THE ROLE OF THE SURGICAL TECHNOLOGIST IN

Wound Management

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Most surgical wounds are the result of a planned procedure and involve precise incisions that cause minimal tissue damage and minimize the risk of infectious complications.[1] However, skin wounds may also result from a wide variety of physical insults, trauma and idiopathic causes.[2]

Rapid and effective wound healing is of paramount importance to the surgeon and to the patient. Failure of wound healing generally leads to potentially life-threatening complications, additional surgical procedures, increased length of hospital stay, increased cost, and long-term disability.[3]

This article provides an overview of the wound healing process and seeks to educate the surgical technologist on how to assess, classify and care for patients with surgical wounds, using evidence-based practice.

THE NORMAL HEALING PROCESS

The healing process begins following a breach in skin integrity and is described as an orchestrated, systematic interdependent, but overlapping process that leads to eventual repair.[4] Wounds heal by either primary or secondary intention.[5] A full thickness surgical incision will be repaired by primary intention.[6]

In primary intention, the wound edges are brought together and held in place by sutures, skin glue or adhesive strips. Within 24–48 hours, the epidermis will have covered the surface of

LEARNING OBJECTIVES

- Summarize the physiology of the wound healing process
- Identify the factors that affect wound healing
- Analyze the principles of moist wound healing and its influences on modern day management
- Accurately assess and classify surgical wounds
- Identify and implement appropriate wound-dressing methods/strategies

the wound, but the healing process will still be continuing underneath.

Healing by secondary intention occurs where there has been an extensive loss of tissue, which means that the wound edges may not be brought together and so the wound has to heal through the process of granulation and epithelialisation.[5] This is a more "chronic" healing process and takes much longer. An example of a wound healing by this method would be the regeneration and repair of a pressure ulcer.

Surgical technologists will mainly be associated with surgical wounds, so this article will concentrate on the acute healing process.

The sequence of events involved in wound healing, whether it is by primary or secondary intention, can be divided into four main stages: hemostasis, inflammation, proliferation and maturation.

Table 1 shows the major phases of wound healing and the interrelated concomitant events, also including information of the cells used to orchestrate these processes.

TABLE 1: THE PHASES OF WOUND HEALING

Phase of Healing	Days Post Injury	Cells involved in the Phase
Hemostasis	Immediate	Platelets
Inflammation	Days 1-7	Neutrophils
Proliferation	Day 3-20	Macrophages
Granulation		Lymphocytes Angiocytes Neurocytes
Contraction		Fibroblasts Keratinocytes
Maturation (Remodelling)	Day 21-2 years	Fibrocytes

HEMOSTASIS

Any skin trauma, surgical or otherwise, that results in the penetration of the dermal layers within the skin, will result in bleeding.

Hemostasis is defined as "the cessation of bleeding following injury," with the amount of bleeding being dependent on the site of wound, size of the blood vessels involved, state of the individual's health and anticoagulation status.[7] Under normal circumstances, this process occurs within 10 minutes of wound formation.[8]

When injured, blood vessel surfaces attract platelets to the site of injury. Platelets adhere, aggregate and form a procoagulant surface, promoting both the generation of thrombin and fibrin.[9] This promotes clot formation and subsequent platelet degranualtion, which releases platelet-derived growth factor (PGDF), a substance that triggers the clotting cascade, which results in vasoconstriction of the affected blood vessels, reducing the blood flow.[10] Hemostasis is also classified as the early inflammatory stage of wound healing.[11]

INFLAMMATION AND WOUND HEALING (1–7 DAYS)

Inflammation is a highly complex cellular surveillance system that is essential for both wound healing and antimicrobial defence.[12] It has long been considered that the inflammatory response during wound healing is instrumental to supplying growth factor and cytokine signals that orchestrate the cell and tissue movements necessary for repair.[13] There are two essential elements to the inflammatory events, namely the vascular and cellular cascades. These occur in parallel and are significantly interlinked.[14] (See figure 1.)

VASCULAR EVENTS:

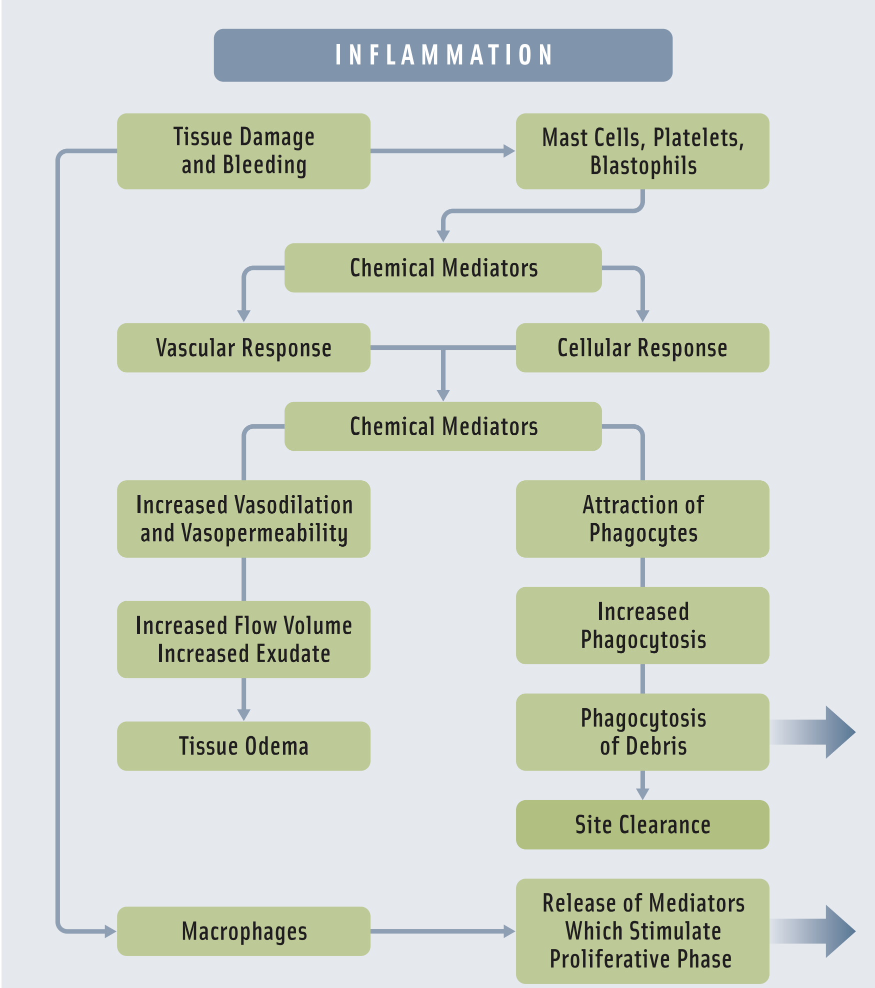
This stage signifies some marked changes in the caliber of the blood vessels, through morphological changes of the vessel wall and also in the flow of the blood through the vessels, which becomes turbulent.[14] This gives rise to the classic signs of inflammation as seen at the wound, which are described in Table 2.

It is important to note that these signs and symptoms of inflammation after wounding are the same as the inflammatory process associated with tissue infection. This needs to be ruled out for the purposes of patient safety.[8]

TABLE 2: SIGNS AND SYMPTOMS OF INFLAMMATION[8, 11]

Signs/Symptoms	Physiological Changes
Rubor	Results from vasodilatation, mediated by prostacyclin and prostaglandins
Calor around the wound bed	Results from increased vasodilatation and increased metabolic activity
Tumor (swelling) in an around the wound bed.	Vascular endothelial gaps enlarge allowing the progression of plasma protein and fluids into the interstitial spaces
Dolor (Pain)	Increased pressure from oedema in the tissues, prostaglandins which irritate the nerve endings and damage to nerve endings.

FIGURE 1: RELATIONSHIP BETWEEN VASCULAR AND CELLULAR CASCADES DURING INFLAMATION



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CELLULAR EVENTS:

The cellular components of the inflammatory response include the early emigration of the Polymorphonucleocytes (PNMs) to the wound site. The process of chemotaxis also attracts several other white blood cells to the wound bed. These include monocytes, leucocytes, eosinophils and basophils.[15] Neutrophil leucocytes may be regarded as the first line of defense against infection at the wound site as they are described as actively phagocytic.[4] This phagocytic activity involves clearing the wound site of dead and devitalized tissue, and also to neutralize and destroy any toxic agents at the site of injury, restoring tissue homeostasis.[16] The process of phagocytosis also releases lactic acid, which is one of the stimulants for proliferation in the next sequence of wound-healing events.[14] A recent study has also highlighted that lymphocytes secrete a selection of lymphokines, which may assist in enhancing the rate of wound closure.[16]

Towards the end of the inflammatory phase, the eicosanoids, which are chemical mediators generated from the inflammatory process, stimulate the synthesis of collagen from fibroblasts and the "ground substance." This ground substance contains water, electrolytes, glycoproteins and a specific class of compounds known as proteoglycans, which are vital for cell-to-cell and tissue adhesions.[17] In addition, macrophage-derived growth factors are now at optimal levels, which is required for the influx of fibroblasts, keratinocytes, and endothelial cells into the wound.

The inflammatory stage of wound healing is complex and metabolically demanding. Thus it is of importance to note that any patient who may also present with diabetes or anaemia may experience a delay in the healing process.[4]

PROLIFERATION (3–20 DAYS)

Proliferation refers to the development of granulation tissue, which takes place over a 28-day period. It involves the migration of fibroblasts, which begin to produce glycosaminoglycans, proteoglycans and the ground substance for granulation tissue and collagen. This is known as the formation of the extra cellular matrix (ECM).[8] Newly-formed capillaries infiltrate the wound site to nourish and support the development of this connective tissue, a process known as angiogenesis.[4] Angiogenesis takes place in distinct steps involving growth factors, cells and the ECM. Unregulated or insufficient vessel growth will result in delayed healing.[18]

Some of the fibroblasts differentiate into specialist myofibroblasts, which are responsible for the process of contraction. Contraction is defined as the pulling of wound edges together as the myofibrils start to contract around the wound edge. The purpose of this process is to reduce the amount of tissue required to fill the wound bed.[7]

MATURATION/REMODELLNG (21 DAYS–2 YEARS)

This is the final stage of healing and can range from 21 days to two years. During this phase, the wound undergoes re-epithelialization, whereby macrophages release epidermal growth factor (EGF), which is responsible for stimulating the growth proliferation and migration of epithelial cells across the wound, covering the granulation tissue.[8]

As the epithelial cells meet in the middle of the wound, the migration stops and the initial cells reconstruct to form a basement membrane. This basement membrane is of great physiological importance as epithelial cells can be easily sheared off the surface during wound dressing changes or vigorous wound cleaning.[5] Although, the production of collagen enhances the tensile strength of the new tissue, it should be noted that this new tissue is not as strong as the original.[4]

The wound at this stage is covered in scar tissue, which, along with the granulation tissue, is remodelled and strengthened over the course of the following one to two years.[7]

MOIST WOUND HEALING

The concept of a moist wound healing environment has been promoted since the early 1960s.[19] The process was first demonstrated in both humans and animals, which observed that by keeping wounds moist, the rates of healing were much quicker than those left to dry out under tensile-based dressings.[20] Moisture in a wound acts as a transport medium for essential growth factors during epithelialisation and also promotes autolytic debridement.[4] Therefore, dry or dead tissue would inhibit wound healing. Moist wound healing has many other clinical benefits as shown in sidebar at right.

The Clinical Benefits of Moist Wound Healing

- Prevention of wound infection[20]
- Improved rates of healing
- Reducing pain[21]
- Reduced scarring
- Reduction in nursing hours spent with dressing changes[22]
- Reduced costs to the NHS

CLASSIFICATION OF SURGICAL WOUNDS

According to Devaney & Rowell, surgical wound classification is an important predictor of the risk of postoperative surgical site infections and their associated risks. A standardized wound classification system has been in place since 1964, whereby all surgical wounds are classified according to their levels of risk of contamination. Table 3 identifies these classifications and gives some general descriptions of wounds within each category, including examples of the procedures from which these wounds have evolved.

Recent research has found that the management of wounds resulting from excision and drainage of the condition pilonidal sinus, caused by in-growth of hair in between the buttocks is controversial. These wounds are classified as dirty/infected and are therefore at risk for post operative wound infection. A recent systematic review has proposed that no clear benefit is apparent from either closure or healing by primary intention as compared to open healing by secondary intention.[24]

TABLE 3: SURGICAL WOUND CLASSIFICATION[23]

Classification	Wound Description	Surgical Procedure Associated with Wound Type	Infection Rate
Clean	• Surgical incision wounds/nonpenetrating • Surgical wounds that do NOT involve the respiratory, alimentary or genital tract	• Exploratory laparotomy • Prosthetic joint replacement • Vascular surgery	1-5%
Clean/ Contaminated	• Surgical wound that involves surgery within the urinary, alimentary, respiratory tract. • No breaks in sterile technique throughout the procedure	• Trans urethral resection of prostate • Bowel resection with formation of colostomy • Bronchoscopy with biopsy	8–11%
Contaminated	• Procedures that have major breaks in sterile procedures including contents from the gastrointestinal tract	• Bile spillage during cholecystectomy • Surgery for diverticular disease	15–20%
Dirty/Infected	• Infected viscera prior to surgery, secondary to the presence of abscess.	• Surgery for perforated appendix/bowel and formation of colostomy • Peritonitis • Insertion of grommets for Otitis media.	27–40%

FACTORS AFFECTING WOUND HEALING

The majority of wounds will heal normally without delay or complications. However, the capacity of the wound to heal swiftly is determined by intrinsic and extrinsic factors that will vary considerably between individual patients.[25, 26] Table 4 identifies factors that the surgical technologist will need to consider when assessing surgical wounds.

TABLE 4: INTRINSIC AND EXTRINSIC FACTORS WHICH AFFECT WOUND HEALING

Intrinsic Factors	Extrinsic Factors.
Age	Nutrition
Disease process including • Diabetes • Peripheral Vascular Disease • Jaundice • Renal disease	Smoking
Wound perfusion	Radiotherapy
Oxygen tension	Wound Infection
Abnormal scarring	Medication Anti Inflammatory Drugs Immunosuppressant therapy

Whilst all these factors are of considerable importance, the role of nutrition is deemed to be a critical component in the wound healing process.[3] It is widely recognized that when patients are in a poor nutritional state, wound healing is impaired and more likely to be complicated by infection.[27] To this end, this section of the review will highlight the nutrients involved in the wound healing process.

An individual's nutritional intake consists of both macronutrients, which include carbohydrates, fats and proteins, and micronutrients, which include minerals and vitamins. All of these substances have been shown to play a vital role in the wound healing process.[28] In addition, it is important to note that fluid status is an essential part of nutrition, as this maintains adequate perfusion to the wound site, which is critical for transportation of both oxygen and nutrients.[29]

It is important to identify any patient who may be at risk of malnutrition by performing a nutritional assessment. Should the patient be found to be at risk for, or be suffering from malnutrition, it is important to devise a suitable nutrition care plan, which will optimize their nutritional intake, thus promoting wound healing.[30]

Early postoperative feeding has been shown to improve wound healing, and commencement within 24 hours of surgery is associated with optimal clinical outcome.[31] Indeed, early food intake, or enteral feeding, which utilises the gut, as opposed to parenteral feeding, which delivers feed intravenously, is also recommended to promote enhanced recovery of patients after surgery.[32] Regular food should contain sufficient energy and nutrients for the vast majority of patients and should be tried prior to any thoughts of possible nutritional support.[33]

Any patient who has failed to achieve their optimal nutritional status through oral feeding, and those who cannot or will not eat may be candidates for enteral tube feeding.[35]

It is also important to note that individuals with infected wounds have an increased requirement for energy, protein and other nutrients, which is secondary to losses of wound exudate and tissue granulation and may therefore benefit from nutritional support.[30]

To this end, it is imperative that both the patient's wounds and nutritional status are assessed on a weekly basis in the hope that this may prevent the development of both wound infection and malnutrition.[36]

WOUND ASSESSMENT AND DRESSINGS

There are currently many sophisticated dressings available, made from a variety of materials, which can be used alone or in conjunction with other forms of dressings.

There are also several attributes of an ideal surgical wound dressing that surgical technologists should take into consideration prior to using any dressing. These are described in sidebar below.

Attributes of a Surgical Wound Dressing[37, 38]

- The ability of the dressing to maintain a moist environment
- Ability of the dressing to absorb and retain exudate without leakage
- Enable gaseous exchange
- Allow ongoing wound assessment
- Absorb wound odor
- Avoidance of wound trauma on removal
- Cost effective and covered by health insurance systems
- Lack of particulate contaminants from the wound dressing
- Promote effective scar formation
- Easy to use
- Require infrequent changing

The dressings used should be easy to apply, painless on removal, allow earlier discharge from the hospital and require fewer dressing changes.[39] The care of wounds and dressings used in wounds healing by primary intention, for example surgical wounds, are generally straight forward. The contact layer of the dressing placed directly over the wound is the most important, as it is required to provide protection from external contamination and absorb exudate. Straight forward surgical wounds that are likely to heal quickly, without complications, require simple, low-cost adhesive film dressings that are transparent, stay in situ for several days, and allow observation.[39] The contact dressing must be able to maintain moisture, permit respiration and allow epithelialisation.[17]

Some acute surgical wounds may be much deeper, causing trauma to underlying tissues, which may result in prolonged bleeding. These types of wounds may benefit from an additional layer of gauze or absorbent pads that provide compression and are classed as secondary dressings. These secondary dressings must not be too absorbent, as they may cause the primary dressing to dry out too quickly and delay the healing process.[40]

It is important to recognize that every patient is an individual and the surgical technologist should take into account the patient's underlying condition, for example whether the patient has diabetes, or any other factors that might delay the wound healing process, prior to making the choice of dressing.[41]

Table 5 identifies the factors that should be taken into account when deciding which dressing to use.

THE NUTRITIONAL EVIDENCE

Nutrient	Role in Wound Healing
Protein *(Requirements should be calculated on an individual basis and monitored closely)*	• Synthesis of new tissue • Optimization of tensile strength • Collagen Synthesis
L Arginine	• Optimizes tensile strength of the wound • Enhances immunity • Improves secretion of growth hormone
Carbohydrate	• Source of energy • Required to prevent protein being used as a source of energy
Fats	• Major source of energy supplying 9 kcal/g • If an individual is overweight, low fat foods may be better choices. • Aim for weight maintenance not weight loss as this will compromise wound healing • Evidence equivocal surrounding the use of omega 3 supplementation and wound healing
Vitamin C	• Vital for collagen synthesis and subsequent cross-linking • Required for angiogenesis • Optimizes tensile strength • Increases the absorption of iron • Boosts immunity • Natural sources from fruit and vegetables are best.
Vitamin A	• Stimulates collagen synthesis via the inflammatory response • Improves cell mediated immunity • Promotes granulation of tissue • Avoid supplementation as this could cause toxicity
Vitamin E	• Antioxidant effect, which can prevent cellular membrane damage
Vitamin B	• Required for release of energy from carbohydrate metabolism
Vitamin K	• Coagulation
Zinc	• Key role in protein and collagen synthesis • May have antibacterial action • Component of many enzymes
Iron	• Involved in collagen synthesis • Optimises tensile strength of the wound • Optimises tissue perfusion by supplying oxygen • Iron deficiency anaemia can delay the wound healing process
Hydration	• Dehydrated skin is less elastic, more fragile and susceptible to breakdown • Dehydration causes a reduced circulating volume and leads to poor perfusion
Copper, Selenium, Manganese & Chromium	• The physiological role in wound healing is apparent but unclear. More research is required to identify and quantify these roles.

TABLE 5: FACTORS TO CONSIDER WHEN DECIDING WHICH DRESSING TO USE[40, 42]	
Factors related to the patient	Type of wound Level of exudate Location of wound Size of wound Likelihood of wound contamination? Depth of wound Dressing cost
Wound assessment	Suitable Dressing
Closed wound healing by primary intention	Simple dressing/adhesive film *(These are semi permeable and allow gaseous exchange/impervious to bacteria)*
Superficial partial thickness	Adhesive film/foam *(Foam dressings are in the form of sheets/liquid and expand to fill a wound cavity)*
Mild to moderate exudate	Hydrocolloid dressing *(Hydrocolloid dressings promote a moist wound healing environment)*
Contaminated wound	Alginate *(Alginate derived from seaweed and in the form of a loose fibrous pad/rope)*
Heavy exudate	Hydrogel/hydrocolloid

CONCLUSION

Wound healing is a highly complex physiological phenomenon, with many factors. Age, nutritional status and general health all play a role in the healing process. By understanding the physiology of wound healing, surgical technologists will gain greater insight into the importance of how their skills can impact the body's healing response.

ABOUT THE AUTHOR

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REFERENCES:

1. Leaper DJ, Harding KG (2006) ABC of Wound Healing. Accessed online at ***http://student.bmj.com/issues/06/07/education/273.php***
2. Lansdown ABG (2007) The role of the pathologist in wound management. British Journal of Nursing. (Tissue Viability Supplement. Vol 16 (20) pp S24 S33
3. Demling RH (2009) Nutrition, anabolism, and the wound healing process: An overview. Eplasty Epublication 2009. February 3, 2009.
4. Benbow M (2008) Exploring the concept of moist wound healing and its application in practice. British Journal of Nursing (Tissue Viability Supplement) Vol 17 (150 S4-S16
5. Lloyd-Jones M (2007) Wound assessment: the role of the healthcare support worker British Journal of Healthcare Assistants: Vol 2 (3) pp 124-126.
6. Mercandetti M, Cohen AJ (2008) Wound Healing and Repair. Accessed online at ***http://emedicine.medscape.com/article/1298129-overview.***
7. Timmons S (2006) Skin Function and Wound Healing Physiology In Wound Essentials Wounds UK Aberdeen.
8. Thompson G & Stephen Haynes J (2007) An overview of wound healing and exudate management. Wound Care December 2007 pp S22-S30.
9. Nurden AT, Nurden P, sanchez M, Andia I et al (2008) Platelets and wound healing. Front Biosci May 1: 13: 3532-48.
10. Rozman P, Bolta Z (2007) Use of platelet growth factors in treating wounds and soft-tissue injuries. Acta Dermatovenerol Alp Panonica Adriat. Dec; 16(4): 156-65
11. De La-Torre J, Chambers J (2008) Wound Healing Chronic Wounds. Accessed online at ***www.emedicine.medscape.com/article/1298452-print***
12. Glaros T & Larsen M (2009) Macrophages and fibroblasts during inflammation, tissue damage and organ injury. Frontiers in Bioscience 14, 3988-3993, January 1, 2009
13. Leibovich SJ, Ross R (1975) The role of macrophage in wound repair. Am J Pathol 78: 71-100.
14. Watson T (2006) Soft Tissue Healing and Repair. Accessed online at; ***www.electrotherapy.org***
15. Lorena D et al (2002) Normal Scarring: Importance of myofibroblasts. Wound Repair & Regeneration. 10 (2): pp 86-92.
16. Keen D (2008) A review of research examining the regulatory role of lymphocytes in normal wound healing. Journal of wound Care 17 (5): 218-220
17. Irvin TT (1985) Wound Healing. Emergency Medicine Journal. 2;3-10 doi:10.1136/emj.2.1.3.
18. Chan LKW (2009): Current thoughts on angiogenesis. Journal of Wound Care: Vol 18 (1): pp 12-16
19. Winter GD (1962) Formation of scab and the rate of epithelialisation of superficial wounds in the skin of young domestic pigs. Nature 193 pp 293-4
20. Slater M (2008) Does moist wound healing influence the rate of infection? British Journal of Nursing (Tissue Viability Supplement) Vol 17 (20) pp S4-S14
21. Gottrup F, Jorgensen B, Karlsmark T et al (2008) Reducing wound pain in venous leg ulcers with Biatain Ibu: a randomized controlled double-blind clinical investigation on the performance and safety. Wound repair Regen. Sep-Oct; 16(5): 615-25.
22. Metzger S (2004) Clinical and financial advantages of moist wound management. Home Healthc Nurse. Sep;22(9): 586-90.
23. Devaney L, & Rowell K (2004) Improving Surgical Wound Classification – why it matters *AORN J.* 2004;80:208-299, 212-223.
24. McCallum I J D, King PM, Bruce J (2008) Healing by primary closure versus open healing after surgery for pilonidal sinus: review and meta analysis. BMJ doi: 10.1136/bmj.39517.808160.BE Published April 7th 2008
25. Brown J (2009) Intrinsic and Extrinsic Factors Affecting Wound Healing. Accessed online at Judith Brown online CPD. ***www.judithbrowncpd.co.uk***
26. Dowsett C (2002) The management of surgical wounds in a community setting. Wound Care June 2002. pp 33-38
27. Edmonds J (2007) Nutrition and wound healing: putting theory into practice. Wound Care December 2007 S31-S34
28. Anderson B (2005) Nutrition and wound healing: the necessity of assessment. British Journal of Nursing. (Tissue Viability supplement) Vol 14 (9) S 30-S38
29. Scholl D, Langkamp B (2001) Nutrient recommendations for wound healing. J Intravenous Nursing. 24 (2): 124-32
30. Ord H (2007) Nutritional Support for patients with infected wounds. British Journal of Nursing Vol 16 (7): pp 1346-1352
31. ALLISON, S.P. and ROWLANDS, B.J., 2006. Early postoperative jejunostomy feeding with an immune modulating diet in patients undergoing resectional surgery for upper gastrointestinal cancer: a prospective, randomized, controlled, double-blind study. Clinical Nutrition, 25(5), 716-726.
32. Weimann A, Braga M, Harsanyi L et al (2006) ESPEN Guidelines on Enteral Nutrition. Surgery including organ transplantation. Clin Nurs 25 (2): 224-4
33. Shepherd AA (2009) Nutrition Support 2: exploring different methods of administration. Nursing Times 10 February vol 105 (5): pp 14-18.
34. Thomas B, Bishop J (2007) Manual of Dietetic Practice. 4th Ed Blackwell Publishing Oxford.
35. National Institute of Clinical Excellence (2006) Nutritional Support in Adults. Oral Nutritional Support, Enteral Tube Feeding and Parenteral Nutrition. Clinical Guideline 32. NICE London
36. National Institute of Health and Clinical Excellence (NICE) (2006) Clinical Practice Guideline for Surgical Site Infection: Prevention and Treatment: Available at ***http://tinyurl.com/5ux79q***
37. Ruszczack Z (2009) Surgical Dressings: Accessed online at ***http://emedicine.medscape.com/article/1127868-overview***
38. Wynne R, Botti M, Stedman H, Holsworth L et al (2003) Effect of three wound dressings on infection, healing comfort and cost in patients with sternotomy wounds, A randomized controlled trial. Accessed online at ***www.chestjournal.org/content/125/1/43.full***
39. Foster L, Moore P (1997) The application of a cellulose based fibre dressing in surgical wounds. Wound Care 6 (10): 469-73
40. Foster L, Moore P (1999) Acute surgical wound care 3: fitting the dressing to the wound. British Journal of Nursing. Vol 8 (4) pp 200-205
41. Vowden K (2004) Wound management: The considerations involved in dressing selection. Nurse Prescribing Vol 2 (4) pp 152-156
42. Sharp K, Mc-Laws ML (2001) Wound dressings for surgical sites. Cochrane Database of Systematic Reviews. Issue 2 Art No: CD003091. DOI 10.1002/14561858.CD003091