Bedsore: “top to bottom” and “bottom to top”

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Abstract Bedsore is a global problem concerning the bedridden, infirm, debilitated and malnourished patients in hospitals and community setups. The cost of treatment is enormous involving billions of dollars to nations and individuals. Mortality increases two to six times if bedsores are present. There is little research done despite its commonness to understand how they occur or why they occur; etiology is not much understood. The two theories called ‘top to bottom’ and ‘bottom to top’ contradict each other. It is thought that ‘pressure’, ‘shear-stress’ and ‘ischemia’ may be causing it in some yet to be understood way.

There is little awareness on how to prevent them or how to treat them if they do occur. Seldom applied, various scales exist and should be used to identify patients at high risk. Braden scale is the most tested and widely accepted scale. The various available dressings and pressure relief devices are mostly inadequately studied; which is superior is a question that begs an answer. This article aims to underline the importance of bedsores by reviewing our current and past knowledge with emphasis on practical implications thereof.

Keyword Pressure ulcer · Bedsore · Decubitus ulcer · Ischemia

Introduction

Bedsore is also known as pressure sore, friction sore, decubitus ulcer¹ or mattress ulcer. It is the bugbear of all physicians who deal with bedridden, sick, old and malnourished patients. It may complicate their course, worsen their prognosis, delay their discharge or lead to their death. Annually, treating pressure sores costs £1.5 billion in the United Kingdom and more than \$3 billion in the United States [1]. It is estimated that in an average 600 bedded hospital in UK² it costs 600,000–3 million £ per year to treat and prevent bedsores and 40,000 £ per patient to treat a grade IV bedsore [2]. Patients with bedsores have mortality 2–6 times that of those without bedsores, all other factors remaining the same [2].

Incidence and prevalence³

Globally bedsore is observed in bedridden, old and debilitated patients. Incidence rates in hospitalized patients vary from 0–65.6% in USA and Canada and 2.2–60% in UK. The prevalence of pressure ulcers is 3–11% in hospitalized patients,

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¹This term is derived from Latin word “decumbere” meaning to lie down or recline.

²Study was commissioned by the National Health Service of United Kingdom

³“Incidence” means the rate at which new bedsores develop in a defined population over a specified time period while “prevalence” refers to number of patients having bedsore at a point of time.

2.5–24% in nursing-home patients and 17% in adults in community [3].

The principal risk factors include conditions that lead to patient being bed-ridden, incontinent and unable to turn sides. The risk factors are listed below [1, 3].

Local risk factors:

- Unrelieved pressure (lying in same position > 2 hours)
- Capillary occlusion and disruption of lymphatic drainage
- Shearing force (aggravated by head-end elevation > 45°)
- Increased temperature and moisture (bed-soiling due to incontinence, poor nursing care, poor ventilation)

Systemic risk factors:

- Ageing
- Decreased mobility (fractures, paraplegia, coma, major surgery)
- Poor nutrition (hypoproteinemia, poor oral intake)
- Arterial disease and hypotension

‘Top to bottom’ and ‘bottom to top’

Our understanding as to why and how bedsores occur is far from being complete. There is no satisfactory animal or bio-mechanical model to help research. Compared to humans, laboratory animals which have tight skins like pig develop pressure sore at low pressures while animals having loose skin like rats develop pressure sores at high pressures; most laboratory animals have a thick protective hair-coat which humans lack. These animal models do not reflect human skin’s anatomy or physiology. Our understanding of bedsores is mostly from experiments conducted on dogs (greyhound dogs are particularly vulnerable for pressure sore formation due to their peculiar body-structure) by Kosiak et al beginning 1959. Theirs was the first attempt to understand how pressure sores occur and is therefore called “cornerstone of modern pressure ulcer research” [3, 4].

A combination of pressure, shear and friction creates bedsores. *Pressure* and *ischemia* are thought to be central to the etio-genesis of bedsores; these are complicated by *shear forces* due to friction. Chronic wetness of the skin further aggravates this situation by favoring colonization of the damaged skin by bacteria, mostly anaerobic [5, 6].

Soft tissue and skin over bony prominences have tenuous blood supply. Thin-walled penetrating arteries come out of bone and travel upwards through the muscle and deep fascia to form anastomoses in deeper layer of subcutaneous tissue and in dermis. The branches wherefrom supply the overlying skin. When a patient lies over hard surface, the skin gets compressed between the bony prominence and the

surface underneath. This generates unusually high compressive ‘tissue pressures’ within skin and subcutaneous tissues. Such high tissue pressures exceed capillary filling pressure within the soft tissues (about 32 mm Hg), occluding dermal capillaries and causes ischemia if not relieved. Friction due to layers of skin sliding over each other generates ‘shear stresses’ within compressed tissue and complicates matters by tearing delicate blood vessels [6]. Pressures within subcutaneous tissue can not be measured clinically hence compressive force on the skin surface (between skin and the bed underneath) using a pressure-measuring probe is measured instead; this is called ‘interface pressure’ [3–6].

In experiments, evidence of soft tissue injury ensues in dogs if interface pressure of 70 mm Hg is maintained for 2 hours or more [5]. There are no human studies but it is based on above data that 60 mm Hg interface pressure for 1–1.5 hours is accepted as *maximum* that can be tolerated by human skin [6]. Healthy patients shall change sides within this time on their own, relieving pressure and preventing tissue damage. This simple protective mechanism is unavailable to an unconscious or paralyzed patient furthermore patients are often intentionally paralyzed in ICUs to facilitate mechanical ventilation.

The older etiological theory Kosiak et al called ‘*top to bottom*’ theory was a simple model. It was believed that ischemia due to compressive occlusion of dermal capillaries lead to loss of overlying epidermis first, producing a superficial ulcer which grows deeper as compressive ischemia of underlying tissue impedes healing [4, 5]. Experiments failed to support this theory. During studies on animals it was noted that bedsores developed *first in subcutaneous tissues*. Also muscles and subcutaneous tissue were found to be more vulnerable to compressive ischemia and necrosis than epidermis and dermis which could withstand higher compressive pressures of up to 150 mm Hg without showing damage [3–7].

Another theory called ‘*bottom to top*’ theory has been proposed to explain these findings but has little scientific proof behind it. It theorizes that shear stress due to friction avulses delicate vessels at the junction of bone and soft tissue. It should be noted that *friction* and not compression is the culprit in this theory. With vessels avulsed ischemia follows; the first to die is soft tissue next to bone. The developing abscess then bursts onto the skin-surface having traveled up [7]. This theory is unable to explain why some bedsores are superficial ulcers.

To detect bedsores and gauge their severity care-givers must rely on clinical signs. The clinical implications of the two theories mentioned before are important. ‘*Top to bottom*’ theory states that to detect bedsores early, early evidence of skin ischemia viz. erythema, blackening or smooth shininess is important and such should be looked for however the ‘*bottom to top*’ theory says that early changes occur in deep layers (subcutaneous tissue) so palpation to find unusual softening or induration over bony prominences

is emphasized upon; skin signs imply that damage is done already [2, 3].

To ‘stage’ and ‘score’ bedsores

Staging system provides for a uniform mechanism to describe bedsores. Scale or score-systems serve to foretell the probability of a patient developing bedsores. A standard staging system is given below in table 1 that was originally devised by Shea in 1975, modified by NPUAP⁴ in 1998 [2]. There are a number of scoring systems (Table 2) to predict risk of bed sores or prognosis, each classifies bedsores for its purpose differently and adds to the existing confusion! Norton scale is called “*father of all pressure sore scales*” as it was the first to be described in 1979 by Doreen Norton [8]. At least 40 scales are described in literature, most have never been validated and each subsequent scale is more complex than the previous one. Braden scale is the most tested and widely accepted of them hence it is recommended for clinical application by most authors. Its sensitivity is 53–100% and specificity 64–90% as reported. Both Waterlow (sensitivity 63% and specificity 83%) and Norton (sensitivity 63–100% and specificity 26–89%) scales over predict the risk, the former more so [2, 8–12].

Table 1 Staging of bedsores (modified from NPUAP¹, 1998) [2]

Stage I	Observable pressure-related changes of intact skin compared to the adjacent / opposite areas, including differences in <ul style="list-style-type: none"> • Color (redness in light skinned, red / blue / purple discoloration in dark skinned) • Temperature (increased warmth / coolness) • Consistency (firm / boggy feel) • Sensation (pain / itching)
Stage II	Partial thickness skin loss <ul style="list-style-type: none"> • involves epidermis ± dermis • ulcer is superficial & • looks like an abrasion, blister or shallow crater
Stage III	Full thickness skin loss <ul style="list-style-type: none"> • Damage & necrosis of subcutaneous tissue seen • May extend up to but not cross deep fascia • Looks like a deep crater without undermining
Stage IV	Full thickness skin loss <ul style="list-style-type: none"> • Extensive tissue destruction ± necrosis • Damage to muscle / bone / tendons / joint capsule

National pressure ulcer advisory panel, USA

⁴National Pressure Ulcer Advisory Panel

The treating physician and care-givers should be aware of the relative risk in various patients having calculated the probability by any of the above-mentioned scale (Table 2) and be able to objectively identify ‘at risk’ patients *before bedsores occur*. It was recognized in most audits that bedsores-warning signs are noticed late therefore *NICE*⁵ *guidelines* recommends that scores should be calculated within 6 hours of admission and periodically thereafter (preferably 8 hourly or at least daily); patients having high or rising scores (moderate to severe probability points according to any of the scale used) should receive preventive treatment without delay [13].

Preventing bedsores

The goal of preventive therapy is to prevent bedsores from occurring and to promote their healing if superficial bedsores do occur. Table 3 gives the *AHCPR*⁶ *guidelines* regarding preventive management for bedsores [14]. Prevention is truly better than cure for bedsores because healing may require a prolonged period of dressings, debridement and then surgery to provide skin cover once healthy granulation is achieved, a very long journey for most patients. Most patients are already very sick and they never complete this long road. Even after healing the recurrence rate is as high as 90% in some series. The most common causes of death in *chronic bedsores*⁷ are renal failure and amyloidosis; patients with *acute bedsores* usually die of co-existent diseases or sepsis [1–3, 14].

Patients at risk for bedsores and those who already have it should never be nursed in ordinary hospital mattress beds [2, 13–15]. The several types of special mattresses, mattress-overlays and beds available for this purpose are together called *pressure relieving devices* (PRDs), or *pressure support devices* (PSD). PRDs are definitely better than simple hospital beds but there is poor evidence to choose anyone as the best among them. The classification is described below. In a low air loss bed there are specially designed channels (called ‘air-sacs’) filled with air, which inflate and deflate in sequence; this distributes weight over a large contact area thus minimizing pressure-points. Air fluidized beds has the patient lying in a bath filled with fine silica particles or ceramic spherules through which filtered air bubbles are injected; the bubbling bed imitates the mechanics of a ‘fluid’. The patient “floats” on this “fluid” like bed. It does not allow pressure-points to develop and bubbles provide oxygen to prevent skin ischemia but it is very expensive⁸. PRDs makes coughing reflex ineffective due to lack of support to patient’s back hence class-II and

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⁷ The division is arbitrary, it is not defined by authors

⁸ The machine costs about Rs 1.6 crore, every year the silica particles have to be changed costing about Rs 4 lakhs!

Table 2 Commonly used scoring-scales for predicting risk of bedsores [8-12]

Norton Scale (1979)	Braden scale (1987)	Knoll scale (1985)	Waterlow scale (1985)	Gosnell scale (1989)
<i>Mental condition:</i>	<i>Sensory perception:</i>	<i>Mental status:</i>	<i>Continence:</i>	<i>Mental status:</i>
Alert (1)	Completely limited (1)	Alert (0)	Complete (urine / stool) (0)	Alert (1)
Apathetic (2)	Very limited (2)	Lethargic (1)	Catheterized & fecal continence (0)	Apathetic (2)
Confused (3)	Slightly limited (3)	Semicomatose/confused (2)	Occasionally incontinent (1)	Confused (3)
Stupor (4)	No impairment (4)	Comatose (3)	Catheterized & fecal incontinence (2)	Stuporous (4)
<i>Incontinence:</i>	<i>Moisture:</i>	<i>Incontinence:</i>	Fecal & urinary incontinence (3)	Unconscious (5)
Fully continent (1)	Constantly moist (1)	None (0)	<i>Mobility:</i>	<i>Continence:</i>
Occasional incontinence (2)	Moist (2)	Occasional (1)	Full mobility (0)	Fully controlled (1)
Usually incontinent- for urine (3)	Rarely moist (4)	Usual (4)	Restless & fidgety (1)	Usually controlled (2)
Usually incontinent- for urine & stool (4)	<i>Mobility:</i>	Total (6)	Apathetic (2)	Minimal control (3)
<i>Mobility:</i>	Completely immobile (1)	<i>Mobility:</i>	Restricted (3)	No control (4)
Full (1)	Very limited (2)	Full (0)	Inert / in traction (4)	<i>Mobility:</i>
Slight restriction (2)	Slightly limited (3)	Limited (1)	Wheelchair bound (5)	Full (1)
Limited mobility (3)	No limitations (4)	Very limited (4)	<i>Sex:</i>	Slightly limited (2)
Very limited mobility (4)	<i>Activity:</i>	Immobile (6)	Male (1)	Very limited (3)
<i>Activity:</i>	Bed-ridden (1)	<i>Activity:</i>	Female (2)	Immobile (4)
Ambulant (1)	Wheelchair bound (2)	Ambulatory (0)	<i>Age:</i>	<i>Activity:</i>
Able to walk with help (2)	Occasionally walks (3)	Needs help (1)	14-49 years (1)	Ambulatory (1)
Wheelchair-bound (3)	Frequently walks (4)	Wheelchair bound (4)	50-64 years (2)	Walks with help (2)
Bedridden (4)	<i>Nutrition:</i>	Bedridden (6)	65-74 years (3)	Chair-ridden (3)
Immobile (5)	Very poor (1)	<i>General health status:</i>	75-80 years (4)	Bed-ridden (4)
<i>Physical condition:</i>	Probably inadequate (2)	Good (0)	≥ 81 years (5)	<i>Nutrition:</i>
Good (1)	Adequate (3)	Fair (1)	<i>Build / weight for height:</i>	Regular (1)
Fair (2)	Excellent (4)	Poor (2)	Average (0)	Occasionally misses food (2)
Poor (3)	<i>Friction & Shear:</i>	Moribund (3)	Above average (1)	Rarely takes food (3)
Very bad (4)	Problem (1)	<i>Nutritional intake:</i>	Obese (2)	
	Potential problem (2)	Fair (1)	Below average (3)	
	No apparent problem (3)	Poor (2)	<i>Appetite:</i>	
		None (3)	Average (0)	
		<i>Fluid intake:</i>	Poor (1)	
		Good (0)	Nasogastric tube (2)	
		Fair (1)	Fluids only (3)	
		Poor (2)	Nil by mouth (4)	
		None (3)	<i>Skin appearance:</i>	
		<i>Predisposing disease:</i>	Healthy (0)	
		Absent (0)	Papery (1)	
		Slight (1)	Dry (1)	
		Moderate (2)	Edematous (1)	
		Severe (3)	Clammy (1)	
			Discolored (2)	
			Broken (3)	
			<i>Special risk factors:</i>	
			Terminal cachexia (8)	
			Cardiac failure (5)	
			Peripheral vascular disease (5)	
			Anemia (2)	
			Smoking (1)	
			Neurological deficits	
			• Moderate (4)	
			• Moderate to severe (5)	
			• Severe (6)	
			Major surgery / trauma	
			• Orthopedic / below waist / spine (5)	
			• On OT table > 2 hours (5)	
			On steroids / anticancer / high dose anti-inflammatory drugs (4)	
Score > 14 indicates high risk of developing bed sore	15-16 = low risk 13-14 = moderate risk < 12 = high risk	≥ 11 = high risk ≥ 16 = should be put on pressure relieving surface	< 10 = low risk 10-14 = at risk 15-19 = high risk	5 = very low risk 20 = very high risk

Table 3 Guidelines for prevention of bedsores (modified from revised AHCPR¹ guidelines, 1994) [13]

Risk assessment

- Identify patients predisposed to developing bedsores by using any of the scales

Measures to relieve pressure

- Turn the patient and reposition periodically
- Use positioning device
- Use support surfaces
 1. Static devices: mattress and bed-overlays
 2. Dynamic devices: low air-loss and air-fluidized beds

Nutritional assessment and supplements

Skin care

- Clean with isotonic saline
- Avoid rubbing / massaging ulcers
- Avoid excessive dryness (i.e. hot water and soap)
- Avoid excessive moisture

Management of urinary and fecal incontinence

¹Agency for health care policy and research, US Dept. of health and human services

III devices are contraindicated in patients having COPD [2, 3, 13, 14].

- Class-I: Simple devices: *bed-overlays and mattresses*
- Class-II: Advanced devices: *low air loss bed* (seek to maintain interface pressure < 32 mm Hg)
- Class-III: Advanced devices: *air fluidized bed* (reduce interface pressure < 10–12 mm Hg)

The *recommended* safe ‘interface pressure’ is 28–38 mm Hg [7]. One study found the interface pressure between skin and support surfaces was as high as 56–60 mm Hg between skin and emergency trolley and 75–80 mm Hg between sacral skin and operation-table. The average sacral pressure when lying on a standard hospital mattress is 100–150 mm Hg, increasing if patient’s head end is raised. The average pressure over ischial tuberosity when a patient sits on a chair is 100 mm Hg [3–7].

Bedsores reflect quality of nursing care in hospital settings, particularly ICUs. A strictly followed protocol of sponging, bed-making and frequently turning the patients on their sides should lower the incidence but such results may be frustratingly marginal and slow. The standard of care is to change position of patients *every 2 hours*. *30° lateral position* avoids pressure on the skin over both sacrum as well as trochanteric prominence; it is therefore strongly recommended [7, 13, 14].

All patients with or without bedsores (but at risk) should be initially nursed in Class-I PRDs (with some exceptions, see below). If bedsores appear or do not heal in 30 days they should be shifted to Class-II device. If bedsores still do not heal after 30 days they are offered Class-III devices. Patients who have had an operation for bedsores within 60 days should be nursed on Class-II device post-operatively. Patients with large stage-III / IV bedsores or multiple bedsores should be nursed in Class-III device [14, 15].

Table 4 lists the alternatives available for treating and preventing bedsores; there are numerous dressing materials and techniques to choose from. Dressing materials may be classified as shown below.

- *Cleaning agents*⁹ which are used to wash away pus and discharge,
- *Debriding agents*¹⁰ which remove slough and dead tissue and
- *Barriers*¹¹ which prevent contamination and give protection by covering wound

A Cochrane review notes that although the annual sales of the “US bedsores industry” exceeds \$ 7 billion, none of the cleaning agents proved better than tap-water in one study! The utility of ‘debriding agents’ is “questionable” with “little evidence of any improvement in wound-healing”. The general thumb-rule to choosing dressing materials is to choose *dry (so as to absorb discharge) dressings for a wet wound* and *wet (so as to provide moisture) dressings for dry wounds*. *Moist / occlusive dressings are better than dry / open dressings*. Surgical debridement may prove necessary for stage II–IV bedsores. The objective is to achieve optimal nutrition, continence, mobility and local wound condition upon which he may be referred to a plastic surgeon for skin-cover [1, 3, 13–16].

Surgery for bedsores

It is often possible to primarily close defects but poor healing of wound make this hazardous; flap cover is preferable.

⁹Normal saline, povidone iodine, various scrubs etc

¹⁰Hypochlorite solution, hypertonic saline, enzymes like collagenase etc

¹¹Saline soaked gauze, thin transparent plastic dressing, foam dressing, alginate or hydrocolloid dressing

Table 4 General management plan & available tools for bedsores [1-3, 7, 15, 16]

	Tools of the trade	Strength of evidence ¹ & comments
No bed sore	Improving nutrition	I/II
	Turning & repositioning the patient every 2 hours	II/III Frequency should be individualized 30° lateral position is best for nursing
	Pressure relieving devices (PRD)	I/II
	<ul style="list-style-type: none"> Specialized Foam mattress Air-filled mattress Low air-loss bed (Flexicair™, Kinair™) Air-fluidized bed (Clinitron™, Fluidair™) 	No device is superior Expensive The goal is to maintain interface pressures < 30-32 mm Hg
	Relieve spasticity	II
	<ul style="list-style-type: none"> Diazepam Baclofen Dantrolene sodium Mephenesine carbonate Dimethothiazine Orciprenaline 	
	Keeping the skin clean & dry	I Urinary catheter & constipating agents ease nursing
	Grade I: (<i>Keep clean & dry, no dressing needed</i>)	I
	Transparent films (Opsite™, Tegaderm™, Bioclusive™) can be used to cover if little or no exudates is present	Irritant chemicals should never be used
	Grade II: (<i>Wash with normal saline and apply dressing</i>)	I/II
Bed sore present	Hydrocolloid occlusive dressing (Duoderm™, Restore™, Tegaserb™, Replicare™)	
	Grade III/IV: (<i>Clean with normal saline & apply dressing ± debridement</i>)	I/II
	Debridement	No dressing material superior to others Hypochlorite solutions have little or no value Proprietary dressing materials are more expensive VAC helps if copious discharge seen Toxic shock syndrome is reported with VAC
	<ul style="list-style-type: none"> Mechanical debridement (dressings) <ol style="list-style-type: none"> Wet to dry dressings <ul style="list-style-type: none"> Normal saline based Sodium hypochlorite based (Dakin's solution, EUSOL) Surgical (sharp) debridement Autolytic debridement (synthetic occlusive dressings which enhance action of endogenous enzymes; DuoDERM™, Contreet™) Enzymatic debridement (dressings contain exogenous enzymes; collagenase, papain, fibrinolysin, streptokinase or streptodornase) Whirlpool bath 	
	Dressing: Silver Sulphadiazine (Silvadene™), Sulfamylon™, Hydrogels (Carrington gels, Clearsite™, Vigilon™, Flexigel™), Xerogels (Sorbsan™)	
	Vacuum assisted closure (VAC) devices (Kinetic™)	

¹ I: very strong evidence to support its use, II: some evidence, III: little or no evidence

Split skin grafting is possible for superficial ulcer. Most ulcers are deep and require more extensive soft-tissue reconstruction therefore myocutaneous flaps are advised. A patient in good general health with stage III / IV bed-sores, healthy granulation tissue in ulcer-bed and minimal pus or slough is a good candidate for flap reconstruction. Table 5 gives a menu of flaps available for reconstruction to choose from. All flaps are contraindicated if the general condition

is poor, there is poor social support or if the patient is expected to be non-compliant with therapy hereafter [17, 18].

Recent advances

The challenge in research is to detect bed sore before clinical changes appear. Interface pressure can be monitored via

Table 5 Choice of flaps available to provide skin-cover for bedsores¹ [17, 18]

Location of bedsores	Flaps available to reconstruct the defect
Ischial tuberosity	Gluteal thigh rotation flap Hamstring flap Biceps femoris flap Tensor fascia lata (TFL) flap Gracilis flap Medially based posterior thigh skin flap
Sacrum	Inferiorly based skin rotation flap Superior gluteus maximus flap Bilateral V-Y advancement flap Transverse lumbosacral flap
Trochanteric prominence	TFL flap & its modifications (Retro-position V-Y flap, Bi-pedicled TFL flap) Vastus lateralis flap Gluteal thigh flap Anterior thigh flap
Multiple bedsores	Total thigh rotation flap

Flap refers to myocutaneous flap unless otherwise mentioned. The first in each category is the flap of choice, others are alternatives to it.

a probe but this is difficult; older probes were bulky and newer smaller probes are prone to malfunction. Another approach is to try and detect tissue damage before it manifests. This is termed '*tissue-status monitoring*' and can be done in two ways.

- Ischemia can be confirmed by measuring skin blood-flow in vulnerable areas by laser Doppler flowmetry, reflective spectro-photometry or trans-cutaneous gas tensions measured at elevated skin temperatures (ischemia will cause increase in carbon di-oxide with a corresponding fall in oxygen i.e. T_cPO_2 falls while T_cPCO_2 rises).
- Sweat from patient's skin at pressure-points can be collected and analyzed for the presence of chemical indicators of ischemia¹² but collection of sweat remains a practical problem.

These esoteric methods are yet to be widely applied clinically.

The list of dressing materials available keeps increasing. Interest has been generated in topical application of various growth factors which appear to stimulate granulation-tissue formation. US-FDA¹³ has recently cleared 0.01% RegranexTM (Becaplermin) gel, which is recombinant hPDGF¹⁴ amidst promising early reports; it is contraindicated if skin cancer is present at the site of application. Other growth factors tested for topical application with promising reports include basic fibroblast growth factor (β -FGF) and platelet rich human plasma [3, 13, 14, 19].

Wound break-down is common even after flap cover. It was earlier thought to be due to lack of sensation in the flap tissue. Sensate flaps like TFL flap (with $L_{4,5}$ lateral femoral cutaneous nerve) or innervated intercostal flap (with intercostal nerve) were devised. The initial results have been disappointing. A recent study also found that nutritional supplementation failed to decrease incidence of bedsores. These findings are expected because primary factors that lead to bedsores formation such as immobility may still be affecting the patient. It has now been realized that unless the disease that lead to bedsores is cured, bedsores are likely to recur [17, 19].

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¹²Like free radicals etc.

¹³Food and drugs authority of USA

¹⁴Human platelet derived growth factor

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