# Factors influencing Wound healing and Using Subcutaneous Drains in Gynecological Surgery

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**Abstract**

**Background:** Multiple factors can lead to impaired wound healing. In general terms, the factors that influence repair can be categorized into local and systemic. Local factors are those that directly influence the characteristics of the wound itself, while systemic factors are the overall health or disease state of the individual that affect his or her ability to heal. Many of these factors are related, and the systemic factors act through the local effects affecting wound healing. Placing pelvic drains following surgeries routinely for early-stage gynecologic malignancies needs to be revisited as it confers no advantage, and by avoiding drains, patients can be discharged to go home earlier.

**Keywords:** Wound healing, Gynecological Surgery, Subcutaneous Drains

## Introduction

Multiple factors can lead to impaired wound healing. In general terms, the factors that influence repair can be categorized into local and systemic. Local factors are those that directly influence the characteristics of the wound itself, while systemic factors are the overall health or disease state of the individual that affect his or her ability to heal. Many of these factors are related, and the systemic factors act through the local effects affecting wound healing **(1).**

**Local Factors That Influence Healing.**

***Oxygenation***

Oxygen is important for cell metabolism, especially energy production by means of ATP, and is critical for nearly all wound healing processes. It prevents wounds from infection, induces angiogenesis, increases keratinocyte differentiation, migration, and re-epithelialization, enhances fibroblast proliferation and collagen synthesis, and promotes wound contraction **(2).**

In addition, the level of superoxide production (a key factor for oxidative killing pathogens) by polymorphonuclear leukocytes is critically dependent on oxygen levels. Due to vascular disruption and high oxygen consumption by metabolically active cells, the microenvironment of the early wound is depleted of oxygen and is quite hypoxic. Several systemic conditions, including advancing age and diabetes, can create impaired vascular flow, thus setting the stage for poor tissue oxygenation. In the context of healing, this overlay of poor perfusion creates a hypoxic wound. Chronic wounds are notably hypoxic; tissue oxygen tensions have been measured transcutaneously in chronic wounds from 5 to 20 mm Hg, in contrast to control tissue values of 30 to 50 mm Hg. n wounds where oxygenation is not restored, healing is impaired.

Temporary hypoxia after injury triggers wound healing, but prolonged or chronic hypoxia delays wound healing (3).

In acute wounds, hypoxia serves as a signal that stimulates many aspects of the wound-healing process. Hypoxia can induce cytokine and growth factor production from macrophages, keratinocytes, and fibroblasts. Cytokines that are produced in response to hypoxia include PDGF, TGF-β, VEGF, tumor necrosis factor-α (TNF-α), and endothelin-1, and are crucial promoters of cell proliferation, migration and chemotaxis, and angiogenesis in wound healing. In normally healing wounds, ROS such as hydrogen peroxide($H_2O_2$) and superoxide ($O_2$ ) are thought to act as cellular messengers to stimulate key processes associated with wound healing, including cell motility, cytokine action (including PDGF signal transduction), and angiogenesis. Both hypoxia and hyperpoxia increase ROS production, but an increased level of ROS transcends the beneficial effect and causes additional tissue damage **(4).**

In summary, the proper oxygen level is crucial for optimum wound healing. Hypoxia stimulates wound healing such as the release of growth factors and angiogenesis, while oxygen is needed to sustain the healing process (Bishop, 2008). One therapeutic option that can sometimes overcome the influence of tissue hypoxia is hyperbaric oxygen therapy. While HBOT can be an effective treatment for hypoxic wounds, its availability is limited **(5).**

***Infections***

Once skin is injured, micro-organisms that are normally sequestered at the skin surface obtain access to the underlying tissues. The state of infection and replication status of the microorganisms determine whether the wound is classified as having contamination, colonization, local infection/critical colonization, and/or spreading invasive infection. Contamination is the presence of non-replicating organisms on a wound, while colonization is defined as the presence of replicating microorganisms on the wound without tissue damage. Local infection/ critical colonization is an intermediate stage, with microorganism replication and the beginning of local tissue responses. Invasive infection is defined as the presence of replicating organisms within a wound with subsequent host injury **(6).**

Inflammation is a normal part of the wound-healing processand is important to the removal of contaminating micro-organisms. In the absence of effective decontamination, however, inflammation may be prolonged, since microbial clearance is incomplete. Both bacteria and endotoxins can lead to the prolonged elevation of pro-inflammatory cytokines such as interleukin-1 (IL-1) and TNF-α and elongate the inflammatory phase. If this continues, the wound may enter a chronic state and fail to heal. This prolonged inflammation also leads to an increased level of matrix metalloproteases (MMPs), a family of proteases that can degrade the ECM. In tandem with the increased protease content, a decreased level of the naturally occurring protease inhibitors occurs. This shift in protease balance can cause growth factors that appears in chronic wounds to be rapidly degraded **(7).**

Similar to other infective processes, the bacteria in infected wounds occur in the form of biofilms, which are complex communities of aggregated bacteria embedded in a selfsecreted extracellular polysaccharide matrix **(8).**

Mature biofilms develop protected microenvironments and are more resistant to conventional antibiotic treatment. Staphylococcus aureus (S. aureus), Pseudomonas aeruginosa (P. aeruginosa), and β-hemolytic streptococci are common bacteria in infected and clinically non-infected wounds **(8).**

P. aeruginosa and Staphylococcus appear to play an important role in bacterial infection in wounds. Many chronic ulcers probably do not heal because of the presence of biofilms containing P. aeruginosa, thus shielding the bacteria from the phagocytic activity of invading polymorphonuclear neutrophils (PMNs). This mechanism may explain the failure of antibiotics as a remedy for chronic wounds **(9).**

**Systemic Factors That Influence Healing**

***Age***

The elderly population (people over 60 years of age) is growing faster than any other age group (World Health Organization [WHO, www.who.int/topics/ageing]), and increased age is a major risk factor for

impaired wound healing. Many clinical and animal studies at the cellular and molecular level have examined age-related changes and delays in wound healing. It is commonly recognized that, in healthy older adults, the effect of aging causes a temporal delay in wound healing, but not an actual impairment in terms of the quality of healing. Delayed wound healing in the aged is associated with an altered inflammatory response, such as delayed T-cell infiltration into the wound area with alterations in chemokine production and reduced macrophage phagocytic capacity. Delayed re-epithelialization, collagen synthesis, and angiogenesis have also been observed in aged mice as compared with young mice. Overall, there are global differences in wound healing between young and aged individuals **(10).**

A review of the age-related changes in healing capacity demonstrates that every phase of healing undergoes characteristic age-related changes, including enhanced platelet aggregation, increased secretion of inflammatory mediators, delayed infiltration of macrophages and lymphocytes, impaired macrophage function, decreased secretion of growth factors, delayed re-epithelialization, delayed angiogenesis and collagen deposition, reduced collagen turnover and remodeling, and decreased wound strength **(11).**

***Sex Hormones in Aged Individuals***

Sex hormones play a role in age-related wound-healing deficits. Compared with aged females, aged males have been shown to have delayed healing of acute wounds. A partial explanation for this is that the female estrogens (estrone and 17β-estradiol), male androgens (testosterone and 5α-dihydrotestosterone, DHT), and their steroid precursor dehydroepiandrosterone (DHEA) appear to have significant effects on the wound-healing process. It was recently found that the differences in gene expression between elderly male and young human wounds are almost exclusively estrogen-regulated **(12).**

Estrogen affects wound healing by regulating a variety of genes associated with regeneration, matrix production, protease inhibition, epidermal function, and the genes primarily associated with inflammation. Studies indicate that estrogen can improve the age-related impairment in healing in both men and women, while androgens regulate cutaneous wound healing negatively **(12).**

***Stress***

Stress has a great impact on human health and social behavior. Many diseases such as cardiovascular disease, cancer, compromised wound healing, and diabetes are associated with stress. Numerous studies have confirmed that stress-induced disruption of neuroendocrine immune equilibrium is consequential to health. The pathophysiology of stress results in the deregulation of the immune system, mediated primarily through the hypothalamic-pituitaryadrenal (HPA) and sympathetic-adrenal medullary axes or sympathetic nervous system **(13).**

Studies in both humans and animals have demonstrated that psychological stress causes a substantial delay in wound healing. Caregivers of persons with Alzheimer's and students undergoing academic stress during examinations demonstrated delayed wound healing **(14).**

The hypothalamic-pituitary-adrenal and the sympathetic-adrenal medullary axes regulate the release of pituitary and adrenal hormones. These hormones include the adrenocorticotrophic hormones, cortisol and prolactin, and catecholamines (epinephrine and norepinephrine). Stress up-regulates glucocorticoids (GCs) and reduces the levels of the proinflammatory cytokines IL-1β, IL-6, and TNF-α at the wound site. Stress also reduces the expression of IL-1α and IL-8 at wound sites—both chemoattractants that are necessary for the initial inflammatory phase of wound healing **(13).**

Furthermore, GCs influence immune cells by suppressing differentiation and proliferation, regulating gene transcription, and reducing expression of cell adhesion molecules that are involved in immune cell trafficking. The GC cortisol functions as an anti-inflammatory agent and modulates the Th1-mediated immune responses that are essential for the initial phase of healing. Thus, psychological stress impairs normal cellmediated immunity at the wound site, causing a significant delay in the healing process.

Stressors can lead to negative emotional states, such as anxiety and depression, which may in turn have an impact on physiologic processes and/or behavioral patterns that influence health outcomes. In addition to the direct influences of anxiety and depression on endocrine and immune function, stressed individuals are more likely to have unhealthy habits, which include poor sleep patterns, inadequate nutrition, less exercise, and a greater propensity for abuse of alcohol, cigarettes, and other drugs. All of these factors may come into play in negatively modulating the healing response **(15).**

***Diabetes.***

Diabetes affects hundreds of millions of people worldwide. Diabetic individuals exhibit a documented impairment in the healing of acute wounds. Moreover, this population is prone to develop chronic non-healing diabetic foot ulcers (DFUs), which are estimated to occur in 15% of all persons with diabetes. DFUs are a serious complication of diabetes, and precede 84% of all diabetesrelated lower leg amputations**.** The impaired healing of both DFUs and acute cutaneous wounds in persons with diabetes involves multiple complex pathophysiological mechanisms. DFUs, like venous stasis disease and pressure-related chronic non-healing wounds, are always accompanied by hypoxia **(16)**

A situation of prolonged hypoxia, which may be derived from both insufficient perfusion and insufficient angiogenesis, is detrimental for wound healing. Hypoxia can amplify the early inflammatory response, thereby prolonging injury by increasing the levels of oxygen radicals. Hyperglycemia can also add to the oxidative stress when the production of ROS exceeds the anti-oxidant capacity. The formation of advanced glycation end-products (AGEs) under hyperglycemia and the interaction with their receptors (RAGE) are associated with impaired wound healing in diabetic mice as well. High levels of metalloproteases are a feature of diabetic foot ulcers, and the MMP levels in chronic wound fluid are almost 60 times higher than those in acute wounds. This increased protease activity supports tissue destruction and inhibits normal repair processes. Several dysregulated cellular functions are involved in diabetic wounds, such as defective T-cell immunity, defects in leukocyte chemotaxis, phagocytosis, and bactericidal capacity, and dysfunctions of fibroblasts and epidermal cells. These defects are responsible for inadequate bacterial clearance and delayed or impaired repair in individuals with diabetes **(17).**

As mentioned above, hypoxia contributes to the compromised healing of DFUs, and diabetic wounds exhibit inadequate angiogenesis. Several studies that have investigated the mechanisms behind the decreased restoration of vasculature in diabetic wounds have implied that EPC mobilization and homing are impaired, and that the level of VEGF, the primary proangiogenic factor in wounds, is decreased in the diabetic state. Stem-cell-based therapies aimed at inducing EPCs or BM-MSCs have shown a promising outcome in diabetic nonhealing wounds, both in animals and in clinical trials. In animal studies, therapeutic restoration of VEGF has been shown to improve repair outcomes significantly**.** The neuropathy that occurs in diabetic individuals probably also contributes to impaired wound healing. Neuropeptides such as nerve growth factor, substance P, and calcitonin gene-related peptide are relevant to wound healing, because they promote cell chemotaxis, induce growth factor production, and stimulate the proliferation of cells. A decrease in neuropeptides has been associated with DFU formation. In addition, sensory nerves play a role in modulating immune defense mechanisms, with denervated skin exhibiting reduced leukocyte infiltration **(18).**

In summary, the impaired healing that occurs in individuals with diabetes involves hypoxia, dysfunction in fibroblasts and epidermal cells, impaired angiogenesis and neovascularization, high levels of metalloproteases, damage from ROS and AGEs, decreased host immune resistance, and neuropathy **(17).**

***Medications***

Many medications, such as those which interfere with clot formation or platelet function, or inflammatory responses and cell proliferation have the capacity to affect wound healing. Here we review only the commonly used medications that have a significant impact on healing, including glucocorticoid steroids, non-steroidal antinflammatory drugs, and chemotherapeutic **(19).**

***Obesity***

Obesity is well-known to increase the risk of many diseases and health conditions, which include coronary heart disease, type 2 diabetes, cancer, hypertension, dyslipidemia, stroke, sleep apnea, respiratory problems, and impaired wound healing. Obese individuals frequently face wound complications, including skin wound infection, dehiscence, hematoma and seroma formation, pressure ulcers, and venous ulcers. An increased frequency of wound complications has been reported for obese individuals undergoing both bariatric and non-bariatric operations. In particular, a higher rate of surgical site infection occurs in obese patients. Many of these complications may be a result of a relative hypoperfusion and ischemia that occurs in subcutaneous adipose tissue. This situation may be caused by a decreased delivery of antibiotics as well. In surgical wounds, the increased tension on the wound edges that is frequently seen in obese patients also contributes to wound dehiscence. Wound tension increases tissue pressure, reducing microperfusion and the availability of oxygen to the wound. In addition to local conditions, systemic factors also play an important role in impaired wound healing and wound complications in obese patients. Obesity can be connected to stress, anxiety, and depression, all situations which can cause an impaired immune response **(20).**

***Alcohol Consumption***

Clinical evidence and animal experiments have shown that exposure to alcohol impairs wound healing and increases the incidence of infection**.** The effect of alcohol on repair is quite clinically relevant, since over half of all emergency room trauma cases involve either acute or chronic alcohol exposure. Alcohol exposure diminishes host resistance, and ethanol intoxication at the time of injury is a risk factor for increased susceptibility to infection in the wound. Studies have demonstrated profound effects of alcohol on host-defense mechanisms, Beyond the increased incidence of infection, exposure to ethanol also seems to influence the proliferative phase of healing **(21).**

***Smoking***

It is well-known that smoking increases the risk of heart and vascular disease, stroke, chronic lung disease, and many kinds of cancers. Similarly, the negative effects of smoking on wound-healing outcomes have been known for a long time. Post-operatively, patients who smoke show a delay in wound healing and an increase in a variety of complications such as infection, wound rupture, anastomotic leakage, wound and flap necrosis, epidermolysis, and a decrease in the tensile strength of wounds **(18).**

In the realm of oral surgery, impaired healing in smokers has been noticed both in routine oral surgery and in the placement of dental implants. Cosmetic outcomes also appear to be worse in smokers, and plastic and reconstructive surgeons are often reluctant to perform cosmetic surgeries on individuals who refuse to quit smoking **(18).**

***Nutrition***

For more than 100 years, nutrition has been recognized as a very important factor that affects wound healing. Most obvious is that malnutrition or specific nutrient deficiencies can have a profound impact on wound healing after trauma and surgery. Patients with chronic or non-healing wounds and experiencing nutrition deficiency often require special nutrients. Energy, carbohydrate, protein, fat, vitamin, and mineral metabolism all can affect the healing process. Together with fats, carbohydrates are the primary source of energy in the wound-healing process. Glucose is the major source of fuel used to create the cellular ATP that provides energy for angiogenesis and deposition of the new tissues. The use of glucose as a source for ATP synthesis is essential in preventing the depletion of other amino acid and protein substrates. Protein is one of the most important nutrient factors affecting wound healing. A deficiency of protein can impair capillary formation, fibroblast proliferation, proteoglycan synthesis, collagen synthesis, and wound remodeling. A deficiency of protein also affects the immune system, with resultant decreased leukocyte phagocytosis and increased susceptibility to infection**.** Collagen is the major protein component of connective tissue and is composed primarily of glycine, proline, and hydroxyproline. Collagen synthesis

requires hydroxylation of lysine and proline, and co-factors such as ferrous iron and vitamin C. Impaired wound healing results from deficiencies in any of these co-factors **(22).**

Wound healing is a complex biological process that consists of hemostasis, inflammation, proliferation, and remodeling. Large numbers of cell types—including neutrophils, macrophages, lymphocytes, keratinocytes, fibroblasts, and endothelial cells— are involved in this process. Multiple factors can cause impaired wound healing by affecting one or more phases of the process and are categorized into local and systemic factors. The influences of these factors are not mutually exclusive. Single or multiple factors may play a role in any one or more individual phases, contributing to the overall outcome of the healing process **(23).**

**Major Wound complication**

**Infection**

A vascularity effectively decreases the ability to combat infection: insufficient oxygen impedes neutrophils from phagocytizing bacteria. Perioperative complications were found to be significantly higher in morbidly obese patients undergoing total knee arthroplasty. It may be assumed that these complications can be easily translated to other surgeries as well. Reasons for complications in morbidly obese patients can be attributed to technical difficulties in operating on obese patients; operations taking more time, thus increasing the chances of contamination; more trauma; and even necrosis of the abdominal wall because of more forceful retraction during surgery **(24).**

**Dehiscence**

Wound dehiscence is defined as wound separation that involves all layers of the abdominal wall and it is associated with a 15 to 20 percent mortality rate**.** Surgical wound dehiscence after major gynecological surgery remains a serious complication. It presents a mechanical failure of wound healing of surgical incisions. Surgical incisions stimulate the healing process which in reality is a complex and continuous process with four different stages: Hemostasis, inflammation, proliferation, and maturation **(25).**

Explanations for the frequency of dehisced incisions among morbidly obese patients include increased tension on the fascial edges at the time of wound closure, thus increasing tissue pressure and reducing micro perfusion and the availability of oxygen **(25).**

Outcomes from different suturing techniques have been studied in several prospective randomized trials. A continuous monofilament fascial closure technique, as opposed to an interrupted technique, improves wound healing in morbidly obese patients undergoing gastric operations **(25).**

**Hematoma and seroma formation**

Collection of pooled blood or serous fluid . The formation of hematomas and seromas creates internal pressure and adds tension on sutured incisions. The etiology is multifactorial, involving inadequate hemostasis, lymphatic disruption, shearing between tissue surfaces, creation of surgical dead space, and systemic coagulopathy. Mediators of inflammation have also been implicated in the formation of seroma in the presence of surgical dead space. The normal migration of macrophages and polymorphonuclear leukocytes and release of histamines and prostaglandins cause vasodilation and production of interstitial fluid. This results in the accumulation of serous fluid in the potential cavity. Prevention of the formation of hematomas and seromas begins preoperatively with the assessment for potential coagulopathy and cessation of antiplatelet drugs and anticoagulants. Intraoperative, attention to surgical hemostasis and placement of drains serve to reduce risks. In the postoperative period, rapid evaluation and evacuation of postoperative fluid collection assist in eliminating further complications **(26).**

***James Moss*** reported upon the history of surgical drainage in an excellent comprehensive report published in 1981.the earliest recorded use of drain is published to ***Hippocrates (circa 460-377bc)*** how made use of hollow pencils to treat empyema. In the third century BC, ***Erasistratus*** of Alexandria is believed to have

used tube drainage for empyema and introduced catheter for urinary retention. During the first century AD ***Aurelius celsus*** in **Rome** used conical metal tube with an adjustable plug to drain ascetic fluid. ***Claudius galen (130-201BC)*** also describe laden tube in the management of ascites (**27).**

Knowledge of drainage practices during the Middle ages is lost. In 1363 the uses of drain was described by ***guy de chauliac***. Drainage was accomplished with atent which consisted of linen cut into small bits, and rolled into a cylinder to prevent premature closure of the wound **(28).**

Later during the renaissance, lucid descriptions of the use of tents – drains – and messes – packs –were made by ambrose pare (1510-1590). Pare's indications for drains included: wounds which required debridement or those in which pus would collected; contaminated wound, abscesses, bites, ulcerated wounds, and orthopedic procedures (**27).**

As late as 1831, the lexican medicum dictionary does not include the term drain, but defence"tent" as arol of lint for dilating openings. In the first half of the 19$^{th}$ century, agum elastic catheter was inserted per vaginum into Douglas pouch to drain the accumulated fluid and serum in the pelvis after ovariotomy.History now gives credit for the invention of the rubber drainage tube to chassaignac of france after his publication in 1859. In 1865, *It was* introduced a glass tubular drain which was modified by Keith and Wells into a cylindrical glass tube open at both ends and having side holes **(29).**

New materials for drain were introduced. It was used an absorbable tubular drain of decalcified ox bone**.** Other drains used included catgut, gutta-percha Molded rubber drains, gauze enclosed in a rubber sheath and horsehair **(28).**

drain or "tampon" was introduced, It was constructed by placing a fenestrated sheet of rubber in the area to be drained and filling it with lengthy strips of gauze. The rubber dam was folder over the gauze and both were brought out through the wound. The gauze was gradually removed over a 48 hour period allowing the dam to collapse before its removal the following day **(30).**

*It was* used tubular and capillary drain as well as combination of each. He placed gauze inside of glass tubes and rubber sheets aiming at prevention of accumulation of fluid in the peritoneum. After less than three decades of widespread use, *It was* believed that, drainage is a marker of imperfect surgery. Also *It was* believed that meticulous surgical technique and obliteration of dead space eliminated the need for drains in nonseptic instances. As prophylactic drainage produces necrosis of the contact tissues towards organisms **(31).**

Before the end of 19's century, the last innovation in drainage was the advent of the suction drain in England. It was placed a smaller perforated catheter inside the glass drain and attached water activated suction (**27).**

The use of drains began to decline gradually and the experience gained during world war I would have a major further marked reduction of their use **(32).**

The indications for the use of drains diminished to their therapeutic use in the presence of free purulent material in considerable quantity and the presence of an abscess sac "The indications for suction drain were used in deep or large abscess cavities when gravity drainage could not be achieved **(30).**

It Was used a 24 Foley's catheter after radical mastectomy and applied intermittent suction with a syringe. Closed suction drainage was made continuous in 1950 by baron of great Britain. the portable closed wound suction unit was developed by Redon and Jost who presented their experience to the surgical academy of Paris in 1954 (**27).**

**In 1950's**, the addition of a mixture of barium by the firestone rubber company resulted in the first radiopaque Penrose drains **(28).**

**Sheppard**, in 1952 was among the first to use sealed drainage of surface wounds and others have subsequently confirmed its effectiveness **(32).**

**Classification of surgical drains**

Drain can be classified based on various factor

Table (**1**) classification of drains (**27).**

| **Based on factor** | **Type** | |
|---|---|---|
| Mechanism | Passive | Active |
| Nature | Tube | Sheet\flat |
| Disposition | Open | Closed |
| Location | Internal | External |
| Property | Inert | Irritant |

**Passive drains:**

These are drain that act by the mechanism of capillary action, gravity or the fluctuation of intra-cavity pressure. Corrugated rubber drain, Penrose drain, sump drain are examples of this type. These drains are used when drainage fluid is too viscous to pass through tubular drains **(31).**

**Active drains**

These are tube drains that are aided by active suction which could be low continuous, low intermittent or high suction drainage. Jackson-Pratt drains, Surgivac drain, Redivac drain are examples. Reliable measurement of effluent can be done. There decreased risk of wound infection, minimal tissue trauma and no skin excoriation. However, regular activation of reservoir is often required (**27).**

**Table (2)** the major difference between active and passive drains (**27).**

| | **Active** | **Passive** |
|---|---|---|
| Function | Work by active suction | Depend on pressure differentials |
| Pressure gradient | Negative pressure (low ,moderate and high) | Positive pressure |
| Drain exit site | Dependant position not necessary | Dependant position necessary for best function |
| Drain site dressing | Minimal or not required | Bulky to absorb fluid output |
| Measurement of effluent | Reliable and accurate | Difficult to quantify |
| Fluid recollection | Unlikely because negative pressure improve tissue apposition | Likely because of limited effect on the dead space |
| Retrograde infection | Lower incidence | High incidence |
| Obstruction of drain | More common | Less common |
| Radiographic studies | Easy to perform | Difficult except in special circumstances like T-tube |
| Pressure necrosis | High incidence | Low incidence |

**Tube drain**

These are hollow tubes of varying materials brought out through a body orifice or stab wound. when they are connected to a bag they become closed but when left alone they remain open drains multiple holes on the end are necessary and essential in case one hole becomes blocked **(33).**

**Sheet drain**

These are drains mad in sheet of gutters or parallel tubes through which fluid passes. corrugated rubber drain, which the fluid tracks through the gutters to the surface, is one commonly used example of this type of drain **(34).**

**Flat drain**

These are drains that are made flat with 3/4 or full length multiple perforations which can be connected to a tubing system, thus, convert it to a close system or left opened. The inner wall of the flat segment usually has internal ribs to prevent it from collapsing or kinking. They are often used for various surgeries, including plastic and reconstructive surgery **(35).**

**Open drain**

These drain empty directly to the exterior into the overlying wound dressing or stoma bag. Corrugated rubber drain, Penrose gauze wick drain and glove finger drain are examples of this type of drain they are mostly used in superficial wounds and cavities. Drained fluid collects in gauze pad or stoma bag which can easily be changed. It's simple and easy to apply. However, it's often difficult to measure the effluent. High rate of wound infection, trauma to the skin from repeated changing of dressings, skin excoriation and erythema due to irritation by the effluent has been noted **(33).**

**Closed drains**

These are hollow tubes of varying materials brought out through a body orifice or stab wound and are connected to closed system of sterile drainage bag. Under water seal drainage system is an example. This drain is mostly used in deep cavities the risk of skin excoriations and surgical wound infection is less. Effluent can easily be collected and measured. However, reflux of the content of a contaminated reservoir has been noted (**27).**

**External drains**

These are that are brought out through the body wall to the exterior. The fluid discharge is channeled from the deepest part of the cavity to the exterior. This can be passive or active drain **(34).**

**Internal drain**

These are drains that are placed internally within luminal organs to create a route or to connect tow luminal organs. They divert retained fluid from primary drainage site or area to distal body passage or cavity in order to bypass an obstruction. They are used in neurosurgery for internal drainage of hydrocephalus, in gastrointestinal surgery where other tube Celestine tube and mousseaubarbuntube could be used to palliate malignant obstruction of the esophagus (**27).**

**Irritant drains**

These are drain made of materials that are irritative to the tissue and so are capable of exciting fibrous tissue response leading to fibrosis and track formation example are latex, plastic and rubber drains **(34).**

**Inert drains**

This group of drains is non-irritative to the tissue and so ideally do not provoke tissue fibrosis. Examples include polyvinyl chloride, silastic and silicone drains. (**27).**

**Figure (1 a)** penrose drain (**27).**

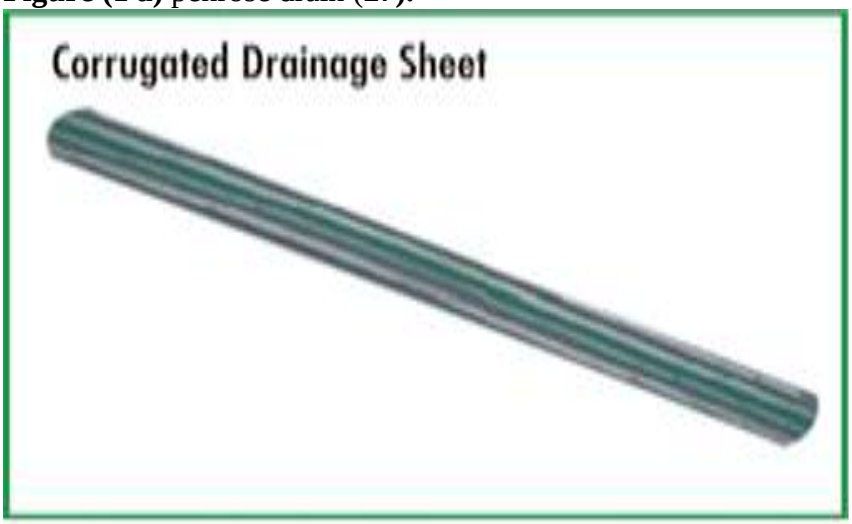
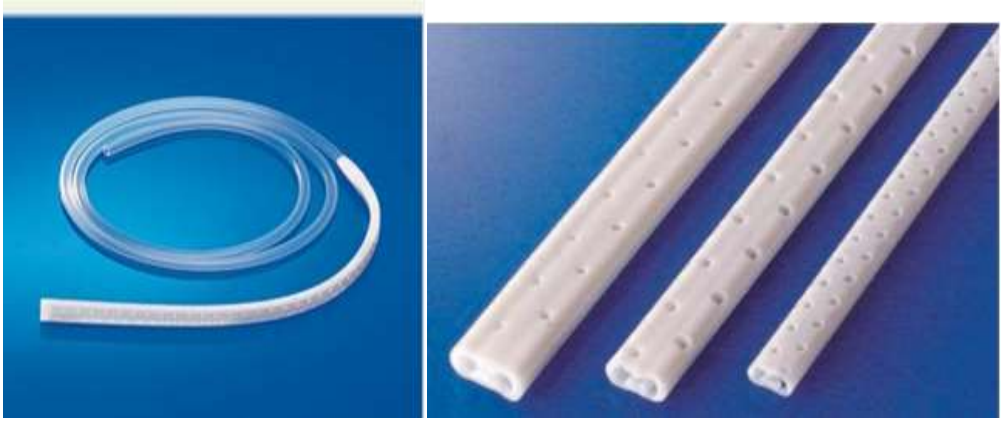



**Figure (1 b)** sheet of corrugated rubber drain (**27).**

Figure(**1 C**) redivac drain (**27).**Fig (**1** d) close tube drain (**27).**

**Figure(1 e) tube drain open (27).**



**Fig(1 f) flate drain (27).**

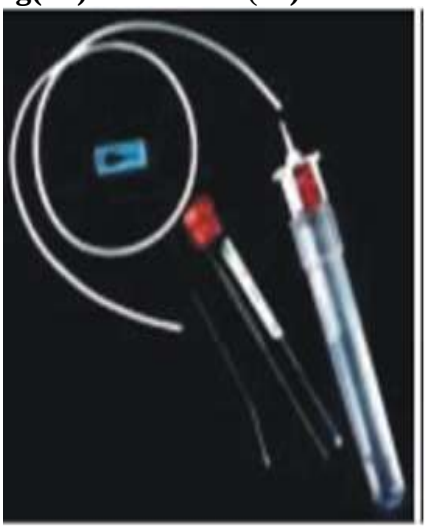


**Fig (1 g)close tube (27).** **fig(1 h) gauze wick drain**

**Purpose and indication of drain**

- **Therapeutic:**-drain permits the exit of gases and liquid and could be used to treat conditions like hydrocephalus,urinary retention, and abscess cavity
- **Palliation**:- It could be used as a palliative measure to bypass a Luminal obstruction
- **Diagnostic:-** T-tube cholangiogram as apost cholecystectomy diagnosis of retained stones in the common bile duct'
- **Prophylactic**: To prevent postoperative complication that could arise from fluid accumulation in a wound cavity

- **Monitoring:-** For instance, monitoring progress by Nasogastric tube in a patient with upper gastro intestinal bleeding monitoring of urinary output
- **Access route:-** for percutaneous therapy e.g useful in percutaneous nephrolithotomy

**(36).**

**Complication of drain**

- **Tissue reaction** particularly when irritant drains are used may be enormous and detrimental. Careful selection and Use of non-irritant drains should prevent its complication
- **Source of contamination: -** the fact that a drain is a conduit allows opposite traffic within it, thus, increasing the possibility of surgical site infection. However, strict aseptic and proper drain care, if observed will limit rate of surgical site infection. Occasionally, antibiotic cover may be necessary particularly in susceptible drains
- **Delayed return of function**:- limitation of movement inpatient with surgical drain may cause a delayed return of function. Early mobilization is paramount in this case
- **Retained foreign body:-**

This may be possible when the drain disintegrate following enzymatic action, trauma or undue traction, Proper selection of drain ,adequate care and prompt removal after use will suffice.

- **Tissue necrosis:-** from pressure of very hard or stiff drain may be prevented by the use of soft drain
- **Bowel herniation: -**May occur through the weak drain site particularly when it was complicated by infection. Proper drain Insertion technique and meticulous care will prevent this complication occasionally, the drain site may need to be closed by one or 2 sutures to prevent herniation
- **Hemorrhage: -** Occurs during insertion or from repeated injury of the surrounding tissue, especially during mobilization and change of dressing. A stiff drain may also precipitate bleeding if it erodes into a large vessel. If this continuous, the drain should be removed under vision and hemostasis secured

(**37).**

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