause this issue to become much more

noticeable. When brain control over T6 and below is lost, the splanchnic arteries, the body's largest reserve of circulating blood, naturally vasoconstrict. Given that the splanchnic arteries are the body's greatest blood reserve, this has a major effect on blood pressure[39].

Antihypertensives must be utilized as a component of a successful treatment strategy for autonomic dysreflexia, which also includes quickly identifying and removing triggers. Frequently, by having the patient sit up and hang their legs over the edge of the bed, symptoms can be somewhat relieved and blood pressure can be lowered. It is advisable to remove any restricting clothing and stockings. Straight bladder catheterization or clearing a blocked urinary catheter tube could both be used to address the problem[40]. Stool impaction in the rectum should be resolved by the application of anaesthetic lubricating jelly. If the poisonous triggering trigger cannot be identified, drug therapy is necessary to reduce rising intracranial pressure until further investigation may identify the cause. In pharmacological therapy, vasodilators that act quickly are used[41]. Examples include oral hydralazine, clonidine, and sublingual or topical nitrates. Ganglionic blockers are additionally used to control sympathetic nervous system discharge. Topical nitroglycerin ointment can be applied sparingly to the forehead or chest wall and removed without risk after blood pressure levels begin to normalize. Autonomic dysreflexia is momentarily eliminated by spinal or general anaesthesia. When a woman experiences autonomic dysreflexia during obstetric delivery, several techniques are used[41].

#### 2.4.3.1.0 **Osteomyelitis**

Osteomyelitis is one type of bone infection (OM). Symptoms include fever, weakness, and pain in a specific bone that is accompanied by redness. While the feet, spine, and hips are most frequently damaged in adults, the long bones of the arms and legs, the femur and humerus, are more frequently affected in children[43].

Even though it doesn't happen often, a fungal infection can be to blame. It could disperse from adjacent tissue or through the blood. Risk factors for osteomyelitis include diabetes, intravenous drug use, having had your spleen removed in the past, trauma to the area, and intravenous drug use. The diagnosis is frequently made using the patient's symptoms and common laboratory tests like C-reactive protein (CRP) and erythrocyte sedimentation rate (ESR) (ESR). This is because, in the early days following an acute illness, plain radiographs are unremarkable. A bone sample, medical imaging, and blood tests are further diagnostic tools[44].

Bacterial osteomyelitis is usually treated with antibiotics and surgery. Patients with limited blood flow may require amputation. Mycetoma infections, a very unusual type of fungal osteomyelitis, must be treated with antifungal medications. Neglected fungal osteomyelitis, specifically mycetoma, where infections of the foot account for the majority of cases, is more likely to result in amputation or substantial skeletal resections than bacterial osteomyelitis. Treatment outcomes for bacterial osteomyelitis are often good when the infection has only recently developed. Per 100,000 people, there are 2.4 afflicted each year. The elderly and children are more frequently affected. Males are more likely to be affected than females. At least Hippocrates mentioned the ailment in the late third century BC. Before the development of antibiotics, there was a substantial danger of mortality[45,46].

**2.4.3.1.1 Symptoms of Osteomyelitis**

Children may have pain in a specific bone with concomitant redness, fever, weakness, and immobility as symptoms of acute bacterial osteomyelitis. Starting points might be sudden or gradual. Possibly enlarged lymph nodes in cases of fungal osteomyelitis, barefoot walking is frequently reported, especially in rural and agricultural areas. Fungal osteomyelitis starts as a skin infection and progresses into deeper tissues before reaching the bone, in contrast to bacterial osteomyelitis, which often travels by blood[45].

In youngsters, the metaphyses, or ends of the long bones, are usually affected. Adults are most typically affected by injury to the vertebrae and pelvis.

Due to the plentiful blood flow to the developing bones, acute osteomyelitis affects children who are otherwise healthy quite frequently. Adults may be affected by impaired host defences brought on by disease or medicine, intravenous drug abuse, infected root-canaled teeth, or other incapacitating diseases (such as immunosuppressive therapy)[46].

Osteomyelitis is a secondary complication of pulmonary TB that affects 1% to 3% of individuals. In this case, the bacteria often begin their infection in the synovium (because of its higher oxygen content) before travelling through the vascular system to the adjacent bone. In tubercular osteomyelitis, the long bones and vertebrae are commonly affected.

Staphylococcus aureus is the organism that is most frequently isolated from all kinds of osteomyelitis[45].

Staphylococcus aureus, which causes around 90% of all cases of bloodstream-sourced osteomyelitis in children, is the most common causative agent. Infants typically isolate S. aureus, Group B streptococci (the most common), and Escherichia coli, but children between the ages of one and sixteen are more likely to isolate S. aureus, Streptococcus pyogenes, and Haemophilus influenza. In particular subpopulations, such as intravenous drug users and patients with splenectomies, gram-negative bacteria, particularly enteric bacteria, are significant pathogens[45].

When a bone is injured and is locally contaminated, adults are more likely to have the disease in its most common form. Staphylococcus aureus is the most frequent bacterium discovered in osteomyelitis, which is disseminated by persistently infected areas. Anaerobes and Gram-negative organisms are also common, including Serratia marcescens, E. coli, and Pseudomonas aeruginosa. The majority of the time, mixed infections are the norm.

Systemic mycotic infections can also cause osteomyelitis. Coccidioides immitis and Blastomyces dermatitidis are the two most common. S. aureus is responsible for around half of all cases of osteomyelitis affecting the vertebral bodies, and TB is responsible for the other half (spread hematogenously from the lungs). Tubercular osteomyelitis of the spine was so common before effective antitubercular medications were developed that it was given the unusual name of Pott's illness. The Burkholderiacepacia complex45 has been associated with vertebral osteomyelitis in drug users.

#### 2.4.3.1.2 Diagnosis of Osteomyelitis

The diagnosis of osteomyelitis is challenging and depends on a combination of clinical suspicion and indirect test markers, such as a high white blood cell count and fever[47]. Imaging is frequently necessary, but it is not always necessary.

Radiographs and computed tomography (CT) are the first methods of diagnosis, however, these procedures are insensitive and have mediocre specificity. Although they could overlook an early or latent diagnosis, they can show cortical damage from advanced osteomyelitis[47].

The most typical application of MRI is confirmation. The presence of oedema, which is indicated by an increased signal on T2 sequences, is sensitive but not specific because it may arise in response to adjacent cellulitis. When bone marrow and cortical injury are validated by examining the T1 sequences, specificity is significantly boosted. The intravenous infusion of illnesses with an MRI, such as severe Charcot arthropathy. Sickle cell anaemia similarly struggles to distinguish between osteomyelitis and avascular necrosis[48].

Nuclear medicine scans can be a useful MRI supplement when a patient has metallic medical equipment that limits or prevents the use of magnetic resonance. Technetium 99 scans with three phases will frequently show improved absorption in each phase. Gallium scans are 100% sensitive for osteomyelitis even though they are non-specific, and they might be helpful for people who use a metallic prosthesis. 90% of osteomyelitis diagnoses using combined

WBC imaging and marrow investigations are accurate. Osteomyelitis is frequently diagnosed by radiologic findings that show a lytic centre surrounded by a ring of sclerosis.

Alternative sample methods, such as surface swabs or needle punctures, are easier to use but do not always produce reliable results. To determine the precise infection, a culture of bone biopsy material is necessary. Osteomyelitis may become complicated by bone fractures, amyloidosis, endocarditis, or sepsis[49].

**2.4.3.1.3Optimization for Osteomyelitis**

Osteomyelitis typically calls for prolonged antibiotic therapy lasting weeks or months. A PICC line or central venous catheter can be implanted to give long-term intravenous medicine. Numerous studies on kids with acute osteomyelitis suggest that oral antibiotic therapy may be necessary because of problems with PICC. In severe cases, it may be necessary to amputate or perform surgical debridement. Oral and intravenous antibiotic use appears to be the same[50].

Due to a dearth of data as of 2019, it is unknown what antibiotics work best for treating osteomyelitis in sickle cell disease patients. The initial first-line antibiotic choice is influenced by the patient's medical history and regional variations in the most common pathogenic organisms. A 42-day course of treatment is provided by several facilities. Drugs that are continually and locally available are more effective at providing therapeutic and preventive benefits. Open surgery, in which the involucrum is opened and the sequestrum is removed, is necessary for treating chronic osteomyelitis; cauterization is an alternative. Hyperbaric oxygen therapy has been shown to enhance the treatment of recalcitrant osteomyelitis[51]

Blow fly larvae were occasionally intentionally introduced to the wounds to feed on the polluted material, successfully cleansing them, before antibiotics were widely available and used. Preliminary research suggests that bioactive glass may be effective in treating long bone infections. But as of 2015, there were no randomized controlled trials to offer evidence. In

severe cases of terminal osteomyelitis in the pelvis, a hemicorporectomy is performed if another treatment is unsuccessful in stopping the infection[52,53].

#### 2.4.3.2 Sepsis

A potentially fatal condition known as blood poisoning, also known as sepsis or septicemia (septicemia in British English), is brought on when the body damages its tissues and organs while battling an infection. Once this initial stage is over, the immune system is subdued. The typical signs and symptoms include fever, rapid breathing, heart rate acceleration, and confusion. A cough associated with pneumonia or painful urination associated with a kidney illness is two other indications of an infection. The body temperatures of young children, the elderly, and people with weakened immune systems may be low or normal rather than raised, and they may not show any symptoms of a specific infection. Severe sepsis affects organ function or blood flow. Insufficient blood flow may be indicated by low blood pressure, elevated blood lactate levels, or decreased urine production. The fluid replacement does not improve septic shock, a low blood pressure condition caused by sepsis[54,55].

Sepsis can be caused by a wide variety of organisms, including bacteria, viruses, and fungi. The major infection typically affects the lungs, brain, urinary tract, skin, and abdominal organs. Risk factors include being extremely young or old, having a weakened immune system as a result of conditions like cancer or diabetes, suffering a serious accident, and having burns. In the past, a diagnosis of sepsis needed the fulfilment of at least two SIRS criteria in the setting of a suspected infection. Condensed sequential organ failure assessment score (SOFA score), often known as the quick SOFA score, replaced the SIRS system of diagnosis in 2016. (qSOFA). Rapid breathing, an altered state of consciousness, and low blood pressure are the three qSOFA sepsis criteria that must be met in at least two cases. Although blood cultures are recommended by sepsis guidelines before starting antibiotic therapy, an infection in the blood is not required for the diagnosis. Medical imaging helps try

to find the infection. Similar signs and symptoms may also be caused by anaphylaxis, adrenal insufficiency, low blood volume, heart failure, or pulmonary embolism[56].

Intravenous fluids and medicines must be administered as soon as possible to treat sepsis. The provision of continuing care is common in a critical care unit. If a significant trial of fluid replacement is insufficient to maintain blood pressure, the use of blood pressure-raising medications is necessary. Mechanical ventilation and dialysis may be necessary to preserve the health of the kidneys and lungs, respectively. A central venous catheter and an arterial catheter may be implanted to get access to the bloodstream and to administer direct care. Additional helpful parameters include cardiac output and superior vena cava oxygen saturation. Patients with sepsis require preventive therapies for deep vein thrombosis, stress ulcers, and pressure ulcers unless other conditions forbid such interventions. Strict blood sugar management with insulin may be advantageous for some persons. Corticosteroids are controversial; while some studies have found advantages, others have not[56].

The intensity of the illness has an impact on the outcome. Mortality risk of up to 30% is associated with sepsis, a 50% risk is associated with severe sepsis, and an 80% risk is associated with septic shock. Sepsis claimed the lives of 11 million people and affected 49 million others in 2017. (1 in 5 deaths worldwide). In the industrialized world, sepsis affects 0.2 to 3 people per 1000 people each year, accounting for nearly a million cases in the United States alone. The prevalence of diseases has increased. While some data indicate that sepsis is more common in men than in women, other data indicate that the condition is more common in women. Since Hippocrates' time, sepsis has been described[54].

The Surviving Sepsis Campaign advocated packed red blood cell transfusion if haemoglobin levels were below 70 g/L and there was no cardiac ischemia, hypoxemia, or serious bleeding. A 2014 study found that blood transfusion to keep target haemoglobin levels above 70 or 90 g/L did not affect survival rates, while patients with lower transfusion thresholds needed

fewer transfusions overall. Erythropoietin is not recommended for the treatment of anaemia in septic shock since it has the potential to cause blood clotting events. A fresh frozen plasma transfusion frequently does not address the underlying coagulation issues before a planned surgical procedure. If the patient's platelet count is below 10 109/L without any risk of bleeding, 20 109/L with a high risk of bleeding, or 50 109/L with active bleeding, platelet transfusion is indicated before planned surgery or invasive procedure. The use of IV immunoglobulin is not recommended due to the lack of certainty regarding its favourable effects. Intravenous immunoglobulin (IVIG) monoclonal or polyclonal preparations do not reduce the death rate in newborns or adults with sepsis. The use of polyclonal IVIG preparations that are IgM-enriched is supported by contradictory research. On the other hand, antithrombin therapy for disseminated intravascular coagulation is similarly ineffective. However, there is no evidence to support any survival benefit for septic shock56 from the removal of inflammatory mediators and bacterial toxins from the blood using blood purification procedures such as hemoperfusion, plasma filtration, and combined plasma filtration adsorption.

When bacteria or their toxins were found in the blood, the terms "blood poisoning" and "septicemia" were employed to characterize the condition. The International Statistical Classification of Disorders and Related Health Problems (ICD) version 9, which was in use in the US until 2013 and includes terminology like "Streptococcal septicemia," utilized the word "septicemia" with multiple modifiers for a variety of diseases. Each diagnosis in the ICD-10 has been converted to sepsis and modified with phrases like "Sepsis owing to streptococcus." Depending on the type of microorganism present, different terms are now used: bacteremia for bacteria, viremia for viruses, and fungemia for fungus. The disorder must be brought on by abnormal levels of bacteria in the blood. By the end of the 19th century, it was widely acknowledged that germs could harm the mammalian host and that the soluble toxins produced during infection were what caused the fever and shock that were typical of severe

infections. Around the start of the 20th century, Pfeiffer used the term endotoxin to describe the pyrogenic component of Vibrio cholera. It was soon found that the majority if not all, gram-negative bacteria expressed endotoxins. The lipopolysaccharide nature of intestinal endotoxins was explained by Shear in 1944. The molecular structure of the substance was discovered by Luderitz et al. in 1973.

In 1965, it was discovered that a certain strain of C3H/HeJ mouse was immune to the endotoxin-induced shock[57]. The genetic locus of this impact was designated Lps. Also demonstrated was how susceptible to gram-negative bacterial infection these mice were. These findings were finally related in 1998 to the identification of the toll-like receptor gene (TLR 4). Genetic mapping research conducted over five years clearly showed that a mutation within TLR4 must be the cause of the lipopolysaccharide resistance phenotype because TLR4 was the only probable locus inside the Lps critical area. The TLR4 gene deficit that led to the endotoxin resistance phenotype was determined to be caused by the cytoplasmic mutation[57].

Some authors argue that sepsis may not always be an unintentional result of the host immune system failing, even though sepsis is typically initiated by mutualistic (or neutral) microbiome components. The majority of the time, however, it is a microbial adaptation to a significant decline in the chances of the host surviving. The sepsis-causing microbe species in this instance benefits from controlling the future cadaver, utilizing its biomass as decomposers, and then dispersing through soil or water to establish mutualistic partnerships with new humans. When it comes to phenotypic plasticity, fungi like Candida spp., bacteria like Streptococcus pneumonia, Escherichia coli, Proteus spp., Pseudomonas aeruginosa, Staphylococcus aureus, Klebsiella spp., Clostridium spp., and Klebsiella spp. Not all sepsis patients have these adaptive microbial strategy shifts[58].

Paul E. Marik developed the "Marik protocol," also referred to as the "HAT" protocol, to treat sepsis in critical care patients by mixing hydrocortisone, vitamin C, and thiamine. Marik's

initial research, which was published in 2017, showed a clear benefit, which helped the approach acquire favour among doctors who specialize in intensive care. This was especially true following the protocol's exposure on social media and on National Public Radio, which led to a news conference where the greater medical community criticized science. The failure of a second independent study to replicate Marik's positive findings raises the possibility that they were tainted by bias. A systematic review of the trials done in 2021 revealed that it was impossible to verify the regimen's claimed benefits. HAT treatment "substantially decreased the period of vasopressor usage and improved the SOFA score," according to a more recent analysis, but "appears not to offer major advantages in other outcomes for patients with sepsis." Overall, as of 2021, there is still a dearth of reliable evidence that vitamin C can effectively treat sepsis[58].

**2.4.3.2.1 Sepsis causes**

Most infections that cause sepsis are bacterial, although they can also be viral, fungus, or parasitic. Gram-positive bacteria were the predominant cause of sepsis before the 1950s discovery of antibiotics. Between the 1960s and the 1980s, the introduction of antibiotics caused gram-negative bacteria to replace other types of bacteria as the primary cause of sepsis. Since the 1980s, it is believed that more than 50% of sepsis cases have been caused by gram-positive bacteria, most frequently staphylococci. More bacteria that are usually involved include Streptococcus pyogenes, Escherichia coli, Pseudomonas aeruginosa, and Klebsiella species. The most typical cause of fungal sepsis is a Candida yeast infection, a frequent condition contracted in hospitals. About 5% of instances of severe sepsis and septic shock are caused by it. Plasmodium, Echinococcus, and Schistosoma are the three parasites that cause parasite sepsis most frequently. Infections that cause severe sepsis most frequently affect the lungs, the abdomen, and the urinary tract. In 50% of instances, a lung infection is the initial

indication of sepsis. Unknown infection causes occur between one-third and fifty per cent of the time[59].

#### 2.4.3.2.2 Signs and Symptoms of Sepsis

Along with symptoms related to the actual cause, people with sepsis may also have symptoms including a fever, low body temperature, rapid breathing, a quick heartbeat, confusion, and oedema. Among the early warning, signs are a rapid heartbeat, decreased urine production, and increased blood sugar. Indicators of established sepsis include confusion, metabolic acidosis (which may be accompanied by a faster breathing rate that causes respiratory alkalosis), low blood pressure because of decreased systemic vascular resistance, a higher cardiac output, and blood-clotting problems that may cause organ failure[60]. Temperature is the most common early symptom of sepsis, however, some people, such as the elderly or those with compromised immune systems, may not even develop a fever. The drop in blood pressure that happens with sepsis, which can make a person feel dizzy, is one of the prerequisites for septic shock. Low blood levels of copper and vitamin C are related to septic shock and oxidative stress[60].

An early diagnosis is necessary for optimal sepsis management because fast therapy is crucial for reducing the death risk from severe sepsis. Some hospitals use electronic health record alerts to spread the word about potential situations as soon as they arise. Diagnostic testing should be carried out during the first three hours of suspected sepsis before antibiotics are given. White blood cell counts, measures of serum lactate, and appropriate culture collection should all be included in these procedures. To find the responsible organism, at least two sets of blood cultures using bottles with both aerobic and anaerobic media are necessary (s). One should be drawn through the skin and at least one through each vascular access device (such as an IV catheter) that has been in place for more than 48 hours. In just around 30% of cases, blood includes microorganisms. Another potential method of detection is the polymerase

chain reaction. If there are any doubts about further sources of infection, cultures from these sites should also be obtained, provided that doing so does not delay the delivery of antibiotics. Urine, cerebral fluid, wounds, and respiratory secretions are a few examples of this sources[61].

The central venous pressure and central venous oxygen saturation should be checked if the first fluid resuscitation of 30 ml/kg fails to improve blood pressure within six hours or if the first lactate is less than four mmol/l (36 mg/dl). The lactate should be evaluated if the initial value was high. Contrary to typical diagnostic techniques, point-of-care lactate measurement receives minimal support. A necrotizing soft tissue infection, an infection that causes inflammation of the lining of the abdominal cavity, an infection of the bile duct, or an intestinal infarction are examples of infections that need to be detected or ruled out very away. Petechiae, purpura, or purpura fulminans, a perforated internal organ (free air on an abdominal X-ray or CT scan), a pneumonia-consistent abnormal chest X-ray (with focussed opacification), and an abnormal chest X-ray are all indications of infection.

**2.4.3.2.3 Management of Sepsis**

By using focused therapy and early detection, sepsis outcomes may be improved. The most recent expert recommendations contain several steps (or "bundles") that must be carried out as soon as practical after diagnosis. Someone with sepsis should have received antibiotics and intravenous fluids within the first three hours if there is evidence of low blood pressure or other indications of insufficient blood flow to organs (as shown by an increased level of lactate). Within this timeperiod of cultures should also be acquired. Blood flow to the organs should be constantly monitored after six hours, and if the lactate level was initially elevated, it should be checked again. The blood pressure should also be appropriate. Similar procedures are frequently used in the UK and are referred to as the "Sepsis Six"; they include giving antibiotics within an hour of being diagnosed, giving blood cultures, determining lactate and hemhaemoglobinvels, checking urine output, giving high-flow oxygen, and giving

intravenous fluids. Surgical drainage of contaminated fluid collections, appropriate support for organ failure, and rapid administration of fluids and medications are further crucial elements of sepsis therapy. Medication and fluid therapy may be utilized in conditions of circulatory failure, renal failure, hemodialysis, mechanical ventilation, and blood transfusions. It's vital to provide the right nutrition throughout a protracted illness, usually through enteral feeding but possibly also through parenteral nutrition. Drugs can also be used to prevent deep vein thrombosis and stomach ulcers[61]. Antibiotics: Two blood cultures (aerobic and anaerobic) should be performed without delaying the initiation of antibiotic therapy. If infections from these regions are suspected, it is advisable to get cultures from additional locations, such as catheter insertion sites (left in place for more than 48 hours), respiratory secretions, urine, wounds, and cerebrospinal fluid. In cases of severe sepsis and septic shock, broad-spectrum antibiotics are indicated (typically two, a broad-spectrum -lactam antibiotic, or broad-spectrum carbapenem paired with fluoroquinolones, macrolides, or aminoglycosides). The likelihood that the patient would survive is influenced by the drugs chosen. Some argue that they should be administered an hour after the diagnosis because waiting an hour to administer antibiotics increases mortality by 6%. Others didn't benefit from early administration in any way. Several factors determine which antibiotic regimen is the best for the first time. These factors include the likelihood that the illness began in a hospital or the community, local patterns of bacterial antibiotic susceptibility, and the organ systems anticipated to be affected. Antibiotic regimens should be reviewed every day and, if required, narrowed. The course of treatment typically lasts 7 to 10 days, and the type of antibiotic given depends on the results of the cultures. To lessen the chance that the patient gets infected with several drug-resistant organisms, antibiotics should either be stopped altogether if an infection is not present or should be reduced in strength dependent on the patient's clinical response. A gram-negative-specific antibiotic should be added when a patient is at a high risk of acquiring numerous drug-resistant bacteria, such as Pseudomonas aeruginosa or Acinetobacter Baumann.

Treatment options for Methicillin-resistant Staphylococcus aureus (MRSA) include teicoplanin or vancomycin. For Legionella infections, a macrolide or fluoroquinolone should be used. If a fungal infection is found, an echinocandin, such as caspofungin or micafungin, is administered to patients with severe sepsis, followed by triazole (fluconazole and itraconazole) for less ill individuals. Prolonged antibiotic prophylaxis is not suggested in situations of SIRS without an infectious aetiology, such as severe pancreatitis and burns. Aminoglycoside can be given once daily without harming the kidneys and still attain the peak plasma concentration required for a therapeutic response. Antibiotics with low volume dispersion include vancomycin, teicoplanin, and colistin, and a loading dosage is required to achieve an appropriate therapeutic level to fight infections. To maintain the level above the minimum inhibitory concentration (MIC) and enhance therapeutic outcomes, beta-lactam antibiotics should be given often without going beyond the advised daily dose. Beta-lactam dosing that is continuous as opposed to intermittent may be better. Access to therapeutic drug monitoring is necessary to ensure proper therapeutic medication levels while preventing the drug from reaching unsafe levels [60,61]. Adults should receive 30 ml/kg of intravenous fluid in the first three hours to achieve the target mean arterial pressure (MAP) of 65 mmHg. Thereafter, the fluid should be adjusted based on blood pressure, urine output, respiratory rate, and oxygen saturation. For children in shock, a starting dose of 20 ml/kg is suitable. When a central venous catheter is used to dynamically measure blood pressure in cases of severe sepsis and septic shock, fluids should be administered until the central venous pressure reaches 8–12 mmHg. Once these goals are accomplished, the central venous oxygen saturation (ScvO2), or the oxygen saturation of venous blood as it returns to the heart as measured at the vena cava, is at its best. If the $ScvO_2$ is less than 70%, blood can be given to raisethe haemoglobin level to 10 g/dL, and then inotropes can be given to raise the $ScvO_2$ to the optimal level. Patients with acute respiratory distress syndrome (ARDS) and sufficient tissue and blood fluid should get more fluids, but with caution. The crystalloid solution is suggested as the preferred fluid

for resuscitation. Albumin can be given if a large amount of crystalloid is required for resuscitation. Crystalloid solutions and hydroxyethyl starch have comparable mortality risks. Starches also increase the possibility of severe renal impairment and the need for blood transfusions. Compared to crystalloids, certain colloid solutions (such as modified gelatin) offer no advantages. Additionally, albumin doesn't appear to have any benefits over crystalloids. Anesthesia: A goal tidal volume of 6 mL/kg of projected body weight (PBW) and a plateau pressure of less than 30 cm $H_2O$ are indicated for patients who require ventilation due to sepsis-induced severe ARDS. High positive end-expiratory pressure (PEEP) is indicated for moderate to severe ARDS in sepsis because it enables more lung units to open up for oxygen exchange. Depending on sex and height, tools are available to calculate projected body weight. Recruitment strategies that momentarily increthe transpulmonary pressure may be necessary for severe ARDS. To improve ventilation, it is suggested that the bed's head be raised if at all possible. However, it is not recommended to utilize 2 adrenergic receptor agonists to treat ARDS since they could reduce survival rates and cause irregular heartbeats. Utilizing a T-piece, CPAP, or inspiratory pressure augmentation during a spontaneous breathing trial will shorten the duration of ventilation. Reduced intermittent or continuous sedation can reduce the duration of mechanical breathing. General anesthesia is suggested for sepsis patients who need surgery to eliminate the infectious cause. Inhalation and intravenous anesthesia administration are both common. The requirement for anesthesia may decline as a result of sepsis. Proinflammatory cytokine levels can be decreased by inhalational anesthetics, which can alter leukocyte adhesion and proliferation, result in lymphocyte apoptosis (cell death), and perhaps negatively impact mitochondrial function. Despite having no impact on the cardiovascular system, etomidate is frequently not recommended as a medication to aid in intubation in this situation because to concerns that it may lead to reduced adrenal function and an increased risk of mortality. However, the scant data does not support the claim that etomidate lowers the risk of passing away. The use of

paralytic medications in sepsis patients without ARDS may lead to shorter stays in the intensive care unit (ICU), hospital, and mechanical ventilation, according to a growing body of studies. Paralytics' usefulness in ARDS cases is still up for debate, though. When used properly, paralytics may aid in efficient mechanical ventilation, but evidence also points to the possibility that mechanical ventilation in cases of severe sepsis may not boost oxygen delivery and consumption[61].

Vasopressors: If a patient has had enough fluid resuscitation but still has a mean arterial pressure below 65 mmHg, vasopressors are recommended. Norepinephrine, commonly known as noradrenaline, is suggested as a beginning point. When vasopressor therapy is started in septic shock later than is indicated, mortality rises. Since studies show that when shock lasts for 24 to 48 hours, there is a relative vasopressin deficit, norepinephrine is usually used as a first-line treatment for a hypotensive septic shock. Since norepinephrine elevates blood pressure via constricting blood vessels, it has little effect on heart rate and stroke volume. Some people might need to take a hazardous amount of vasopressor in order to boost their mean arterial pressure. To reduce the amount of vasopressor required, epinephrine may be administered. Epinephrine is a less frequent first-line treatment for hypotensive shock because it reduces blood supply to the abdominal organs and increases lactate levels. Vasopressin can be used in septic shock because research has shown that it is relatively inactive during the first 24 to 48 hours of shock. Contrarily, vasopressin reduces blood flow to the heart, hands, feet, and abdominal organs, denying these tissues oxygen. Dopamine is typically not recommended. Dopamine is more effective than norepinephrine at increasing the heart's stroke volume, but it also suppresses the immune system and causes more irregular heartbeats. The kidney-protective properties of dopamine are not supported by any research. In hypotensive septic shock, dobutamine can also be administered to increase cardiac output and re-establish normal blood supply to the tissues. Dobutamine is not used as frequently as

epinephrine because of its side effects, which include lowering blood flow to the intestines. Dobutamine also causes an unnatural rise in heart rate, which boosts cardiac output[62].

Biological Products: The Surviving Sepsis Campaign advocated packed red blood cells transfusion if hemoglobin levels were below 70 g/L and there was no cardiac ischemia, hypoxemia, or serious bleeding. A 2014 study found that blood transfusions to keep target hemoglobin levels above 70 or 90 g/L had no effect on survival rates, while patients with lower transfusion thresholds needed fewer transfusions overall. Erythropoietin is not recommended for the treatment of anaemia in septic shock since it has the potential to cause blood clotting events. A fresh frozen plasma transfusion frequently does not address the underlying coagulation issues before a planned surgical procedure. If the patient's platelet count is below 10 109/L without any risk of bleeding, 20 109/L with a high risk of bleeding, or 50 109/L with active bleeding, platelet transfusion is indicated before a planned surgery or invasive procedure. The use of IV immunoglobulin is not recommended due to the lack of certainty regarding its favourable effects. Intravenous immunoglobulin (IVIG) monoclonal or polyclonal preparations do not reduce the death rate in newborns or adults with sepsis. The use of polyclonal IVIG preparations that are IgM-enriched is supported by contradictory research. On the other hand, antithrombin therapy for disseminated intravascular coagulation is similarly ineffective. However, there is no evidence to support any survival benefit for septic shock from the removal of inflammatory mediators and bacterial toxins from the blood using blood purification techniques such as hemoperfusion, plasma filtration, and combined plasma filtration adsorption[63].

#### 2.4.3.3 Amyloidosis

A group of diseases known as amyloidosis occur when tissue begins to collect abnormal proteins known as amyloid fibrils. Numerous vague and non-specific signs and symptoms of amyloidosis exist. They include fatigue, weight loss, dyspnea, palpitations, peripheral oedema,

breathlessness upon standing, and disorientation. A swollen tongue and periorbital purpura are two specific symptoms of AL amyloidosis. Small ffibreneuropathy, autonomic dysfunction, biceps tendon rupture, bilateral carpal tunnel syndrome, and lumbar spinal stenosis are non-cardiac symptoms of wild-type ATTR amyloidosis[64].

There are 36 different types of amyloidosis, and each one is caused by a specific protein misfolding. These 36 proteins come in 19 forms that are localized, 14 systemic forms, and 3 forms that can self-identify as either. These proteins may become irregular due to both hereditary and acquired environmental factors. The four most common types of systemic amyloidosis are light chain (AL), inflammation (AA), dialysis-related (A2M), hereditary, and old age (ATTR and familial amyloid polyneuropathy)[65].

A diagnosis may be given when multiple peripheral nerves are damaged, it is unknown why, there is organ enlargement, and protein is found in the urine. To confirm the diagnosis, a tissue sample is performed. Because of the variety of presentations, diagnosing patients frequently require some time.

Reducing the concentration of the troublesome protein is the aim of treatment. Sometimes, this can be done by figuring out what the root cause is and dealing with it. Each year, 2 per million people get AA amyloidosis and 3-13 per million people get AL amyloidosis. Between 55 and 60 years of age is when these two divisions frequently start. Without therapy, people can expect to live anywhere between six months and four years. In the developed world, systemic amyloidosis claims the lives of about 1 in 1,000 people each year. Amyloidosis was first described at least in 1639[66].

The diagnosis of amyloidosis frequently requires tissue biopsies. It is looked for any signs of usual amyloid deposits in the biopsy. On the tissue, various stains are applied. Congo red is the best stain for recognizing amyloid, giving the amyloid proteins an apple-green appearance under a microscope when combined with polarized light. Another choice is thioflavin T stain.

Only a few of the imaging techniques used are a DPD scan and an SAP scan. Subcutaneous belly fat also referred to as a "fat pad biopsy," is the first-choice area for biopsy because it is the simplest spot to gather a tissue sample. But an internal organ that has been destroyed can also be directly sampled for tissue. Since an abdominal fat biopsy is not completely sensitive and may result in false negatives[67], a negative result does not exclude the diagnosis of amyloidosis.

Alternative, less invasive biopsy methods can also be used. For example, bone marrow or rectal mucosa biopsy can diagnose a patient in up to 85% of cases without requiring a direct biopsy of the afflicted organ. The amyloid accumulation in the joints will produce less signal on T1 and T2 weighted MRI scans. Amyloidoma is indicated by a low T1 signal with gadolinium injection and a low T2 signal. The detection of abnormal proteins in the bloodstream (using protein electrophoresis or light chain analysis), the binding of particular antibodies to the amyloid found in the tissue (using immunohistochemistry), and the extraction of the protein and identification of its amino acids are three methods for determining the type of amyloid protein.

While AL amyloidosis is typically missed by immunohistochemistry, AA amyloidosis is commonly picked up by it. Laser microdissection and mass spectrometry are the most precise methods for differentiating between the various kinds of amyloidosis. Doctors typically look for plasma cell dyscrasia, which occurs when memory B cells make immunoglobulins or immunoglobulin-like substances abnormally while diagnosing AL, which was long thought to be the most common kind of amyloidosis. Ninety per cent of people with AL amyloidosis report positive results from urine or serum immunofixation electrophoresis[65,67].

Immunofixation electrophoresis may not be available at all places, despite being more sensitive than conventional electrophoresis. If the electrophoresis came back negative but you have a strong clinical suspicion that you have AL amyloidosis, you may wish to consider

immunohistochemical labelling of a bone marrow sample to look for dominant plasma cells. ATTR is no longer recognized to be a rare disease and is now thought to be the most common form of amyloidosis. People without signs of plasma cell dyscrasias who have a family history of idiopathic neuropathies or heart failure could develop familial transthyretin-associated amyloidosis or age-related wild-type ATTR (ATTRv) amyloidosis. ATTR can be recognized via isoelectric focusing, which separates mutant forms of transthyretin. By searching for specific transthyretin mutations that are known to raise the risk of amyloidosis, genetic testing can be utilized to corroborate findings. Clinical evidence shows AA in people with chronic inflammation or recurrent infections. AA can be found using immunohistochemistry staining[67].

The three most common kinds of amyloidosis are AL, ATTR, and AA, with a global incidence of 30 per 100,000 persons. The average age upon diagnosis is 64.

AL has the highest incidence, with an estimated 12 cases per million people annually and a frequency of between 30,000 and 45,000 cases in the US and the EU.

The most typical form of amyloidosis in developing countries is AA, which can aggravate chronic infections with TB, osteomyelitis, and bronchiectasis. AA amyloidosis is brought on by an increase in the extracellular deposition of the serum amyloid A (SAA) protein. Both directly and indirectly, SAA protein levels can rise as a result of infections, inflammation, and malignancies. The most common causes of AA amyloidosis in the West are familial Mediterranean fever, psoriasis, inflammatory bowel disease, and rheumatoid arthritis. Patients on long-term hemodialysis (14–15 years) are more prone to amyloidosis due to the accumulation of HLA 1 complex light chains, which the kidneys would normally filter away.

According to postmortem results, one-fourth of older people had ATTR amyloidosis (wild-type transthyretin amyloidosis). 13–19% of individuals with heart failure and sustained ejection fraction have ATTR, a form of systemic amyloidosis that is fairly common [67].

#### 2.4.3.3.1 Amyloidosis Treatment

The course of treatment depends on the type of amyloidosis that is present. High dosage melphalan treatment followed by stem cell transplantation has shown promise in early studies and is recommended for stage I and stage II AL amyloidosis. Nevertheless, about 20–25 ppercentof individuals are eligible for stem cell transplantation. Cyclophosphamide-bortezomib-dexamethasone chemotherapy is now the recommended therapeutic option for AL Amyloidosis patients who are ineligible for transplants[68].

Symptoms might improve if the underlying ailment is treated in AA. Patients with inflammation brought on by AA amyloidosis often take TNF-alpha inhibitors for 20 months, such as infliximab and etanercept. If TNF-alpha medications are ineffective, interleukin-1 inhibitors (such as anakinra, canakinumab, and rilonacept) and interleukin-6 inhibitors (such as tocilizumab) may be investigated[69].

How ATTR amyloidosis should be treated depends on whether it is categorized as wild-type or variant. Both problems can be treated with tafamidis, an oral drug with low toxicity that prevents the disintegration of correctly folded proteins. Studies show that tafamidis reduced hospitalization and mortality due to heart failure. Up until recently, the only treatment for variant ATTR amyloidosis was liver transplantation[68]. Flunisal, inotersen, and patisiran are a few recent therapies.

Flunisal binds to misfolded mutant TTR protein to prevent its accumulation, working similarly to tafamidis. It may lessen the severity of peripheral neuropathy and the impairment brought on by the development of the disease, according to evidence with a low degree of certainty.

By suppressing the expression of both the wild-type and mutant TTR genes, inotersen lowers levels of amyloid precursors. It appears to stop the progression of peripheral neuropathy, according to somewhat certain evidence. To evaluate the long-term efficacy and safety of

inotersen use in patients with mutant TTR-related amyloidosis, a phase-III clinical trial will still be used as of 2021. Both inotersen and diflunisal may reduce quality-of-life declines, however, the evidence supporting this benefit is conflicting. It is uncertain how using inotersen would affect those with cardiac ATTR and more research is required. Inotersen was approved by the European Medicines Agency in 2018 to treat polyneuropathy in people with familial transthyretin amyloidosis. Since then, its use has received permission from the USA, the EU, and Canada[69].

Patisiran functions similarly to interest. Patisiran appears to reduce the worsening of disease-related disability and peripheral neuropathy, according to evidence with a reasonable degree of certainty. Additionally, research with a low degree of certainty shows that patisiran somewhat reduces the likelihood of adverse events when compared to a placebo and slows the loss in quality of life. There is no evidence that the fatality rates differ. Although this is still being investigated and requires more research, preliminary data from the use of patisiran in patients with variant cardiac ATTR suggest a potential reduction in mortality and hospitalization. Patisiran was not recommended by NICE in the UK in 2018 for inherited transthyretin-related amyloidosis. However, more evaluations are being conducted as of July 2019. But in the US, it was permitted for this use.

The roles of inotersen and patisiran in cardiac ATTR amyloidosis are still being researched. In a clinical trial using the CRISPR gene-editing technology in 2021, multiple subjects experienced a decline in TTR levels of "80% to 96%, on par or better than the average of 81 per cent." Vutrisiran has been approved by the American Food and Drug Administration (FDA) for the treatment of adult patients with polyneuropathy brought on by hereditary transthyretin-mediated (hATTR) amyloidosis.

#### 2.4.3.4 Anaemia

Anaemia, also known as anaemia (British English), is a condition that affects the ability of the blood to carry oxygen because of an unusually low number ofhaemoglobinn or red blood cells. Anaemia causes slow, confusing symptoms that include fatigue, weakness, dyspnea, headaches, and a reduced ability to exert oneself. Severe anaemia may manifest as confusion, feeling like one is about to pass out, losing consciousness, and increased thirst. There must be enough anaemia before someone starts to appear pale. Depending on the underlying cause, other symptoms could manifest. Anaemia before to surgery may increase your chance of needing a blood transfusion afterward. Short-term or long-term anaemia can range from mild to severe[71].

Anaemia can be caused by blood loss, a decrease in red blood cell production, or an increase in red blood cell lysis. Two sources of bleeding are trauma and gastrointestinal bleeding. Reduced production may be caused by several diseases, including iron shortage, vitamin B12 deficiency, thalassemia, and several bone marrow cancers. Malaria infections, autoimmune diseases, and genetic problems like sickle cell anemia can all result in an accelerated breakdown. Anaemia can also be classified based on the size of the red blood cells and the amount of hemoglobin in each cell. If the cells are small, they are called microcytic anemia, large cells are called macrocytic anemia, and normal-sized cells are called normocytic anemia. Anemia is defined as hemoglobin levels in males and women of less than 120 to 130 g/L (12 to 13 g/dL) or less than 130 to 140 g/L (13 to 14 g/dL), respectively. The cause must then be identified through additional testing[71-73.]

When they are first diagnosed, many persons with chronic disease-related anemia don't have any visible inflammation or dietary issues. Patients with spinal cord injuries, astronauts, elderly people with limited mobility, bedridden people, and test volunteers for bed rest are only a few of them who have decreased limb loading[74].

For a number of demographic groups, including pregnant women, preventative use of iron supplements is helpful. Dietary supplementation is not recommended unless the underlying cause has been determined. Depending on a patient's symptoms and signs, blood transfusions are commonly given. Asymptomatic individuals should not take them unless their hemoglobin levels are between 60 and 80 g/L (6 and 8 g/dL) or below. This advice may also be helpful for some people who are bleeding suddenly. Only patients with severe anaemia are advised to use erythropoiesis-stimulating medications[75].

Nearly one-third of people on the planet have anaemia, the most common blood condition. Anaemia due to iron deficiency affects about 1 billion people worldwide. Iron deficiency-related anemia claimed the lives of about 183,000 people in 2013, down from 213,000 in 1990. Although it also affects older people and women who are more fertile than usual, this illness affects children the most (especially during pregnancy). The prefixes an- and haima are the components of the Ancient Greek word anaemia, which means "absence of blood."

Anaemia is one of the six global diet-related and WHO nutrition goals for 2025 that were adopted by the World Health Assembly in 2012 and 2013. One of SDG 2's objectives is to eradicate anaemia, and efforts to meet international goals contribute to the accomplishment of all 17 Sustainable Development Goals (SDGs)[75].

Anaemia, according to some experts, is the most common blood disorder. A person with anaemia may not show any symptoms at all, or if they do, they may start mild and worsen as the anaemia worsens, depending on the underlying cause. A person with anaemia could feel weak, tired, have problems concentrating, and possibly experience dyspnea when they exert themselves.

The signs of anaemia might come on suddenly or gradually. At first, there can be few or no symptoms. The body may adapt and compensate if the anaemia is persistent and advances slowly. In this case, no symptoms might emerge until the anaemia deteriorates. A few

symptoms that may be present include breathlessness, headaches, dizziness, lack of physical activity (accompanied by shortness of breath and rapid heartbeat), cold hands and feet, constant coldness, sore tongue, pale or yellow skin, nausea, poor appetite, easy bruising and bleeding, and muscle weakness. Anemia that is rapidly advancing typically results in more severe symptoms like headaches, sweating, increased thirst, and confusion. There might also be additional symptoms, depending on the underlying cause[76].

The body may increase cardiac output in cases of more severe anemiato make up for the blood's reduced ability to carry oxygen. Palpitations, angina (if a prior heart problem occurs), intermittent claudication of the legs, and heart failure symptoms and signs can all result from this.

Pallor is not a trustworthy symptom, even though it may be evident upon inspection (pale skin, mucosa, conjunctiva, and nail beds). The sclera of certain persons with iron-deficiency anaemia could appear blue. Leg ulcers, koilonychia (in iron deficiency), jaundice (when hemolyticanaemia results in aberrant red blood cell breakdown), nerve cell damage (from vitamin B12 deficiency), deformed bones (in thalassemia major), and vitamin B12 insufficiency are all possible signs of particular forms of anaemia (seen in sickle-cell disease). Hyperdynamic circulation symptoms that may be present in severe anaemia include tachycardia (rapid heartbeat), bounding pulse, flow murmurs, and cardiac ventricular hypertrophy (enlargement). There could be signs of heart failure. Although the habit of eating things other than food, such as ice, paper, wax, grass, hair, or dirt, is known as pica, it frequently affects people with normal haemoglobin levels despite being a sign of iron deficiency. Chronic anaemia can result in behavioural abnormalities in children as a direct result of infants' poor brain development and school-age children's inferior academic ability. Restless legs syndrome affects more people with iron deficiency anaemia than the general population[74,76].

### 2.4.3.5 Gangrene

Gangrene is a type of tissue death brought on by a lack of blood supply. Possible symptoms include the emergence of red or black skin, numbness, swelling, soreness, and cooling. Most often, the hands and feet are affected. A fever or sepsis might arise if an infectious pathogen caused gangrene[77].

Hypernatremia, radiation wounds, meningitis, group B streptococcal infection, Raynaud's syndrome, diabetes, peripheral vascular disease, smoking, severe trauma, alcoholism, HIV/AIDS, frostbite, influenza, dengue fever, malaria, chickenpox, plague, and smoking are risk factors. Necrotizing fasciitis, gas gangrene, internal gangrene, dry gangrene, and wet gangrene are some of the classifications for it. Based on symptoms, gangrene is identified, and further testing, such as medical imaging[77], confirms the diagnosis.

Treatment options could include surgery to remove the dead tissue, antibiotics to treat any infection, and steps to deal with the underlying cause. Other surgical methods include debridement, amputation, or the application of maggot therapy. Bypass surgery or angioplasty may be used to treat the underlying cause. In some cases, hyperbaric oxygen therapy may be beneficial. How frequently the condition manifests is unknown[78].

As early as 1028, flies and maggots were frequently used to treat chronic wounds or ulcers to stop the spread of necrotic tissue because some species of maggots only eat dead flesh, leaving nearby living tissue intact. This practice essentially vanished when an enzyme, acetonitrile, and antibiotics were added to the list of wound treatments. However, the use of maggot treatment has recently regained some credibility and is sometimes very effective in cases of prolonged tissue necrosis. While overseeing the performance of his Te Deum in January 1687, French Baroque composer Jean-Baptiste Lully cut his toe with the tip of his pointed staff, causing gangrene (which was used as a baton). Despite the infection spreading to his leg, the composer's toe was refused to be amputated; as a result, he passed away in

March of that year. Four days before his 77th birthday, on September 1, 1715, French King Louis XIV passed away from gangrene in his leg. Sebald Justinus Brugmans, Professor at Leyden University, Director of the Medical Bureau of the Batavian Republic from 1795 on, and Inspector-General of the French Imperial Military Health-Service in 1811 rose to prominence in the fight against hospital gangrene and its prevention. He painstakingly investigated and detailed the causes of this terrible disease, which he was convinced was spreadable, in a book on gangrene that was published in 1814. He meticulously reviewed every possible hygienic rule and submitted his work. His writings were well received, and they were essential in convincing the vast majority of authors who came after him that gangrene was an infectious disease[78].

John M. Trombold is the author. "A doctor in the Union Army named Middleton Goldsmith meticulously investigated hospital gangrene during the American Civil War and developed a revolutionary treatment regimen. During the Civil War, 45% of deaths from hospital gangrene were caused by this disease. Goldsmith's method was applied to over 330 occasions and resulted in a fatality rate of under 3%. " Goldsmith advised employing debridement as well as topical and intravenous bromide treatments on infected wounds to minimize the occurrence and virulence of "poisoned miasma." Union surgeons were given copies of his book to encourage the use of his methods. Father Camille Bulcke succumbed to gangrene on August 17, 1982.

In ischemic tissue, a type of coagulative necrosis called dry gangrene appears when there is inadequate blood flow to keep the tissue viable. Instead of being a disease in and of itself, it is a symptom of other illnesses. The word "dry" is only applied to a limb or the belly (in other locations, this same type of necrosis is called an infarction, such as a myocardial infarction). Although acute limb ischemia can result in dry gangrene, peripheral arterial dysfunction most often does. As a result, people with arteriosclerosis, high cholesterol, diabetes, and smokers

frequently get dry gangrene. Low oxygen levels in the ischemic limb prevent putrefaction, and germs cannot survive there. The affected area is dry, shrunken, and dark reddish-black. Most of the time, the line of separation causes total separation, and if the gangrenous tissue is not surgically removed, a process known as autoamputation, the gangrenous tissue will ultimately fall off. Dry gangrene is the result of ongoing ischemia without infection. If ischemia is detected early and ischemic wounds rather than gangrene are visible, revascularization may be utilized to treat it (via vascular bypass or angioplasty). However, once gangrene has taken hold, the injured tissues cannot be saved. Because it is not accompanied by infection, dry gangrene is less urgent than gas gangrene or wet gangrene, both of which provide a risk of sepsis. Dry gangrene may eventually develop into wet gangrene if an infection gets to the dead tissues. Peripheral vascular disease, which raises the risk of dry gangrene, is more likely to occur in people with diabetes mellitus. However, because high serum glucose promotes the proliferation of microorganisms[79], it also raises the risk of moist gangrene, particularly in people with poorly controlled blood sugar levels.

Wet gangrene, which differs from dry gangrene in that it is characterized by thriving bacteria, has a poor prognosis because sepsis is brought on by the unrestricted exchange of infected fluid with circulatory fluid. Two types of saprogenic bacteria, such as Clostridium perfringens or Bacillus fusiformis, infect the tissue in wet gangrene, causing it to swell and emit an unpleasant odour. Wet gangrene frequently advances swiftly as a result of venous or arterial blood flow restriction (more frequently). Blood that has been stagnating in the affected area is saturated, which promotes the rapid growth of germs. When toxic by-products of bacteria are consumed, sepsis and ultimately death result from its systemic expression. It is black, decaying, stinking, bloated, and soft. To lessen the systemic effects of the infection, an emergency salvage amputation, such as a guillotine amputation, is occasionally necessary due to the high mortality rate of infected gangrene (approximately 80% without therapy and 20%

with care). Such an amputation can lead to a formal amputation, such as one below or above the knee[80].

Gas accumulates within tissues as a result of the bacterial disease gas gangrene. It can be caused by clostridial or nonclostridial species, most frequently the C. perfringens strain that produces alpha toxin. The infection spreads rapidly as the bacteria's gases move and contaminate nearby healthy tissue. Due to its quick propensity to spread to nearby tissues, gas gangrene must be treated as a medical emergency. This kind of gangrene has a high mortality rate (50%) even with treatment and can be fatal if left untreated. The most common causes of gas gangrene are clostridial species, which are primarily found in soil and produce exotoxins, as well as other anaerobes such as Bacteroides and anaerobic streptococci.

These environmental bacteria can enter the muscle through cuts, where they subsequently flourish in necrotic tissue where they emit toxic toxins that destroy nearby tissue and also produce gas. 5.9 per cent hydrogen, 3.4 per cent carbon dioxide, 74.5 per cent nitrogen, and 16.1 per cent oxygen were the gas compositions in one clinical sample. Infection, necrosis, and gas production are some of the symptoms of gas gangrene. Toxaemia and shock frequently progress rather quickly[79,80].

**2.5 Pressure Ulcer Epidemiology**

Pressure ulcer-related deaths increased from 14,000 in 1990 to 29,000 in 2013. Each year, there are more than 2.5 million pressure sores in the US. In the US, the prevalence of bedsores varies from 0.4% to 38% in acute care settings, from 2.4% to 23.98% in long-term care, and from 0% to 17% in in-home care. There is a wide range, just like the prevalence: 0 to 29% for home care, 2.3 to 28% for long-term care, and 10 to 18% for acute care. Intensive care units (ICUs) have a significantly higher risk of bedsores due to immunocompromised patients, with 8–40% of patients developing them. Pressure ulcer incidence, however, is significantly influenced by the method used to collect the data. The European Pressure Ulcer Advisory

Panel (EPUAP) technique has similar statistics for pressure ulcers in acutely ill hospital patients. Pressure ulcer prevalence in Europe ranged from 8.3 per cent (Italy) to 22.9 per cent using this method, albeit there are differences between countries (Sweden). Recent Jordanian research has also indicated a value in this range. According to some studies, white or black nursing home residents experience pressure ulcers differently.

Pressure sores are still common, with a prevalence of 5 to 9% and more than 70% occurring in adults over the age of 70. They are occasionally wrongly attributed to poor nursing care, although it would be more beneficial to view them as a potentially treatable side effect of an acute immobility illness. Preventive measures include identifying patients who are at risk, using appropriate nursing care techniques, and using specialist equipment. Most specialized equipment is overly complex and has not been proven to be effective in clinical trials. The airwave system, polystyrene bead bed system, and Vaperm mattress are among the best researched and most efficient systems. The management of an established sore includes the treatment of the underlying medical condition(s), attention to hydration and nutrition, prevention of further tissue damage, use of specialized dressings, and methods that support the inflammatory healing response. Gauze, boric acid, and chlorinated lime solution (Eusol), which are considered "conventional" wound treatments, are seriously questioned. There are several more recent products available right now, but none of them has been put through rigorous clinical testing. As a good place to start, pressure sores can be categorized into four clinical types based on the extent of tissue damage and the depth of the ulcer. The type 1 sore, which is the least severe, can be covered with polyurethane film dressings. Deeper ulcers (types 2 and 3) can be treated quickly and easily with hydrocolloid or alginate dressings that improve the local wound environment, which promotes tissue repair. However, there might not be a good dressing for sacral (near-anal) sores, which are more difficult to treat than sores on other parts of the body because of dressing detachment. Cavity ulcers can be treated with silastic foam, hydrocolloid, or alginate dressings (type 4). The best tools for manually

removing necrotic material are a scalpel and scissors, but when used properly, streptokinase/streptodornase (Varidase Topical) may also be effective. The best method for treating ulcers is warm normal saline because antiseptics are generally ineffective. While systemic antibiotics should only be used when surrounding cellulitis is present[81], metronidazole is useful for painful sores.

## 2.6 Classification of Pressure Ulcer

The definitions of the various pressure ulcer phases are routinely updated by the European Pressure Ulcer Advisory Panel (EPUAP) and the National Pressure Injury Advisory Panel (NPUAP) in the United States and Europe, respectively. Many classification systems are used globally[82], depending on the health system, the medical area, and the categorization's objective (such as health care versus prevalence study versus financing). In brief, they are as follows:

Stage I: Normally over a bony prominence, intact skin with a confined, non-breathable patch of redness. Skin that is heavily pigmented could not blanch visibly and would instead have a distinct colour from the surrounding skin. The area has a different thickness and temperature from the tissue around it. Dark-skinned individuals may have difficulty identifying Stage 1 patients and may mistake them for those who are "in danger" (a heralding sign of risk).

Stage II: The dermis has lost some of its thickness as seen by a shallow open ulcer with a reddish-pink wound bed but no slough. A serum-filled blister that is either whole or open/ruptured may also be present. A superficial, glossy, or dry ulcer that is not sloughed or bruised is how it manifests. This stage should not be used to describe skin rips, tape burns, perineal dermatitis, maceration, or excoriation.

Stage III: Complete tissue thickness loss. Although bone, tendon, or muscle are not visible, subcutaneous fat might be. Slough may be evident, but it cannot conceal the extent of tissue loss. includes tunnelling and destroying. The depth of stage 3 pressure ulcers varies by

anatomical location. Stage 3 ulcers on the nasal bridge, ear, occiput, and malleolus may be shallow and devoid of (adipose) subcutaneous tissue. On the other hand, areas with a lot of adiposities might lead to very profound stage 3 pressure ulcers. Bone and tendon cannot be felt or seen right away.

Stage IV: Complete tissue loss with skeletal muscle, tendon, or bone visibility Eschar or slough may be seen in some parts of the wound bed. frequently include tunnelling and undermining. Pressure ulcers in stage 4 differ in depth based on anatomical location. Since there is no (adipose) subcutaneous tissue on the nasal bridge, ear, occiput, or malleolus, these ulcers may be shallow. Stage 4 ulcers that have migrated to muscles or supporting tissues increase the risk of developing osteomyelitis (such as fascia, tendons, or joint capsules). A bone or tendon that is exposed is obvious or palpable. In 2012, the National Pressure Injury Advisory Panel classified pressure ulcers with discernible cartilage as stage 4 pressure ulcers.

Unstageable: Full-thickness tissue loss happens when the slough and/or eschar in the wound bed completely conceal the depth of the ulcer. Slough can be brown, grey, green, tan, yellow, or tan. Only when enough slough and/or eschar have been removed to expose the wound's base can the precise depth and stage be determined. Eschar that is stable (dry, adherent, unbroken, without erythema or fluctuance) and covers the heels and is acting as protection should not be removed.

### 2.7 Prevention of Pressure Ulcer

The UK's Royal College of Nursing published a report on pressure ulcer risk assessment and prevention that includes suggestions urging care providers to identify those who are at risk and take protective steps on their behalf. Recent studies in the US and South Korea aimed to automate risk assessment and categorization by training machine learning models using electronic health data. In partnership with wound organizations from 15 different countries, the NPIAP, EPUAP, and Pan Pacific Pressure Injury Alliance updated the global evidence-

based clinical practice guideline in 2019. An international team of more than 180 clinical professionals developed the 2019 guideline, which updates the 2009 EPUAP/NPUAP clinical guideline and the 2014 NPUAP/EPUAP/PPPIA clinical guideline. The suggested techniques for treating pressure ulcers include bed rest, pressure-redistributing support surfaces, nutritional support, repositioning, wound care (such as debridement, and wound dressings), and biophysical agents (e.g. electrical stimulation). However, the use of many of these therapies is not supported by sufficient scientific research. More research is needed to find the best ways to help treat pressure ulcers, such as via repositioning. Furthermore, it is not yet known whether giving systemic or topically administered antibiotics can help with pressure ulcer therapy[83]. 2.7.1 **Redistribution of Pressure**: The most important part of treatment for someone with bedsores or at risk of acquiring them is redistributing pressure such that no pressure is applied to the pressure ulcer. Ludwig Guttmann established a routine in the 1940s that entailed switching paraplegics every two hours to aid in the healing of bedsores. Such people used to have a two-year life expectancy, and their most common causes of death were blood and skin diseases. Guttmann had adopted the technique from the research of Boston physician Donald Munro. There is insufficient evidence to determine which positioning is more likely to avoid pressure sores—positioning the patient at 30 degrees or the conventional 90 degrees. Nursing homes and hospitals often have protocols in place to avoid the development of pressure ulcers in bedridden patients. Examples include twisting and repositioning on a regular schedule to relieve pressure. Turning and repositioning may take place more frequently depending on the degree of threat to the person [84].

2.7.2 **Support for Surface**: High-end, high-density foam mattresses are 60% less likely to cause new pressure sores than conventional foam mattresses, according to a 2015 Cochrane review. Sheepskin mattress protectors have also been said to lower the possibility of getting fresh pressure sores. There hasn't been much research done on the effectiveness of mattresses with alternating pressure. Pressure-redistributive mattresses are used to reduce excessive

pressure on the body's prominent or bony areas. Several important concepts are used to illustrate how these support surfaces function. This language was established by the NPUAP's Support Surface Standards Initiative. Numerous support surfaces distribute pressure by immersing and/or encasing the body. Examples of support surfaces with many alternately pumped air chambers are anti-decubitus mattresses and cushions. Techniques to standardize the products and evaluate their effectiveness have only recently been developed through the efforts of the S3I inside NPUAP. For disabled individuals, pressure sores can be avoided by routinely shifting pressure and using a wheelchair cushion with pressure relief features.

### 2.7.3 Nutrition

It's also important to get enough protein and calories. Vitamin C has been shown to reduce the risk of pressure ulcers. Others who are bedridden are less prone to get bed sores than people who take less vitamin C. Maintaining a suitable nutritional level is also necessary to prevent pressure ulcers in newborns. If one is unable to maintain adequate nutrition through protein and calorie consumption, it is advised to use supplements to support the proper nutrition levels. Skin maintenance is also important since damaged skin cannot withstand pressure. However, pressure ulcers are distinct from skin damage brought on by contact with excrement or urine. These skin wounds are classified as dermatitis related to incontinence.

A blood-filled blister or a purple or maroon patch of discoloured intact skin brought on by pressure or shear injury to the underlying soft tissue could indicate possible deep tissue damage. The region may be preceded by tissue that is painful, hard, mushy, swampy, warmer, or colder than the neighbouring tissue. People with a dark complexion may have difficulty recognizing significant tissue injury. A shallow wound bed could get a tiny blister on top of it. A thin layer of eschar may form over the wound as it progresses. Evolution could occur quickly, exposing new tissue layers even with the finest care.

## 2.8 Treatment of Pressure Ulcer

In partnership with wound organizations from 15 different countries, the NPIAP, EPUAP, and Pan Pacific Pressure Injury Alliance updated their global evidence-based clinical practice guideline in 2019. An international team of more than 180 clinical professionals developed the 2019 guideline, which updates the 2009 EPUAP/NPUAP clinical guideline and the 2014 NPUAP/EPUAP/PPPIA clinical guideline. The suggested techniques for treating pressure ulcers include bed rest, pressure-redistributing support surfaces, nutritional support, repositioning, wound care (such as debridement, and wound dressings), and biophysical agents (e.g. electrical stimulation). However, the use of many of these therapies is not supported by sufficient scientific research. More research is needed to find the best ways to help treat pressure ulcers, such as via repositioning. According to a Cochrane systematic review of randomized controlled trials published in 2020, additional research is needed to determine whether electrical stimulation is an effective pressure ulcer treatment. Furthermore, it is not yet known if either topical or internal antibiotics can help treat pressure ulcers. The excision of necrotic tissue is necessary for the majority of pressure ulcers. The heel is frequently an exception when there is the insufficient blood supply to the leg. Necrotic tissue supports bacterial growth, which can substantially impede wound healing. Five distinct techniques can be used to remove necrotic tissue.

1. Autolytic debridement is the gradual, often painless process of encouraging autolysis with the body's enzymes and white blood cells. It is most effective in those with healthy immune systems.

2. Biological debridement, often called maggot debridement therapy, involves deploying medical maggots to feed on decomposing tissue to rid a lesion of excess bacteria. Even though using live maggots as a medical device was long out of favour, the FDA approved it in January 2004.

3. Chemical debridement, also known as enzymatic debridement, is the procedure of eliminating necrotic tissue using prescribed enzymes.

4. Mechanical debridement involves utilizing debriding dressings, a whirlpool, or an ultrasound to remove slough from a stable wound.

5. Surgical debridement, sometimes referred to as sharp debridement, is the fastest method since it enables a surgeon to quickly remove dead tissue.

It remained unclear which topical drug or dressing was preferable for treating pressure ulcers, according to a 2017 Cochrane study. Gauze might not be as successful as foam dressings, protease-modulating dressings, or collagenase ointment in accelerating healing. The right wound dressing should be selected based on the wound and the condition of the surrounding skin. Some studies suggest that antibacterial drugs that encourage epithelization may improve wound healing. But there isn't a universal agreement on the best dressings for pressure ulcers. Cochrane reviews describe the research on alginate dressings, foam dressings, and hydrogel dressings. Due to a lack of reliable evidence, it is unknown whether these dressings are superior to alternative treatments.

**2.9 Factors at Risk of Pressure Ulcer**

There are more than 100 risk factors for pressure ulcers. A patient may be in danger due toconditions like immobility, diabetes mellitus, peripheral vascular disease, malnutrition, cerebral vascular accident, and hypotension. Having dry skin, a low body mass index, being incontinent of urine and/or faeces, being physically restrained, having a malignancy, and having a history of pressure ulcers are other risk factors[85]. People who are 70 years old or older and smokers in the past month are also at risk.

When lying down, the body can assume a broad variety of shapes and positions. The following is a list of the most well-known ones.

Supine is the term for lying on your back, face up, or on the ground.

Prone: lying on one's chest, face down ("lying down" or "going prone").

When lying on either side, the body can be straight or bent/curled forward or backwards.

The head and limbs are close to the knees and the body is curled up in a sitting or sleeping position in the fetal position. One of the variations of the lateral recumbent or three-quarters prone posture of the body that can be utilized to treat an unconscious but breathing sufferer is the recovery position (coma position). When a medical professional uses the word "decubitus" to describe a patient's position, they first specify the part of the body the patient is resting on. For instance, the patient would be in the right lateral decubitus posture while lying on the right side (RLDP). When a patient is lying on their left side, they are considered to be in the left lateral decubitus posture (LLDP)[86].

Angina decubitus, sometimes known as "chest soreness when lying down," is another example. In radiography, this expression means that the patient lies flat on their back and that the horizon is parallel as the X-ray is being taken.

### 2.9.1 **Hyperglycemia**

A group of metabolic diseases collectively referred to as diabetes mellitus is characterized by chronically increased blood sugar levels (hyperglycemia). Common symptoms include frequent urination, increased thirst, and increased hunger. If untreated, diabetes can cause a variety of health problems. Acute consequences include hyperosmolar hyperglycemia, diabetic ketoacidosis, and even death. Serious long-term effects include cardiovascular illness, stroke, chronic kidney disease, foot ulcers, eye damage, nerve damage, and cognitive impairment. Either insufficient insulin synthesis by the pancreas or incorrect insulin use by body cells causes diabetes. The task of enabling the entry of glucose from food into cells for cellular energy usage falls under the purview of the hormone insulin. The following are the

three main types of diabetes mellitus: The loss of beta cells in the pancreas results in an insufficient production of insulin, which causes type 1 diabetes. Previously, this type was referred to as "insulin-dependent diabetes mellitus" or "juvenile diabetes." The loss of beta cells is caused by an autoimmune response. The cause of this autoimmune reaction is unknown. Although Type 1 diabetes typically develops in childhood or adolescence, it can also affect adults. Type 2 diabetes is caused by insulin resistance, a condition in which cells do not respond to insulin as they should. As the condition gets worse, there can potentially be an insulin shortage. The term "adult-onset diabetes" or "non-insulin-dependent diabetes mellitus" was previously used to describe this type. Type 2 diabetes is more common in older people, but it has increased in prevalence in younger people due to a sharp rise in childhood obesity rates. The most prevalent cause is a combination of excessive body weight and insufficient activity.

Gestational diabetes, the third primary type of diabetes, affects pregnant women who have never had the condition. Blood sugar levels in pregnant women with gestational diabetes frequently return to normal after birth. Women who had gestational diabetes during pregnancy have a higher risk of developing type 2 diabetes later in life[86].

Type 1 diabetes can only be managed with insulin injections. Eating a balanced diet, exercising frequently, maintaining normal body weight, and quitting smoking can all help prevent and treat type 2 diabetes. Type 2 diabetes can be treated with oral anti-diabetic medications, either with or without insulin. Blood pressure must be under control, and patients must practice appropriate foot and eye hygiene. Oral medications and insulin can both produce low blood sugar levels (hypoglycemia). Weight loss surgery may be an effective treatment for type 2 diabetes. Once the baby is born, gestational diabetes typically disappears. By 2019, 463 million adults worldwide (8.8% of the adult population) were predicted to have diabetes, with type 2 diabetes making up more than 90% of cases. Similar rates apply to both

men and women. Trends indicate that rates will probably continue to rise. People with diabetes have a danger of dying young that is at least twice as high. Worldwide, 4.2 million people passed away from diabetes in 2019. It ranks as the sixth leading cause of death globally. The world economy was predicted to have lost $727 billion in 2017 due to diabetes-related medical expenses. Diabetes cost the US economy close to $327 billion in 2017. Spending on medical treatment is 2.3 times higher on average for diabetics[86].

**Signs and Symptoms of Diabetes Mellitus**

The common symptoms of uncontrolled diabetes include unintentional weight loss, polyuria (increased urine production), polydipsia (increased thirst), and polyphagia (increased hunger). In contrast to type 2 diabetes, where symptoms may develop more gradually or not at all, type 1 diabetes symptoms may occur abruptly (within weeks or months). Several additional indications and symptoms, though not particular to the disease, may point to the onset of diabetes. In addition to the previously described symptoms, they also include itchiness of the skin, blurred eyesight, headaches, and fatigue. Long-term high blood sugar levels can alter the shape of the eye's lens and impair vision because the lens absorbs glucose. Long-term loss of vision may occur as a result of diabetic retinopathy. A set of skin rashes known as diabetic dermadromes can appear in people with diabetes[87].

Diabetes patients may also have diabetic ketoacidosis (DKA), a metabolic disorder characterized by nausea, vomiting, and stomach discomfort as well as the acetone odour on the breath, Kussmaul breathing, and in extreme cases, a lowered level of consciousness (typically but not exclusively in type 1 diabetes). Emergency hospital care is necessary for DKA. A relatively rare but serious condition known as a hyperosmolar hyperglycemic state (HHS), which is more common in type 2 diabetes and mostly caused by dehydration brought on by high blood glucose, affects the body's ability to retain water.

Both type 1 and type 2 diabetics may experience hypoglycemia, a low blood sugar brought on by treatment, depending on the medication being taken. Most incidents are modest and not life-threatening. Uncomfort, shaking, sweating, and an increase in hunger are examples of symptoms that might occur in mild settings. However, in extreme circumstances, symptoms might include confusion, violent behaviour changes, seizures, unconsciousness, and, very rarely, fatalities or lasting brain damage. Rapid breathing, sweating, and cool, pale skin are signs of low blood sugar, however, these signs are not always present. Consuming food or drinks high in quickly absorbed carbohydrates is a self-treatment option for mild to severe cases. Instances that are severe enough to render a patient unconscious require intravenous glucose or glucagon injections for treatment.

All forms of diabetes carry a higher risk of long-term complications. These typically develop after several years (10–20), however, in those who haven't previously been diagnosed, they might be the first symptom. The main chronic problems are brought on by blood vessel injury. Diabetes increases a person's risk of cardiovascular disease by two times, and coronary artery disease is responsible for about 75% of their fatalities. Two other macrovascular diseases include stroke and peripheral artery disease[87]. These consequences are also a substantial risk factor for severe COVID-19 illness.

The three main effects of diabetes caused by damage to microscopic blood vessels are damage to the eyes, kidneys, and nerves. Diabetic retinopathy, or damage to the blood vessels in the retina of the eye, causes eye damage that can result in progressive vision loss and eventually blindness. Furthermore, diabetes increases the risk of glaucoma, cataracts, and other eye diseases. It's recommended that diabetics visit an eye doctor once a year. Injuries to the kidneys from diabetes, known as diabetic nephropathy, can result in tissue scarring, protein loss in the urine, and eventually chronic kidney disease, which may necessitate dialysis or kidney transplantation. Diabetes' most prevalent adverse impact, diabetic neuropathy, harms

the body's nerves. Skin damage may result in symptoms like numbness, tingling, sudomotor dysfunction, discomfort, and altered perception of pain. Diabetic foot problems, such as diabetic foot ulcers, can occur, are occasionally challenging to treat, and occasionally require amputation. Additionally, proximal diabetic neuropathy causes painful muscle atrophy and weakening. Cognitive decline and diabetes are connected. People with diabetes lose cognitive function 1.2–1.5 times more quickly than those without the disease. Falling occurs more frequently in elderly persons with diabetes, especially those taking insulin.

**Diabetes Mellitus Causes**

The six classifications of diabetes mellitus include type 1 diabetes, type 2 diabetes, hybrid forms of the disease, hyperglycemia that initially manifests during pregnancy, "unclassified diabetes," and "other special sorts." "Hybrid kinds of diabetes" include immune-mediated adult diabetes and type 2 diabetes which is prone to ketosis. Both gestational diabetes mellitus and pregnancy-related diabetes mellitus are regarded as "hyperglycemia first recognized during pregnancy" (type 1 or type 2 diabetes first diagnosed during pregnancy). The "other special categories" are made up of more than a dozen various elements. Diabetes is a more complicated condition than previously thought, and people might have multiple types. Only one kind of diabetes, diabetes mellitus, is simply referred to as "diabetes"[88].

Type 1 diabetes is characterized by the loss of the insulin-producing beta cells in the pancreatic islets, which leads to insulin insufficiency. Subtypes of this class include idiopathic and immune-mediated subtypes. The majority of type 1 diabetes cases are immune-mediated, in which beta cells and subsequently insulin are killed by an autoimmune attack mediated by T cells. It contributes to about 10% of cases of diabetes mellitus in North America and Europe. Most affected people are often healthy and at a healthy weight when symptoms first arise. Especially early on, insulin sensitivity and response are frequently normal. Because it usually affects children, type 1 diabetes has historically been referred to as "juvenile diabetes,"

however the majority of patients with the condition today are adults. In the past, the term "brittle" diabetes—also referred to as unstable diabetes or labile diabetes—was used to describe the sharp and regular variations in blood sugar levels that are common in insulin-dependent diabetes and frequently happen for no apparent reason. However, because it lacks a biological basis, this expression shouldn't be used. But type 1 diabetes can also manifest as dangerously low blood sugar levels, unpredictable and erratic high blood sugar levels, and the risk of diabetic ketoacidosis. Additional effects include infection, gastroparesis (which results in abnormal dietary carbohydrate absorption), and endocrinopathies such as Addison's disease. Another is a compromised counterregulatory response to low blood sugar. It is estimated that only 1% to 2% of persons with type 1 diabetes regularly encounter these occurrences.

Numerous genes, including specific HLA genotypes, are known to influence the likelihood of developing type 1 diabetes. In people who are genetically predisposed to the disease, diabetes can develop as a result of one or more environmental factors, such as a viral infection or nutrition. Although other viruses have been mentioned, there is currently insufficient evidence to support this notion in humans. The specific mechanism through which gliadin, a protein present in gluten, may contribute to the formation of type 1 diabetes through dietary factors is uncertain. Type 1 diabetes can strike at any age, and most cases are identified in adults. Latent autoimmune diabetes of adults (LADA), which manifests more gradually than type 1 diabetes in children, is the diagnosis made when the condition affects an adult. Some people refer to this condition as "type 1.5 diabetes" because of this distinction. Adults with LADA are frequently initially misdiagnosed as having type 2 diabetes because of their age rather than a reason.

Type 2 diabetes has two characteristics, including insulin resistance and perhaps decreased insulin production. The reduced sensitivity of body tissues to insulin is believed to be caused in part by the insulin receptor. However, the specific problems are still a mystery. Diabetes

mellitus cases that have a known defect are categorized differently. Kind 2 diabetes mellitus is the most common type. Many people with type 2 diabetes exhibit symptoms of prediabetes (impaired fasting glucose and/or impaired glucose tolerance) before meeting the criteria for the condition. The development of prediabetes into overt type 2 diabetes can be stopped or reversed by lifestyle modifications or medications that improve insulin sensitivity or reduce the liver's production of glucose. Genetics and lifestyle decisions are the primary causes of type 2 diabetes. Numerous lifestyle factors have been linked to type 2 diabetes, such as obesity (defined as a body mass index of greater than 30), inactivity, poor diet, stress, and urbanization. 30% of cases are attributed to people with Chinese or Japanese heritage, 60% to 80% to people with European or African ancestry, and 100% to Pima Indians and residents of the Pacific Islands. Any weight individual can have an elevated waist-hip ratio. Foods and drinks with added sugar have been linked to an increased risk. The kind of lipids consumed is also important, with saturated and trans fats increasing risk while polyunsaturated and monounsaturated fats reduce it. If you consume too much white rice, especially if you are Chinese or Japanese, your risk of having diabetes may increase. If they don't exercise, some people may be more likely to acquire diabetes. Neglect has the biggest effect, although adverse childhood experiences like abuse, neglect, and issues at home all increase the risk of type 2 diabetes by 32% in adults. Unhealthy lifestyles (such as poor nutrition and minimal exercise), metabolic anomalies, dyslipidemia, and weight gain are potential risk factors for antipsychotic drug side effects.

There are several similarities between type 2 diabetes and gestational diabetes, one of which is a combination of relatively low insulin secretion and responsiveness. It occurs in 2–10% of pregnancies and could get better or disappear after delivery. All expecting mothers are encouraged to get tested starting between 24 and 28 weeks of gestation. It is usually found in the second or third trimester due to the surge in insulin-antagonist hormone levels that occurs at this time. However, it is shown that 5 to 10% of women with gestational diabetes also have

another kind of diabetes, typically type 2, after giving birth. Although entirely controllable, gestational diabetes necessitates constant medical attention. Possible management techniques include dietary adjustments, blood glucose monitoring, and, in rare cases, the use of insulin. Even though it may only be transient, untreated gestational diabetes can be harmful to the mother's or the fetus's health. The unborn child is at risk for macrosomia (large birth weight), congenital heart and central nervous system abnormalities, and skeletal muscle malformations. The development of fetal surfactant may be inhibited by elevated insulin levels in a fetus' blood, leading to neonatal respiratory distress syndrome. An increased blood bilirubin level may be caused by red blood cell deterioration. Extreme circumstances can lead to perinatal mortality, which most usually happens as a result of insufficient placental perfusion brought on by vascular injury. A symptom of labour induction could be diminished placental function. A cesarean section may be performed if there is substantial fetal discomfort or a higher risk of injury from macrosomia, such as shoulder dystocia.

Mature-onset diabetes of the young (MODY), an unusual autosomal dominant hereditary type of diabetes, is caused by one of many single-gene abnormalities that hinder insulin production. It occurs in only 1% to 2% of all cases, which is a far lower percentage than the three major categories. The name of the illness alludes to early hypotheses regarding its composition. Since the age at which this gene-related sickness manifests itself and its severity varies depending on the specific gene defect, there are at least 13 different subtypes of MODY. MODY sufferers frequently control it without insulin.

Certain types of diabetes are brought on by the body's tissue receptors failing to respond to insulin, even when insulin levels are normal, which makes them distinct from type 2 diabetes. This type of diabetes is incredibly uncommon. Genetic mutations can cause defects in beta cell functionality (autosomal or mitochondrial). In some cases, aberrant insulin activity could also have a hereditary component. Any disorder that badly affects the pancreas can lead to

diabetes (for example, chronic pancreatitis and cystic fibrosis). Diabetes can be caused by conditions that release insulin-antagonistic hormones too frequently (which is typically resolved once the hormone excess is removed). While some toxins damage pancreatic beta cells and some drugs decrease the generation of insulin, other chemicals increase insulin resistance (especially glucocorticoids which can provoke "steroid diabetes"). When the current taxonomy was adopted in 1999, the World Health Organization (WHO) advised against using the diagnostic phrase malnutrition-related diabetes mellitus (ICD-10 code E12). Another form of diabetes that some people may develop is double diabetes. This happens when a type 1 diabetic acquires insulin resistance, a trait that distinguishes type 2 diabetes, or if type 2 diabetes runs in the family. The initial finding was made around 1990 or 1991.

Insulin is the primary hormone that regulates the uptake of glucose from the blood into the majority of body cells, including the liver, adipose tissue, and muscle, except smooth muscle, where insulin functions through IGF-1[96]. Therefore, an inadequate supply of insulin or the insensitivity of its receptors are the underlying causes of all forms of diabetes mellitus.

The three main sources of glucose for the body are the digestion of food, the breakdown of glycogen (glycogenolysis), the liver's form of glucose storage, and gluconeogenesis, the body's method of creating glucose from non-carbohydrate substrates. Insulin is crucial for maintaining glucose levels within the body. The process of gluconeogenesis as well as the transport of glucose into fat and muscle cells may both be accelerated by insulin. Additionally, it can hasten the process by which glucose is stored as glycogen. The pancreatic islets of Langerhans contain beta cells, sometimes referred to as -cells, which release insulin into the blood in response to elevated blood glucose levels, typically following meals. Insulin is used by almost two-thirds of all body cells to remove glucose from circulation for use as fuel, as a building block for other molecules that are required, or for storage. Low glucose levels result in decreased insulin production from beta cells and the conversion of glycogen to glucose.

This process is heavily regulated by the hormone glucagon, which competes with insulin in the body. If there is not enough insulin available, cells do not respond well to the effects of insulin (insulin resistance), or the insulin itself is faulty, the body's cells that need glucose cannot properly absorb it and it cannot be properly stored in the liver and muscles. In general, this results in persistently high blood glucose levels, insufficient protein synthesis, and other metabolic problems, including metabolic acidosis in conditions of severe insulin deficiency. The kidneys approach a reabsorption threshold and the body excretes glucose in the urine when blood sugar levels are continuously high (glycosuria). As a result of the kidneys' inability to reabsorb water, there is an increase in the osmotic pressure of the urine, increased polyuria, and more fluid loss. The osmotically restored lost blood volume, which comes from water in body cells and other compartments, causes dehydration and increased thirst (polydipsia). Low intracellular glucose also makes people more hungry, which leads to overeating (polyphagia).

There were 425 million people with diabetes worldwide in 2017, up from 382 million in 2013 and 108 million in 1980. The prevalence of diabetes among adults is 8.8%, which is more than twice as high as the rate of 4.7 per cent in 1980. This is when the shifting age distribution of the global population is taken into account. The data shows that Type 2 instances make up almost 90% of all cases. According to some data, rates for both sexes are nearly similar, but in many populations with higher type 2 incidence, there is a male excess in diabetes. This might be a result of sex-related variations in insulin sensitivity, the impact of obesity and localized body fat deposition, and other risk factors such as high blood pressure, smoking, and alcohol use.

The WHO estimates that diabetes killed 1.5 million people in 2012, making it the ninth leading cause of death. 2.2 million additional deaths globally were directly linked to high blood sugar, the increased risks of cardiovascular disease, and other connected problems, even

though diabetes is typically reported as the main cause of death on death certificates (such as kidney failure). Premature death is frequently brought on by these factors. For instance, the International Diabetes Federation (IDF) employed modelling to estimate the overall number of fatalities that may have been caused by diabetes either directly or indirectly in 2017. According to their estimates, diabetes killed 4.0 million people worldwide.

Even though diabetes can occur anywhere in the world, more developed countries have a higher prevalence of the disease, particularly type 2. The highest rate increases have been seen in low- and middle-income countries, where more than 80% of diabetes fatalities occur. The majority of diabetics are estimated to live in Asia and Africa in 2030, where the incidence is anticipated to increase at the fastest rate. The global nutrition transition, which is defined by increased intake of foods that are high in calories but low in nutrients, the trend of urbanization, sedentary lifestyles, fewer physically demanding professions, and the rise in rates in emerging nations are all significant reasons (often high in sugar and saturated fats, sometimes referred to as the "Western-style" diet). There may be a 48 per cent increase in diabetes incidence worldwide between 2017 and 2045 [88].

**Diabetes Mellitus Management and Prevention**

There is no proven prophylactic treatment for type 1 diabetes. Type 2 diabetes, which makes up 85–90% of cases worldwide, can frequently be avoided or delayed by maintaining normal body weight, engaging in regular exercise, and eating a healthy diet. Increased physical activity lowers diabetes risk by 28%. (greater than 90 minutes each day). Dietary changes that are effective in preventing diabetes include choosing healthy fats such as the polyunsaturated fats found in fish, nuts, and vegetable oils as well as maintaining a diet high in whole grains and fibre. Reducing sugary beverages, eating less red meat, and limiting other sources of saturated fat will help avoid diabetes. Because smoking is associated with an increased risk of developing diabetes and its complications, quitting smoking can also be an important

preventative measure. There is a connection between type 2 diabetes and the main reversible risk factors everywhere in the world (overweight, poor nutrition, inactivity, and tobacco use). There is growing evidence that the underlying causes of diabetes are influenced by the main factors causing social, economic, and cultural change—globalization, urbanization, population ageing, and the general health policy environment.

The goal of diabetes treatment is to keep blood sugar levels as close to normal as possible without actually lowering them. Usually, this can be accomplished by changing your diet, getting more exercise, lowering weight, and using the correct drugs (insulin, oral medications). Since difficulties are substantially less frequent and less severe in people with well-controlled blood sugar levels, it is essential to educate oneself about the condition and actively participate in therapy. The American College of Physicians recommends aiming for a therapeutic HbA1C level of 7-8%. There is additional thought given to other medical conditions that can hasten the negative effects of diabetes. These include being overweight, having metabolic syndrome, having high blood pressure, smoking, and not exercising frequently. Many patients with at-risk diabetic feet utilize specialized footwear to lessen their risk of developing ulcers, even though there is conflicting evidence regarding its efficacy. Similar principles may be used to control diabetes in the general population, however, there may be significant considerations when tailoring therapies, particularly for particular groups. Insufficient research has been done to determine whether type 2 diabetes self-management programs for people with severe mental illness are effective in producing results that are equivalent to those seen in the general population.

People with diabetes can benefit from the knowledge of the condition and its treatments, dietary changes, and exercise to maintain both short-term and long-term blood glucose levels within acceptable ranges.

In addition, because they are linked to higher risks of cardiovascular disease, lifestyle adjustments are advised to lower blood pressure. Weight loss can reduce the risk of heart disease, halt the development of type 2 diabetes from prediabetes, or induce partial remission in diabetics. No one diet works best for diabetics overall. It is frequently recommended to adhere to healthy eating patterns like the Mediterranean diet, low-carb diet, or DASH diet, even though research does not favour one healthy eating plan over another. For persons with type 2 diabetes who cannot fulfil the glycemic objectives or if minimizing anti-glycemic drugs is a priority, low or very-low-carbohydrate diets are a possible choice. This is so that the ADA's claim that "lowering overall carbohydrate intake for those with diabetes has provided the most evidence for improving glycemia" may be supported. Type 2 diabetics who are overweight can benefit from any diet that helps them lose weight. Most diabetes medications act by lowering blood sugar levels in different ways. There is general agreement that people with diabetes who maintain strict glucose control, keeping the glucose levels in their blood within normal ranges, reduce complications like kidney or eye issues. For people who are older and may have a higher risk of hypoglycemia, it is questionable if this is appropriate and cost-effective. Anti-diabetic medications can be divided into many different kinds. Type 1 diabetes must be managed with insulin, and the "basal-bolus" regimen (long-acting insulin for the basal rate and short-acting insulin with meals) is advised as it most closely resembles the release of insulin from the body naturally. The majority of persons with type 2 diabetes receive treatment orally (with drugs like metformin), but others eventually require intravenous administration of insulin or GLP-1 agonists. Metformin is frequently recommended as the first-line treatment for type 2 diabetes since there is compelling evidence that it lowers mortality. It reduces the quantity of glucose the liver produces to work. People with type 2 diabetes may also benefit from other pharmaceutical classes, the majority of which are taken orally, in lowering blood sugar levels. Some of these include thiazolidinediones, acarbose, sitagliptin, sulfonylureas, the enzyme inhibitor dipeptidyl

peptidase-4 (DPP-4) that inactivates the incretins GLP-1 and GIP, medicines that stimulate insulin release, and drugs that increase the excretion of glucose in the urine (SGLT2 inhibitors). When treating type 2 diabetes with insulin, a long-acting formulation is usually used at first while oral medications are maintained. Once the glucose targets are reached, the insulin doses are increased. Numerous international standards recommend treating blood pressure targets for diabetics that are lower than 140/90 mmHg due to the significant risk of cardiovascular disease. However, there is little evidence to back up the suggested numbers for the lower targets. Despite a higher risk of adverse events, further systematic analysis in 2019 found no evidence of additional benefits from blood pressure lowering to targets between 130 and 140 mmHg. Treating goals lower than 140 mmHg may cause damage, according to a 2016 systematic review. To reduce their risk of developing end-stage renal disease, experiencing a cardiovascular event, or passing away, people with diabetes and albuminuria should take a renin-angiotensin system inhibitor, according to recommendations made by the American Diabetes Association in 2015. There is some evidence that renin-angiotensin system inhibitors like aliskiren and angiotensin receptor blockers are less effective than ACEIs for preventing cardiovascular disease (ARBs). However, a more recent analysis found that the effects of ACEIs and ARBs on significant cardiovascular and renal outcomes are comparable. No evidence combining ACEIs and ARBs offers any extra benefits over other diabetes management strategies.

### 2.9.2 Peripheral Artery Disease

Peripheral arterial disease is characterized by abnormal constriction of arteries other than those that supply blood to the heart or brain (PAD). The terms "narrowing" that affects the heart or the brain are both used for coronary artery disease and cerebrovascular illness. Peripheral artery disease can affect any artery, including those in the arms, neck, or kidneys, even though it most usually affects the legs. The name for the symptom, which is walking-

related leg pain that goes away with rest, is intermittent claudication. Additional symptoms include skin ulcers, bluish skin, cold skin, or uneven nail and hair growth in the affected leg. Examples of problems include infections, tissue death that can need amputation, coronary artery disease, or stroke. Up to 50% of PAD patients don't have any symptoms.

The primary risk factor for PAD is smoking. Additional risk factors include diabetes, high blood pressure, kidney problems, and high blood cholesterol. The most common underlying cause of peripheral artery disease, particularly in persons over 40, is atherosclerosis. Other mechanisms include vasculitis, fibromuscular dysplasia, trauma, blood clots, atherosclerosis, and blood clots. Ankle-brachial index (ABI), which is less than 0.90, is frequently used to diagnose PAD. It is calculated as the systolic blood pressure at the ankle divided by the systolic blood pressure in the arm. Other options include duplex ultrasound and angiography. Angiography is more accurate and allows for concurrent therapy, but it also comes with increased risks.

It is unknown whether screening for peripheral artery disease in persons without symptoms is advantageous because the matter has not been properly investigated. People with intermittent claudication brought on by PAD benefit more from giving up smoking and obtaining supervised exercise therapy. Additionally useful medications include statins, ACE inhibitors, and cilostazol. Due to the increased risk of heart attacks, aspirin is often recommended for people with more severe illnesses, but it does not seem to help individuals with milder illnesses. One anticoagulant that frequently offers minimal benefit is warfarin. Treatment options for the condition include atherectomy, angioplasty, and bypass grafting.

In 2015, over 155 million people had PAD globally. It becomes more common as we become older. In the industrialized world, it affects around 5.3% of persons in their 45th to 50th year and 18.6% of adults in their 85th to 90th year. In developing nations, it affects 4.6% of persons between the ages of 45 and 50 and 15% of adults between the ages of 85 and 90. In

the industrialized world, men and women both experience PAD at about the same rates, however in the developing world, women are more prone to encounter it. PAD has grown, from 16,000 deaths in 1990 to almost 52,500 deaths in 2015.

**PAD Symptoms and Signs**

The signs and symptoms of peripheral artery disease depend on the area of the body that is affected. In about 66 per cent of PAD patients, there are either no symptoms at all or strange symptoms. The most frequent presenting symptom is intermittent claudication, which causes agonizing pain and cramping when moving or exercising. Normal resting reduces the pain, which is often felt in the damaged leg's calf muscles. This occurs because exercise causes the muscles in the legs to use up more oxygen. Normally, the arteries would be able to improve blood flow, increasing the amount of oxygen that reached the exercised leg. The artery, however, is unable to respond appropriately to the muscles' increased need for oxygen when they have PAD. Leg muscles become overly lactic acid-saturated as a result, causing pain that only goes away with rest.

Additional symptoms could include hip, thigh, or buttock pain, pains, or cramps; loss of muscle mass in the affected limb's muscles; hair loss on the affected limb; touchably cold, glossy, or smooth skin; diminished or absent pulse in the feet; numbness or coldness in the toes; and unhealed wounds or ulcers on the affected limb.

In patients with severe PAD, complications like gangrene and critical limb ischemia might happen. Critical limb ischemia occurs when the blood flow obstruction in the artery is so severely compromised that the blood is unable to maintain the oxygenation of tissue at rest. In addition to the pain that gets worse with rest, this may cause numbness or tingling in the foot and toes. Additional side effects of severe PAD include gangrene, sores caused by vascular insufficiency, sexual dysfunction, and lower limb tissue loss. Diabetes patients get foot

gangrene at a rate that is 30 times higher than the normal population. Many of these grave repercussions have long-lasting ramifications.

**PAD Risk Factors**

The risk factors for PAD are the same as those for atherosclerosis. These include age, sex, and ethnicity. Males are two times more likely to develop PAD than females. The prevalence of PAD differs by a factor of 2:1 between people of colour and people of European descent. The strongest risk associations are found in hyperlipidemia, hypertension, diabetes mellitus, chronic renal illness, and smoking. When three or more of these factors are present, the likelihood of getting PAD increases tenfold. Smoking—tobacco use in any form—is the single greatest risk factor for peripheral vascular disease worldwide. Smokers may have a 10-fold increase in their risk of PAD, according to a dose-response correlation. Additionally, it has been shown that exposure to secondhand smoking promotes changes in the blood vessel's endothelium, which can lead to atherosclerosis. Smokers are 2-3 times more likely to develop lower extremity PAD than coronary artery disease. Smokers make up more than 80% to 90% of people with peripheral vascular disease of the lower limbs. The risk of PAD increases as cigarettes are smoked more frequently and for longer periods of time[89].

Diabetes mellitus has been associated with a 2-4 times greater risk of PAD than high blood sugar. It does this by reducing the activity of smooth muscle and endothelial cells in the peripheral arteries. Peripheral vascular disease in the lower limbs is closely connected with the severity and duration of diabetes. The medical word for an extremely high level of fat or cholesterol is dyslipidemia, sometimes known as high blood cholesterol. The causes of dyslipidemia include a rise in total cholesterol and/or high triglyceride levels, low levels of the protein known as high-density lipoprotein (HDL cholesterol), low levels of the protein known as low-density lipoprotein (LDL cholesterol), and/or high levels of LDL cholesterol. This aberration in blood cholesterol levels has been connected to accelerated peripheral

vascular disease. The management of dyslipidemia with diet, exercise, and/or medication is associated with a considerable decrease in the risk of heart attack and stroke. Hypertension - Hypertension, or high blood pressure, can increase a person's risk of developing PAD. Similar to PAD, high blood pressure has been linked to an increased risk of heart attacks, strokes, and abdominal aortic aneurysms. The most common PAD symptom, intermittent claudication, is 2.5–4 times more common in men than in women with high blood pressure. The other risk factors being studied include levels of many inflammatory mediators like C-reactive protein, fibrinogen, homocysteine, and lipoprotein A. People with greater blood homocysteine levels are twice as likely to develop the peripheral vascular disease. Despite the genetics of risk factors for the disorder such as diabetes and high blood pressure, no specific genes or gene mutations have been directly connected to the onset of peripheral artery disease.

The diagnosis or identification of peripheral artery disease requires a physical exam, a history of symptoms, and confirmation tests. These tests may include imaging ultrasounds, computed tomography scans, or magnetic resonance angiography (MRA) scans (CT). If a patient displays symptoms of peripheral artery disease, a doctor will then conduct specific exam findings on them. If an atypical physical exam result is found, a medical expert might consider a particular diagnosis. But to confirm a diagnosis, confirmatory testing is required. These findings indicate a link to peripheral vascular disease:

• Pulses that are diminished or absent

• Muscle atrophy or loss

• The wounded limb's blueness can be seen.

• The affected limb's temperature is lower than the other limb's (feels cooler).

• overgrown nails

• Hair loss and shiny or smooth skin

Buerger's test can identify pallor when the damaged limb is elevated. Next, the affected limb is moved from the elevated position to a sitting position and checked for reactive hyperemia, which is characterized by redness. The Buerger's test measures arterial sufficiency, or the ability of the artery to deliver oxygen-rich blood to the tissue it supplies [90].

The wound on the lower extremity that is not healing

Whenever there is a possibility of peripheral vascular disease, the ankle-brachial index should be the initial test carried out (ABI). The ankle blood index (ABI) measures the difference between the systolic blood pressure in the ankle and the upper arm. It is a rapid, non-invasive examination. This is based on the idea that if blood pressure readings in the ankle are lower than those in the arm, a blockage in the arteries sending blood from the heart to the ankle is likely to be the cause.

An ABI of 0.90 to 1.40 is considered to be typical. If a person's ABI is less than 0.90, they are said to have PAD. However, PAD can also be categorized as mild to moderate if the ABI is between 0.41 and 0.90 and as severe if it is less than 0.40. These associations can provide insight into the course of a disease. ABI values more than 1.40 indicate non-compressible arteries, whereas values between 0.91 and 0.99 are considered borderline. ABI calculations over 1.40 may indicate the stiffness of the vessel wall brought on by calcification, which can occur in people with uncontrolled diabetes. ABIs that are very high (>1.40) are frequently considered to be false negatives; as a result, such findings demand further investigation and higher-level studies [49]. In the next two years, cardiovascular diseases are more likely to claim the lives of people with non-compressible arteries.

Patients with normal ABIs and suspected PAD can test their ABIs while working out. An ABI baseline is taken before to activity. The patient is then instructed to exercise for up to five minutes until claudication discomfort appears (typically, patients are made to walk on a treadmill at a steady speed). At this time, the ankle pressure is once again measured. To

diagnose PAD, an ABI decline of 15% to 20% would be required a foot or leg if the ABIs are abnormal, the next step is usually Doppler ultrasonography to locate the obstruction and assess the degree of atherosclerosis. Further imaging can be done using angiography, which entails putting a catheter into the common femoral artery and carefully guiding it to the target artery. While a radio-dense contrast chemical is injected, an X-ray is taken. Any blockage that limits blood flow that was visible on the X-ray can be found and treated using procedures like atherectomy, angioplasty, or stenting. Contrast angiography is the imaging technique that is the most accessible and well-liked. Modern computerized tomography (CT) scanners provide direct imaging of the vascular system as an alternative to angiography. The non-invasive diagnostic procedure known as magnetic resonance angiography, or MRA, uses a large magnet, radio waves, and a computer to produce exact images of the blood veins inside the body. Benefits of MRA include its ability to give thorough, high-resolution imaging of the abdomen, pelvis, and lower extremities in a single sitting while also being safe.

Pressure ulcers can develop almost anywhere on the body, they are most frequently found over the lower trunk's bony structures, such as the sacrum, greater trochanter, and ischium (cumulative figure: 45.9%), followed by the heels and malleolus[97] (cumulative figure: 34% 3). (Figure 2). Some of the worst injuries involve the sacrum and the heel. The risk of eventual amputation after heel ulcers may reach 42% if there is underlying vascular disease of the limb[98]. These two anatomical sites typically account for the majority of pressure ulcers, even though numbers vary depending on the clinical specialty, and they thus represent a crucial area for preventative care. Because these wounds can affect anyone, from the very young or temporarily incapacitated to the very old and infirm, pressure ulceration risk does not discriminate based on age, gender, or ethnicity.

The severity of a pressure ulcer is not always immediately obvious; for instance, some pressure ulcers may start in the deep tissue underneath healthy skin (suspected deep tissue

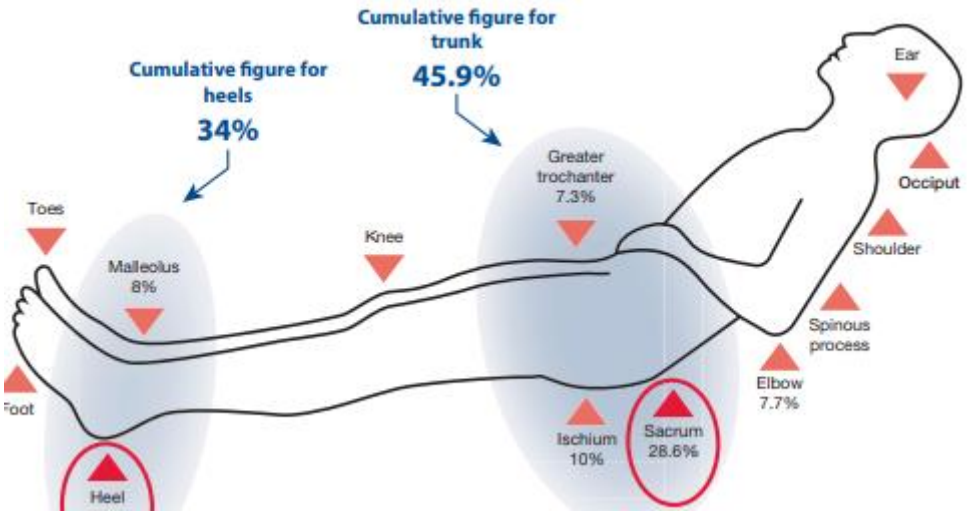
injury)[99]. Others may have slough and/or eschar covering the wound bed; these wounds are reported as unstageable up until the point at which the wound bed is visible. FIGURE 2: Diagram of the major anatomical sites linked to the occurrence of pressure ulcers 45.9% 34% Cumulative heel gauge Combined figure for the trunk rumen 28.6% Elbow 7.7% Spinal mechanism Higher trochanter in the ear 7.3% Knuckle Malleolus 8% Toes Heel 26% Category/Stage II: Partial Thickness of the Foot Shoulder Occiput Ischium 10% Pressure Ulcer Classification System Skin Decay Presented as a shallow open ulcer with a reddish-pink wound bed and no slough, the dermis has lost some of its thickness. Also possible to see an intact or open/ruptured blister filled with serum. presents as a superficial, glossy or dry ulcer that is not sloughed or bruised. Skin rips, tape burns, perineal dermatitis, maceration, or excoriation are not appropriate terms to use to describe this Category/Stage. Bruising may be a sign of significant tissue damage.

**System for Classification of International Npuap/Epuap Pressure Ulcer**

In comparison to neighboring tissue, the region may be painful, hard, soft, warmer, or cooler. People with dark skin tones may have a hard time being diagnosed with Category/Stage I. People who are "at risk" may be indicated (a heralding sign of risk). Stage III (Category): Full Thickness Skin Decay loss of tissue in its whole. Bone, tendon, or muscles are not revealed, although subcutaneous fat may be apparent. Slough might be visible, but it doesn't hide how much tissue has been lost. includes digging tunnels and undermining. The depth of a Category/Stage III pressure ulcer varies by anatomical location. The bridge of the nose, ear, occiput and malleolus do not have subcutaneous tissue and Category/Stage III ulcers can be shallow. In contrast, areas of significant adiposity can develop extremely deep Category/Stage III pressure ulcers. Bone/tendon is not visible or directly palpable. Stage IV: Full Thickness Tissue Loss (Category/Stage IV) tissue loss in its entirety with exposed bone, tendon, or

muscle. Some areas of the wound bed may have eschar or slough include tunneling and undermining frequently.

The anatomical location affects how deep a Category/Stage IV pressure ulcer is. These ulcers can be shallow because there is no subcutaneous tissue in the occiput, malleolus, bridge of the nose, or ear. Osteomyelitis may result from Category/Stage IV ulcers that spread into muscles and/or supporting tissues (such as fascia, tendons, or joint capsule). Exposed bone or a tendon is discernible or palpable[100]. Unstageable: Unknown Depth Full thickness tissue loss with slough (yellow, tan, gray, green, or brown) and/or eschar (tan, brown, or black) covering the base of the ulcer in the wound bed[101]. The exact depth of the wound, and thus its Category/Stage, cannot be ascertained until enough slough and/or eschar have been removed to reveal its base. The body's natural (biological) coating is represented by stable (dry, adhesive, unbroken, without erythema or fluctuance) eschar on the heels, which should not be removed. Deep Tissue Injury Suspected: Unknown Depth Purple or maroon localized patch of discolored intact skin or blood-filled blister as a result of pressure and/or shear injury to the underlying soft tissue. Compared to nearby tissue, the area may be preceded by tissue that is painful, firm, mushy, swampy, warmer, or cooler. Dark-skinned people may have trouble identifying deep tissue injuries. A small blister may develop on top of a deep wound bed. The wound might develop further and develop a thin layer of eschar over it. Even with the best care, evolution might be quick, exposing new tissue layers, minimizing avoidable damages. Clinicians are becoming more aware of the risk of pressure ulcers associated with medical devices like splints, traction, respiratory support, and anti-embolic stockings, which are collectively referred to as device related pressure ulcers, in addition to the risk posed to patients when lying or sitting. According to recent research, people who use an additional medical device may be up to 2.4 times more likely than those who do not to suffer a pressure-related injury.



## Endnotes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## Chapter Three

## Methodology

## 3.0 Study Design

The research was a systematic review that includes a cross-sectional study that involves the prevalence and risk factors of pressure ulcers in Sub-Saharan Africa. The study was conducted in September 2022. The search range was from January 1990 to August 2022.

## 3.1 Keywords

Pressure Ulcers, Bedsores, Pressure Sore, Sore Bed, Decubitus Ulcer, Ulcer Decubitus and Prevalence, Period Prevalence, Point Prevalence and Factor Risk,Risk Factor, Social Risk Factors, Health Correlates, Population at Risk

## 3.2 Study Population

The study population comprised of studies on the prevalence and risk factors of pressure ulcers in sub-Saharan Africa

### 3.2.1 Inclusion Criteria

Studies that report on pressure ulcers among children, adolescents and adults in sub-Saharan Africa. Studies that are conducted in the hospital, long-term care, and community surveys will be included (including patients within any given age group). Studies that report on the prevalence, risk factors, preventive strategies, and anatomical positions and gave a sample size.

### 3.2.2 Exclusion Criteria

Studies that do not have prevalence data, risk factors or where the sample size was not given will be excluded.

## 3.3 Search Terms

1. **Pressure ulcer (MeSH)**

   Ulcers, Pressure

Sore, Bed

Bedsores

Decubitus Ulcer

Pressure Ulcers

Sores, Pressure

Bedsore

Ulcers, Decubitus

Sores, Bed

Pressure Sore

Pressure Sores

Ulcer, Pressure

Ulcer, Decubitus

Sore, Pressure

Bed Sore

Bed Sores

Decubitus Ulcers

**2. Prevalence (MeSH)**

Prevalences

Period Prevalence

Period Prevalences

Prevalence, Period

Point Prevalence

Point Prevalences

Prevalence, Point

**3. Risk Factors (MeSH)**

Factor, Risk

Risk Factor

Social Risk Factors

Factor, Social Risk

Factors, Social Risk

Risk Factor, Social

Risk Factors, Social

Social Risk Factor

Health Correlates

Correlates, Health

Population at Risk

Populations at Risk

Risk Scores

Risk Score

Score, Risk

Risk Factor Scores

Risk Factor Score

Score, Risk Factor

4. **Children (MeSH)**

   Child

5. **Adolescent (MeSH)**

Adolescents

Adolescence

Teens

Teen

Teenagers

Teenager

Youth

Youths

Adolescents, Female

Adolescent, Female

Female Adolescent

Female Adolescents

Adolescents, Male

Adolescent, Male

Male Adolescent

Male Adolescents

6. **Adult (MeSH)**

   Adult

7. **Sub-Saharan Africa (MeSH)**

   Sub-Saharan Africa

   Africa, Sub-Saharan

### 3.4.1 Search Strategy

A systemic review of primary research will be undertaken and the prevalence and risk factors of pressure ulcers will be determined based on their scores on the pressure ulcer prevalence, risk factors and the anatomical location assessment materials (questionnaire). In terms of language, only the articles published in English will be included in the analysis. The search will be conducted in electronic databases such as Google Scholar, PubMed, and Cochrane Library using the following MeSH terms: “Pressure Ulcers”, “Bedsores”, “Pressure Sore”, “Sore Bed”, “Decubitus Ulcer”, “Ulcer Decubitus” and “Prevalence”, “Period Prevalence”, “Point Prevalence” and “ Factor Risk”, “Risk Factor”, “Social Risk Factors”””” Health Correlates”, “Population at Risk” search will be carried out from January 1st 1990 to August 1st 2022.

The full original search terms in PUBMED are:

((("pressure ulcer"[MeSH Terms] OR ("pressure"[All Fields] AND "ulcer"[All Fields]) OR "pressure ulcer"[All Fields] OR ("ulcers"[All Fields] AND "pressure"[All Fields]) OR "ulcers pressure"[All Fields] OR ("pressure ulcer"[MeSH Terms] OR ("pressure"[All Fields] AND "ulcer"[All Fields]) OR "pressure ulcer"[All Fields] OR ("sore"[All Fields] AND "bed"[All Fields]) OR "sore bed"[All Fields]) OR ("pressure ulcer"[MeSH Terms] OR ("pressure"[All Fields] AND "ulcer"[All Fields]) OR "pressure ulcer"[All Fields] OR "bedsore"[All Fields] OR "bedsores"[All Fields]) OR ("pressure ulcer"[MeSH Terms] OR ("pressure"[All Fields] AND "ulcer"[All Fields]) OR "pressure ulcer"[All Fields] OR ("decubitus"[All Fields] AND "ulcer"[All Fields]) OR "decubitus ulcer"[All Fields]) OR ("pressure ulcer"[MeSH Terms] OR ("pressure"[All Fields] AND "ulcer"[All Fields]) OR "pressure ulcer"[All Fields] OR ("pressure"[All Fields] AND "ulcers"[All Fields]) OR "pressure ulcers"[All Fields]) OR ("pressure ulcer"[MeSH Terms] OR ("pressure"[All Fields] AND "ulcer"[All Fields]) OR "pressure ulcer"[All Fields] OR ("sores"[All Fields] AND "pressure"[All Fields]) OR "sores pressure"[All Fields]) OR ("pressure ulcer"[MeSH Terms] OR ("pressure"[All Fields] AND "ulcer"[All Fields]) OR "pressure ulcer"[All Fields] OR "bedsore"[All Fields] OR "bedsores"[All Fields]) OR ("pressure ulcer"[MeSH Terms] OR ("pressure"[All Fields] AND "ulcer"[All Fields]) OR "pressure ulcer"[All Fields] OR ("ulcers"[All Fields] AND "decubitus"[All Fields]) OR "ulcers decubitus"[All Fields]) OR ("pressure ulcer"[MeSH Terms] OR ("pressure"[All Fields] AND "ulcer"[All Fields]) OR "pressure ulcer"[All Fields] OR ("sores"[All Fields] AND "bed"[All Fields]) OR "sores bed"[All Fields]) OR ("pressure ulcer"[MeSH Terms] OR ("pressure"[All Fields] AND "ulcer"[All Fields]) OR "pressure ulcer"[All Fields] OR ("pressure"[All Fields] AND "sore"[All Fields]) OR "pressure sore"[All Fields]) OR ("pressure ulcer"[MeSH Terms] OR ("pressure"[All Fields] AND "ulcer"[All Fields]) OR "pressure ulcer"[All Fields] OR ("pressure"[All Fields] AND "sores"[All Fields]) OR "pressure sores"[All Fields]) OR ("pressure ulcer"[MeSH Terms] OR ("pressure"[All Fields] AND "ulcer"[All Fields]) OR "pressure ulcer"[All Fields] OR ("ulcer"[All Fields]

AND "pressure"[All Fields]) OR "ulcer pressure"[All Fields]) OR ("pressure ulcer"[MeSH Terms] OR ("pressure"[All Fields] AND "ulcer"[All Fields]) OR "pressure ulcer"[All Fields] OR ("ulcer"[All Fields] AND "decubitus"[All Fields]) OR "ulcer decubitus"[All Fields]) OR ("pressure ulcer"[MeSH Terms] OR ("pressure"[All Fields] AND "ulcer"[All Fields]) OR "pressure ulcer"[All Fields] OR ("sore"[All Fields] AND "pressure"[All Fields]) OR "sore pressure"[All Fields]) OR ("pressure ulcer"[MeSH Terms] OR ("pressure"[All Fields] AND "ulcer"[All Fields]) OR "pressure ulcer"[All Fields] OR ("bed"[All Fields] AND "sore"[All Fields]) OR "bed sore"[All Fields]) OR ("pressure ulcer"[MeSH Terms] OR ("pressure"[All Fields] AND "ulcer"[All Fields]) OR "pressure ulcer"[All Fields] OR ("bed"[All Fields] AND "sores"[All Fields]) OR "bed sores"[All Fields]) OR ("pressure ulcer"[MeSH Terms] OR ("pressure"[All Fields] AND "ulcer"[All Fields]) OR "pressure ulcer"[All Fields] OR ("decubitus"[All Fields] AND "ulcers"[All Fields]) OR "decubitus ulcers"[All Fields])) AND ("epidemiology"[MeSH Subheading] OR "epidemiology"[All Fields] OR "prevalence"[All Fields] OR "prevalence"[MeSH Terms] OR "prevalance"[All Fields] OR "prevalences"[All Fields] OR "prevalence s"[All Fields] OR "prevalent"[All Fields] OR "prevalently"[All Fields] OR "prevalents"[All Fields] OR ("prevalence"[MeSH Terms] OR "prevalence"[All Fields] OR ("period"[All Fields] AND "prevalence"[All Fields]) OR "period prevalence"[All Fields]) OR ("prevalence"[MeSH Terms] OR "prevalence"[All Fields] OR ("period"[All Fields] AND "prevalences"[All Fields]) OR "period prevalences"[All Fields]) OR ("prevalence"[MeSH Terms] OR "prevalence"[All Fields] OR ("prevalence"[All Fields] AND "period"[All Fields]) OR "prevalence period"[All Fields]) OR ("prevalence"[MeSH Terms] OR "prevalence"[All Fields] OR ("point"[All Fields] AND "prevalence"[All Fields]) OR "point prevalence"[All Fields]) OR ("prevalence"[MeSH Terms] OR "prevalence"[All Fields] OR ("point"[All Fields] AND "prevalences"[All Fields]) OR "point prevalences"[All Fields]) OR ("prevalence"[MeSH Terms] OR "prevalence"[All Fields] OR ("prevalence"[All Fields] AND "point"[All Fields]) OR "prevalence point"[All Fields])) AND (("risk factors"[MeSH Terms]

OR ("risk"[All Fields] AND "factors"[All Fields]) OR "risk factors"[All Fields] OR ("factor"[All Fields] AND "risk"[All Fields]) OR "factor risk"[All Fields] OR ("risk factors"[MeSH Terms] OR ("risk"[All Fields] AND "factors"[All Fields]) OR "risk factors"[All Fields] OR ("risk"[All Fields] AND "factor"[All Fields]) OR "risk factor"[All Fields]) OR ("risk factors"[MeSH Terms] OR ("risk"[All Fields] AND "factors"[All Fields]) OR "risk factors"[All Fields] OR ("social"[All Fields] AND "risk"[All Fields] AND "factors"[All Fields]) OR "social risk factors"[All Fields]) OR ("risk factors"[MeSH Terms] OR ("risk"[All Fields] AND "factors"[All Fields]) OR "risk factors"[All Fields] OR ("factor"[All Fields] AND "social"[All Fields] AND "risk"[All Fields])) OR ("risk factors"[MeSH Terms] OR ("risk"[All Fields] AND "factors"[All Fields]) OR "risk factors"[All Fields] OR ("factors"[All Fields] AND "social"[All Fields] AND "risk"[All Fields]) OR "factors social risk"[All Fields]) OR ("risk factors"[MeSH Terms] OR ("risk"[All Fields] AND "factors"[All Fields]) OR "risk factors"[All Fields] OR ("risk"[All Fields] AND "factor"[All Fields] AND "social"[All Fields]) OR "risk factor social"[All Fields]) OR ("risk factors"[MeSH Terms] OR ("risk"[All Fields] AND "factors"[All Fields]) OR "risk factors"[All Fields] OR ("risk"[All Fields] AND "factors"[All Fields] AND "social"[All Fields]) OR "risk factors social"[All Fields]) OR ("risk factors"[MeSH Terms] OR ("risk"[All Fields] AND "factors"[All Fields]) OR "risk factors"[All Fields] OR ("social"[All Fields] AND "risk"[All Fields] AND "factor"[All Fields]) OR "social risk factor"[All Fields]) OR ("risk factors"[MeSH Terms] OR ("risk"[All Fields] AND "factors"[All Fields]) OR "risk factors"[All Fields] OR ("health"[All Fields] AND "correlates"[All Fields]) OR "health correlates"[All Fields]) OR ("risk factors"[MeSH Terms] OR ("risk"[All Fields] AND "factors"[All Fields]) OR "risk factors"[All Fields] OR ("correlates"[All Fields] AND "health"[All Fields]) OR "correlates health"[All Fields]) OR ("risk factors"[MeSH Terms] OR ("risk"[All Fields] AND "factors"[All Fields]) OR "risk factors"[All Fields] OR ("population"[All Fields] AND "risk"[All Fields]) OR "population at risk"[All Fields]) OR

("risk factors"[MeSH Terms] OR ("risk"[All Fields] AND "factors"[All Fields]) OR "risk factors"[All Fields] OR ("populations"[All Fields] AND "risk"[All Fields]) OR "populations at risk"[All Fields]) OR ("risk factors"[MeSH Terms] OR ("risk"[All Fields] AND "factors"[All Fields]) OR "risk factors"[All Fields] OR ("risk"[All Fields] AND "scores"[All Fields]) OR "risk scores"[All Fields]) OR ("risk factors"[MeSH Terms] OR ("risk"[All Fields] AND "factors"[All Fields]) OR "risk factors"[All Fields] OR ("risk"[All Fields] AND "score"[All Fields]) OR "risk score"[All Fields]) OR ("risk factors"[MeSH Terms] OR ("risk"[All Fields] AND "factors"[All Fields]) OR "risk factors"[All Fields] OR ("score"[All Fields] AND "risk"[All Fields]) OR "score risk"[All Fields]) OR ("risk factors"[MeSH Terms] OR ("risk"[All Fields] AND "factors"[All Fields]) OR "risk factors"[All Fields] OR ("risk"[All Fields] AND "factor"[All Fields] AND "scores"[All Fields]) OR "risk factor scores"[All Fields]) OR ("risk factors"[MeSH Terms] OR ("risk"[All Fields] AND "factors"[All Fields]) OR "risk factors"[All Fields] OR ("risk"[All Fields] AND "factor"[All Fields] AND "score"[All Fields]) OR "risk factor score"[All Fields]) OR ("risk factors"[MeSH Terms] OR ("risk"[All Fields] AND "factors"[All Fields]) OR "risk factors"[All Fields] OR ("score"[All Fields] AND "risk"[All Fields] AND "factor"[All Fields]) OR "score risk factor"[All Fields])) AND 1990/01/01:2022/08/01[Date - Publication]) AND ("africa south of the sahara"[MeSH Terms] OR ("africa"[All Fields] AND "south"[All Fields] AND "sahara"[All Fields]) OR "africa south of the sahara"[All Fields] OR ("sub"[All Fields] AND "saharan"[All Fields] AND "africa"[All Fields]) OR "sub saharanafrica"[All Fields] OR ("africa south of the sahara"[MeSH Terms] OR ("africa"[All Fields] AND "south"[All Fields] AND "sahara"[All Fields]) OR "africa south of the sahara"[All Fields] OR ("africa"[All Fields] AND "sub"[All Fields] AND "saharan"[All Fields]) OR "africasub saharan"[All Fields]))) AND (1990/1/1:2022/8/1[pdat])

The full-text search results were entered into an EndNote database for screening.

### 3.5 Study Selection Process

All search records in EndNote were checked for duplication. After removing duplicates, the remaining records were screened for abstract and title using a set of inclusion criteria developed using the PICOTS framework (Population, Intervention, Comparisons, Outcomes, Time, Studies). Firstly, abstract and title screening using the inclusion criteria was used, followed by a review of the remaining full-text publications using both the inclusion and exclusion criteria. Two review authors (KT and OA)) independently assessed the eligibility and methodology quality of all the potential studies we identified as a result of the search strategy. We resolved the disagreement through discussion.

Inclusion criteria included: All original research articles conducted only in Sub-Saharan Africa settings that fulfil the following criteria were included in this systematic review. Those articles that were published only in English, conducted with cross-sectional studies, and had a quantitative research design were selected.

**Exclusion Criteria**

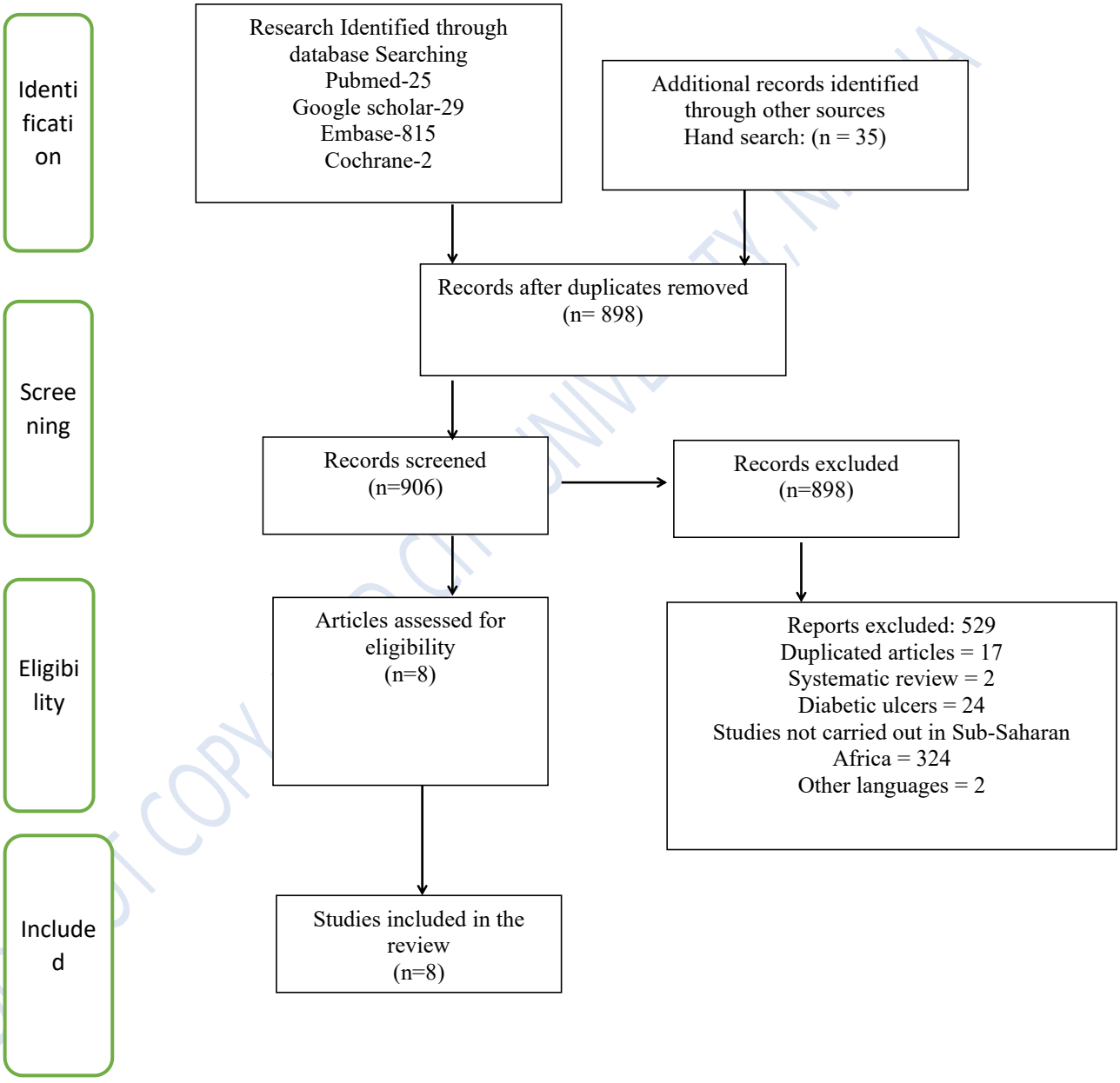
Studies that do not have prevalence data, risk factors or where the sample size was not given and qualitative studies will be excluded.

All of the full-text articles were filtered and sorted in ENDNOTE. Excluded articles were labelled with the reason for exclusion, while included articles were labelled by relevant categories according to the purpose and domain of the study. The relevant categories were the prevalence data, and risk factors of pressure ulcers in Sub-Saharan Africa. Following the completion of the full-text screening, articles with identical authors were reviewed to ensure that all studies presented unique data without duplication.

The final included articles and their category labels were exported to spreadsheets. All included articles had basic information extracted: First Author name, Publication Year, Title of Journal, Period of participants' recruitment, Region of recruitment, Site of study, City/Area,

Sample Size, Study Design, Population type, Inclusion criteria, Exclusion criteria, Type of Analysis, Number of participants with pressure ulcers, the risk factors of pressure ulcers, the anatomical location of pressure ulcers, number of studies that reported strategic prevention measure for pressure ulcers and the grading scores of pressure ulcers. For each relevant category, relevant articles were reviewed with key findings documented.

**PRISMA flow diagram for new systematic reviews which included searches of databases, registers and other sources**



# Chapter Four

## Results and Discussions of Findings

### 4.0 Introduction

The full search summary is presented in the Transparent Reporting of Systematic Reviews and Meta-analyses (PRISMA) flow diagram shown in Figure 4.0. A total of 906 records were identified through the electronic searches. After removal of duplicates, 889 records remained for eligibility screening.

Following the abstract review, 889 full-text articles were screened in using the inclusion criteria.

Subsequently, in full-text review, 881 of the 889 articles were screened out by applying the exclusion criteria. A summary of the number of full-text articles and their reasons for exclusion are presented in Figure 1. 99.1% of the records were excluded because the purpose of the study was not captured.

### 4.1 Description of Included Studies

After applying the screening criteria, 8 studies were eligible for inclusion in this review (Table 1.1) and were analysed. Study designs were predominantly cross-sectional design (75%), but there is one cohort study (15%) and one Prospective study (15%). Four (50%) of the studies were conducted in Ethiopia, two (25%) were conducted in Nigeria, one (12.5%) in South Africa and one (12.5%) from Kenya. All studies consented participants and had ethics approval from governing bodies.

**Included studies (Table 1)**

| S/N | FIRST AUTHOR'S NAME | YEAR | TITLE | COUNTRY | STUDY DESIGN |
|---|---|---|---|---|---|
| 1 | MeleseMelakuKuruche | 2016 | Prevalence and Associated Factors of Pressure Ulcer among Adult Inpatients in Wolaita Sodo University Teaching Hospital, Southern Ethiopia | Ethiopia | Cross Sectional Studies |
| 2 | DinkieTadele Bereded | 2018 | Prevalence and risk factors of pressure ulcer in hospitalized adult patients; a single center study from Ethiopia | Ethiopia | Cross Sectional Studies |
| 3 | OK Idowu | 2011 | Risk factors for pressure ulceration in a resource constrained spinal injury service | Nigeria | Prospective Study |
| 4 | C Joseph | 2015 | Prevalence of | South Africa | Observational |

| | | | | | |
|---|---|---|---|---|---|
| | | | secondary medical complications and risk factors for pressure ulcers after traumatic spinal cord injury during acute care in South Africa | | Studies |
| 5 | Peter KiteywoSisimwo | 2015 | Prevalence and risk factors for Pressure ulcers among adult inpatients at a tertiary referral hospital Kenya, 2021 | Kenya | Cross Sectional |
| 6 | Adegoke BOA | 2013 | Pressure ulcer prevalence among hospitalised adults in university hospitals in South-west Nigeria | Nigeria | Cross Sectional |
| 7 | Tihitina Assefa | 2017 | Prevalence of Bed Sore and its associated Factors among Patients admitted at Jimma | Ethiopia | Cross-Sectional Study |

| | | | | | |
|---|---|---|---|---|---|
| | | | University Medical Center, Jimma Zone, Southwestern Ethiopia, 2017 Cross-sectional study | | |
| 8 | HaileyesusGedamu, | 2014 | Prevalence and Associated Factors of Pressure Ulcer among Hospitalized Patients at Felegehiwot Referral Hospital, Bahir Dar, Ethiopia | Ethiopia | Cross Sectional Study |



### 4.2 Eligibility Criteria

Following the PICOS model:

**Patient, Population, or Problem:** Any age, including children, in any setting, specifically including hospital patients from any clinical area but not restricted to hospital settings

**Intervention, Prognostic Factor, or Exposure**: Pressure ulcers prevalence and risk factors where available by grade.

**Comparison:** Differences between various types of study.

**Outcome**: Pooled estimate of pressure ulcer prevalence and risk factors in Sub-Saharan Africa.

Papers considered to those based in Sub-Saharan Africa, published after 1990, and for which a full paper was available. Other language papers were not accepted. Additionally, if a publication had a title that seemed relevant but no abstract, the complete manuscript of that paper was identified and included if it was determined to fulfil the review's inclusion criteria. Studies that involved physical examinations of the subjects or data extraction from health records, databases, interviews, or surveys were taken into consideration. Specialty units, such those for spinal cord injuries, were included in addition to the normal hospital's patient specialist. Studies on riskfactors, point prevalence, and period prevalence were also considered.

**PICOTS (Population, Intervention, Comparisons, Outcomes, Time, Studies) (Table 2)**

| | |
|---|---|
| Population | Any age, including children, in any setting, specifically including hospital patients from any clinical area but not restricted to hospital settings in Sub-Saharan Africa |
| Intervention | Pressure ulcers prevalence and risk factors where available by grade. |
| Comparisons | Differences between various types of study. |
| Outcomes | Pooled estimate of pressure ulcer prevalence and risk factors in Sub-Saharan Africa. |
| Time | 1/1 /1990 to 1/8/2022 |
| Studies | Observational studies, Cross-Sectional and Prospective study |

## 4.3 Participation Studies

The majority of the studies reviewed were of general hospital patients but few were of spinal cord injury patients and some employed skin assessment. Six studies were of medical/surgical/ICU hospital populations and the rest of the studies were of spinal injury patients. Eight studies were considered in total with 2855 participants. Of the studies with physical examination there were six studies based on medical/surgical/ICU and/or general hospital patients which had a total of 2609 participants. The other two studies had no physical examination with 246 participants. In the studies, four of the data were collected by nurses in which some of the nurses were trained by the researcher while two were collected by the researcher themselves.

**Participation Studies (Table 3)**

| SN | Author | Year | Clinical area | Sample size | Data Collection | Skin assessment |
|---|---|---|---|---|---|---|
| 1 | MeleseMelakuKuruche | 2016 | Medical/surgical/ICU | 239 | Nurses | Yes |
| 2 | DinkieTadele Bereded | 2018 | Medical/surgical/gynecological and orthopedic wards | 355 | Nurses | Yes |
| 3 | OK Idowu | 2011 | Spinal cord injury | 105 | Researcher | No |
| 4 | C Joseph | 2015 | Spinal cord injury | 141 | Weekly Audit | No |
| 5 | Peter KiteywoSisimwo | 2015 | Medical/surgical | 216 | Qualified Research Assistant | Yes |
| 6 | Adegoke BOA | 2013 | Medical/surgical | 1211 | Researcher | Yes |
| 7 | Tihitina Assefa | 2017 | Medical ward, Surgical ward, Maternity ward and Intensive care units | 166 | Nurses Trained by Clinician | Yes |
| 8 | HaileyesusGedamu, | 2014 | medical ward, surgical ward, and gynecological wards | 422 | Nurses Trained by Researcher | Yes |



## 4.4 The Prevalence of Pressure Ulcers in Sub-Saharan Africa

All the studies reported prevalence on pressure ulcers. The total prevalence in the eight studies was 328. From the studies, Melese K et al., (2016), Dinkie T et al., (2018), Tihitina A et al., (2017) and Gedamu H et al., (2014) reported that the prevalence of pressure ulcer in Ethiopia was high among the hospitalized patients their report respectively were 32(13.4%), 53(14.9%) 16(9.6) and 71(16.8%). Furthermore, the studies from Nigeria also reported high prevalence among their participants; Idowu K et al., (2011) and Adegoke B et al., (2013), 39(3.22). Adegoke B et al., (2013) further concluded that although the mean prevalence of pressure ulcers among hospitalized adults in University Hospitals in South-west Nigeria was high the males have higher prevalence compared to the females 3.59%, 2.83% respectively. This was similar to the result from Gedamu H et al., (2014) who also reported high prevalence of pressure ulcers in hospitalized patients in Felegehiwot with higher prevalence seen in males 42 participants and 29 among females.

Other studies did not differentiate the prevalence between the gender therefore, conclusion on the gender with higher prevalence could not be reported in this study.

In addition, the study conducted in South Africa by Joseph C et al., (2015) it was reported that the pressure ulcers was the most common secondary medical complications following acute tSCI as the study also reported the prevalence of 42(29.5%), among its participants. In Kenya as well, Peter Kiteywo et al., (2015) reported from his study on the prevalence and risk factors for pressure ulcers among adult inpatients at tertiary referral hospital that although pressure ulcer has been given substantial consideration within the hospital, the still have a high prevalence 36(16.66).



| SN | Author | Year | Country | Sample size | Mean age | Prevalence | Gender |
|---|---|---|---|---|---|---|---|

| | | | | | | | M | F |
|---|---|---|---|---|---|---|---|---|
| 1 | MeleseMelakuKuruche | 2016 | Ethiopia | 239 | 34 | 32(13.4) | | |
| 2 | DinkieTadele Bereded | 2018 | Ethiopia | 355 | | 53(14.9) | | |
| 3 | OK Idowu | 2011 | Nigeria | 105 | 35.5 | - | | |
| 4 | C Joseph | 2015 | South Africa | 141 | >18 | 42(29.8) | | |
| 5 | Peter KiteywoSisimwo | 2015 | Kenya | 216 | 59.3 | 36(16.66) | | |
| 6 | Adegoke BOA | 2013 | Nigeria | 1211 | | 39(3.22) | 3.59% | 2.83% |
| 7 | Tihitina Assefa | 2017 | Ethiopia | 166 | >18 | 16(9.6) | | |
| 8 | HaileyesusGedamu, | 2014 | Ethiopia | 422 | 32 | 71(16.8) | 42 | 29 |

**Pressure Ulcer prevalence (Table 4)**

### 4.5 Risk factors for Pressure Ulcers in Sub-Saharan Africa

Generally the risk factorsassociated with the presence of pressure ulcer that was reported in the studies are absence of change of position by nurses, bedbound, friction, nutrition, delay in admission and shear problems. Only one out of the 8 studies did not report the risk factors of pressure ulcers. The first study by Melese K et al., (2016) was carried out from March 1 to April, 2015 in medical, surgical and in intensive care units. They assessed the prevalence and associated factors of pressure ulcer among adult in patients in Wolaita Sodo University Teaching Hospital, Southern Ethiopia, they identified the risk factors related to pressure ulcer. Out of the 239 participants 32 inpatients were found to develop pressure ulcers. It was reported that absence of change of position by nurses, bedbound, friction and shear problems are associated with the presence of pressure ulcer. Melese K et al., (2016) also stated that prolonged length of stay in

hospital above 21 days without changing position result in pressure ulcer. Only 7 participants changed their position and 25 were not change throughout their stay. It was also reported that most of the individual that developed pressure ulcer did not use pressure ulcer relieving device and only few used airings pressure relieving device.

The second study by Dinkie T et al., 2018) with total sample size of 355 and with the aim to assess the prevalence of pressure ulcer and its risk factors among adult hospitalised patients at Dessie Referral hospital in Ethiopia, has the prevalence of 53 among all the participants. From the participants with pressure ulcers it was reported that 6 have their position changed while others were not changed. This is an indicator to the high prevalence of the pressure ulcers in the hospital. Furthermore, Dinkie T et al., (2018) stated that lack of regular positioning and activity, friction and prolonged hospitalization are risk factors for pressure ulcers.

The third study by Idowu K et al., (2011) carried out in Lagos Nigeria in patients with traumatic spinal cord injury revealed that the poor nutritional status and delay in admission are significant factors in the prevalence of pressure ulceration in spinal cord injury.

Tihitina A et al., (2017) and Gedamu H et al., (2014) also reported that prolonged length of stay in hospital, poor nutrition and poor mobility with slight limit of sensory perception, and friction and shearing forces were significantly associated with the presence of pressure ulcer.



| SN | Author | Year | Sample | Clinical | Mean | Change | Risk Factors |
|---|---|---|---|---|---|---|---|

| | | | **size** | **area** | **age** | **position** | | |
|---|---|---|---|---|---|---|---|---|
| | | | | | | Yes | No | |
| 1 | MeleseMelakuKuruche | 2016 | 239 | Medical/surgical/ICU | 34 | 7 | 25 | • Long stayed in the hospital<br>• Limited sensory perception<br>• Diabetes mellitus<br>• Bed ridden<br>• Friction and shearing forces<br>• Nurses not changing position<br>• Antidepressant medications |
| 2 | DinkieTadele Bereded | 2018 | 355 | Medical/surgical/gynecological and orthopedic | | 6(2.9) | 47 (32.2) | • Long stayed in the hospital |

| | | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| | | | | wards | | | | • Limited sensory perception<br>• Diabetes mellitus<br>• Friction and shearing forces<br>• Nurses not changing position<br>• Antidepressant medications |
| 3 | OK Idowu | 2011 | | Spinal cord injury | 35.5 | | | • Poor nutrition |
| 4 | C Joseph | 2015 | 141 | Spinal cord injury | >18 | | | • Vertebral injury |
| 5 | Peter KiteywoSisimwo | 2015 | 216 | Medical/surgical | 59.3 | | | • Poor nutrition, poor support surfaces, |

| | | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| | | | | | | | | poor mobility |
| 6 | Adegoke BOA | 2013 | 1211 | Medical/surgical | | (92.3%) BY RELATIVE | | --- |
| 7 | Tihitina Assefa | 2017 | 166 | Medical ward, Surgical ward, Maternity ward and Intensive care units | >18 | 150 (90.4%) | 16(9.6) | • Poor nutrition<br>• poor support surfaces<br>• poor mobility |
| 8 | Haileyesus Gedamu, | 2014 | 422 | medical ward, surgical ward, and gynecological wards | 32 | 5(7%) | 93% (66) | • Prolonged length of stay in hospital>21, slight limit of<br>• sensory perception, and friction and shearing forces |

**Risk factors of pressure ulcers (Table 5)**

## 4.6 Best-Adopted Strategy to Prevent Pressure Ulcers in Sub-Saharan Africa

No studies reported strategy to prevent pressure ulcers in sub-Saharan Africa.

## 4.7 Risk Assessment and Grading Scores for Pressure Ulcer

Two studies reported pressure ulcers without stating a grading System (Joseph C et al., 2015 and Kiteywo et al., (2015), others used NPUAP or EPUAP grading systems, one used the grading system employed in PUPPS (Adegoke B et al., 2013 ) which is consistent with NPUAP.

One study reported unstageable ulcers or deep tissue injuries (Dinkie T et al., 2018)

Whereas three studies did not report the stages of their participants pressure ulcers (Idowu K et al., (2011), Adegoke B et al., (2013) and Joseph C et al., 2015). Among the participants the staging reported for I, II, III, IV are 94, 66, 28 and 11 respectively. Three studies used Braden risk assessment tools while two used ASIA risk assessment too though three studies did not mention the assessment tools used in their study (table 6)

| SN | Author/ year | Year | PU classification | Risk assessment tool | Stage | | | |
|---|---|---|---|---|---|---|---|---|
| | | | | | I | II | III | IV |
| 1 | MeleseMelakuKuruche | 2016 | EUPAP | Braden | | 14(43.75) | 9(28.12) | 3(9.375) |
| 2 | DinkieTadele Bereded | 2018 | EUPAP | Braden | 34(64) | 16(30) | 3(6) | |
| 3 | OK Idowu | 2011 | NPUAS | ASIA | | | | |
| 4 | C Joseph | 2015 | | ASIA | | | | |
| 5 | Peter KiteywoSisimwo | 2015 | | | 13(36.1) | 10(27.8) | 9(25) | 4(11.1) |
| 6 | Adegoke BOA | 2013 | pressure ulcer point Prevalence Survey (PUPPS) | | | | | |

| | | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| 7 | Tihitina Assefa | 2017 | EUPAP | BRADEN | 3(18.5%) | 7(44%) | 4(25%) | 2(12.5%) |
| 8 | Haileyesus Gedamu, | 2014 | EUPAP | | 44( 62%) | 19(26.8%) | 3(4.3%) | 2(2.8%) |

**Risk Assessment and Grading Scores for Pressure Ulcer (Table 6)**

### 4.8 Anatomical Location of the Pressure Ulcer

The sacral region was the most common pressure ulcer site in the studies and this occurred in 171 cases in all the studies. The other pressure ulcer sites include buttocks 66, heel 46, occipital 4 and Trochanter 56 patients were recorded in all the studies.

| SN | Author/ year | Year | Site |
|---|---|---|---|
| 1 | MeleseMelakuKuruche | 2016 | 18(56.25) Buttock<br>12(37.5) Sacral Area |
| 2 | DinkieTadele Bereded | 2018 | Sacral 26(49.1)<br>Heel 12(22.6)<br>Elbow 4(7.5)<br>Occiput 2 (3.8) |

| | | | |
|---|---|---|---|
| | | | Shoulder 2(3.8)<br>Sacral And Shoulder 3(5.7)<br>More Than Two Sites 4(7.5) |
| 3 | OK Idowu | 2011 | Sacral 56(45.9)<br>Trochanteric 44(36.1)<br>Heel 18(14.8)<br>Elbow 2 (1.6)<br>Shoulder Blade And Occipital Region 1(0.8) |
| 4 | C Joseph | 2015 | Cervical 23(31)<br>Thoracic 18(33)<br>Lumbar 1(8) |
| 5 | Peter KiteywoSisimwo | 2015 | Buttock 14(38.9)<br>7(19.4) Heel |
| 6 | Adegoke BOA | 2013 | Buttocks 34(43.6%)<br>Sacrum14(18.0%)<br>And Heel 6(7.7%)<br>Trochanter 8(10.3%) |

| | | | |
|---|---|---|---|
| 7 | Tihitina Assefa | 2017 | 7(44 %) Sacral Area,<br>4(25) % Trochanter,<br>3(12.5 %) Heel And<br>2(18.5 %) Occipital Region |
| 8 | HaileyesusGedamu, | 2014 | 70.4% (50)<br>Sacral Area<br>10% (7)<br>Both Sacral And Shoulder |

**Anatomical location of the pressure ulcer (Table 7)**

# Chapter Five

## Discussion, Conclusions and Recommendations

### 5.1 Discussion

We carried out this research because Sub-Saharan Africa had few information accessible on pressure ulcer's prevalence and risk factor. The review only found a small number of studies which may possibly be because the majority of the countries did not research on the prevalence and risk factors of pressure ulcers, more so, the studies it may be that the journals indexed were not published by databases, or that the review's exclusion criteria prevented them from being taken into account. Studies on the prevalence of ulcers are lacking in the following countries: Zambia, Zimbabwe, Ghana, Botswana, Tanzania, Mali Sudan, and Congo. In this study, for the prevalence of the pressure ulcer the only countries with data were Ethiopia (n =4), Nigeria (n =2), South Africa (n =1) and Kenya (n=). The spinal cord injury studies were from Nigeria (n =1) and South Africa (n=1). Thus out of 46 countries in Sub-Saharan Africa only eight countries had studies on pressure ulcer prevalence and risk factors.

In our review it is difficult to directly compare the age mean as many studies gave age ranges rather than means, but for general hospital participants the age ranged from the lowest, a median range of 18–32 [105] and a mean of 34 [102] to the highest, a mean of 5 9.3[106] so it was revealed that the participants are younger.

Of the eight included studies, they were found from Pubmed, Google scholar and internet searches. In studies where physical examination was employed it was never stated whether this was a head to toe examination of the patient and thus there remains some doubt as to whether some ulcers may have been missed. In some studies where the point prevalence was mentioned it was not category to in according to their gender so as to know in the gender with highest prevalence. Where grades of pressure ulcers were reported there were few prevalence recorded for grade IV ulcers seen in some studies, only one study (102) including observational studies did report grade I stage and this may be due to difficulty in assessing dark

toned skin for grade I ulcers. The overall prevalence in the 4 countries was high and the studies revealed that generally when patient remain in a position for more than 21 days with prolonged hospitalization give strong indication to presence of pressure ulcer. Among other factors gathered in the studies, poor nutrition and problem of sensory perception can subject patient to pressure ulcer. Therefore, nurses and patient's relative are encouraged to change the patient's position often.

## 5.2 Conclusion

In Sub-Saharan Africa, hospitalized patients had a high prevalence of pressure ulcers, according to this systematic review. Long-term hospital stays, sensory perception issues, the patient's primary diagnoses, poor nourishment, inadequate support surfaces, restricted mobility, the nurses' change of the patient's posture, and friction and shearing forces were all strongly linked to the prevalence of pressure ulcers. Additionally, those who had friction and shear as well as stayed more than twenty-one days in the hospital were more likely to acquire pressure ulcers.

## 5.3 Recommendation

More prospective studies are required to determine the prevalence and risk factors for pressure ulcers in Sub-Saharan Africa. Additionally, given the few studies that were examined and identified the pressure ulcer risk factors, it is critical to prioritize and put preventive measures in place for patients who are expected to stay in the hospital for an extended period of time. Nurses should be informed of the risk factors for pressure ulcers and urged to practice changing the patient's posture every two hours. The patient's relative should also acquire knowledge about the pressure ulcer risk factors and strategies to prevent it.

## 5.4 Limitations

The subject of the study has only been published in four Sub-Saharan Africa countries. The comparison of data was impeded by the paucity of literature on the prevalence and risk factors of pressure ulcers in various Sub-Saharan African countries. The review studies did not provide specific indices for the distribution of risk factors of pressure ulcer.

## Endnotes


1. Melese Meleku,Arba, Aseb, Atinafu Nega, and Esayas Aydiko. 2020. "Bed-Sore and Associated Factors among Patients Admitted at Surgical Wards of Wolaita Sodo University Teaching and Referral Hospital, Southern Ethiopia." American Journal of Clinical and Experimental Medicine 8, no. 4: 62. https://doi.org/10.11648/j.ajcem.20200804.11.

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## APPENDIX I

**Search Terms**

1. **Pressure ulcer (MeSH)**

   Ulcers, Pressure

   Sore, Bed

   Bedsores

   Decubitus Ulcer

   Pressure Ulcers

   Sores, Pressure

   Bedsore

   Ulcers, Decubitus

   Sores, Bed

   Pressure Sore

   Pressure Sores

   Ulcer, Pressure

   Ulcer, Decubitus

   Sore, Pressure

   Bed Sore

   Bed Sores

   Decubitus Ulcers

2. **Prevalence (MeSH)**

Prevalences

Period Prevalence

Period Prevalences

Prevalence, Period

Point Prevalence

Point Prevalences

Prevalence, Point

**3. Risk Factors (MeSH)**

Factor, Risk

Risk Factor

Social Risk Factors

Factor, Social Risk

Factors, Social Risk

Risk Factor, Social

Risk Factors, Social

Social Risk Factor

Health Correlates

Correlates, Health

Population at Risk

Populations at Risk

Risk Scores

Risk Score

Score, Risk

Risk Factor Scores

Risk Factor Score

Score, Risk Factor

4. **Children (MeSH)**

   Child

5. **Adolescent (MeSH)**

Adolescents

Adolescence

Teens

Teen

Teenagers

Teenager

Youth

Youths

Adolescents, Female

Adolescent, Female

Female Adolescent

Female Adolescents

Adolescents, Male

Adolescent, Male

Male Adolescent

Male Adolescents

**6. Adult (MeSH)**

Adult

**7. Sub-Saharan Africa (MeSH)**

Sub-Saharan Africa

Africa, Sub-Saharan

**University Compliance Certificate**

This is to certify that this thesis by Kafila Abimbola OYENUGA with Matric Number LCU/PG/001931in the Department of Public Health, Faculty of Basic Medical and Applied Sciences, Lead City University, Ibadan is in full compliance with the approved university format.

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