Regional anaesthesia with sedation protocol to safely debride sacral pressure ulcers

Daniel K O'Neill, Bryan Robins, Elizabeth A Ayello, Germaine Cuff, Patrick Linton, Harold Brem

O'Neill DK, Robins B, Ayello EA, Cuff G, Linton P, Brem H. Regional anaesthesia with sedation protocol to safely debride sacral pressure ulcers. Int Wound J 2012; 9:525–543

ABSTRACT

A treatment challenge for patients with sacral pressure ulcers is balancing the need for adequate surgical debridement with appropriate anaesthesia management. We are functioning under the hypothesis that regional anaesthesia has advantages over general anaesthesia. We describe our regional anaesthesia protocol for perioperative and postoperative management.

Key words: Anaesthesia • Pressure ulcer • Regional • Sacral • Wound

INTRODUCTION

A pressure ulcer can be defined as a 'localised injury to the skin or underlying tissue, usually over a bony prominence that results from pressure, including pressure associated with shear' (1). These ulcers can be extensive with damage to the skin and underlying soft tissue, muscle and even exposing bone (2). These wounds may go unnoticed for extended periods of time because they are more prevalent in bed bound, paralysed patients as well as patients older than 65 years (2). The annual occurrence of these pressure ulcers is approximately 1·3–3 million (3), including over 500 000 hospitalisations where pressure ulcers were diagnosed (4). Nearly three out of four extended stays were in people over 65 years of age (4), and this age group also accounted for 56·5% of principal diagnoses as well. More than 90% of the pressure ulcer diagnoses were a secondary diagnosis relating from another principal problem (4), costing hospitals $10·2 billion in addition to the $752 million caused by primary pressure ulcers (4). Pressure ulcers cause longer hospital stays (4) and increase the risk of infection on top of the treatment costs outlined above (5). There is substantial evidence that supports the fact that pressure ulcers are also associated with increased mortality and morbidity rates (6) and are the cause of death in more than 104 000 persons per year in the USA alone (7). Therefore, it is vital that these ulcers are properly diagnosed and treated.

Key Points

- more than 90% of the pressure ulcer diagnoses were a secondary diagnosis relating from another principal problem
- it is vital that these ulcers are properly diagnosed and treated

Authors: DK O'Neill, MD, Department of Anesthesiology, New York University School of Medicine, New York, NY 10016, USA; B Robins, BS, NYU-NIH Summer Program, New York University Langone Medical Center, New York, NY 10016, USA; EA Ayello, PhD, RN, ACNS-BC, CWON, MAPWCA, FAAN, Faculty, Excelsior College School of Nursing, The John A. Hartford Institute of Geriatric Nursing, New York University College of Nursing, New York, NY, USA; G Cuff, BSN, MPH, CCRP, Department of Anesthesiology, New York University Langone Medical Center, New York, NY 10016, USA; P Linton, MD, Department of Anesthesiology, New York University School of Medicine, New York, NY 10016, USA; H Brem, MD, Division of Wound Healing and Regenerative Medicine, Department of Surgery, Winthrop University Hospital, Mineola, NY, USA

Address for correspondence: DK O'Neill, MD, Department of Anesthesiology, New York University School of Medicine, New York, NY 10016, USA

E-mail: Daniel.ONeill@nyumc.org

international wound journal

Key Point

- the vital aspect in the pain management and treatment of chronic wounds is first making the decision to perform the surgical debridement then finding the most effective mixture of topical, subcutaneous and systemic anaesthesia

Challenges in healing sacral pressure ulcers

Pressure ulcers in adults usually occur over bony prominences such as the sacrum or the heel (8,9). The sacrum located in the lower dorsum is the most common site for pressure ulcers, where approximately 32% of pressure ulcers occur according to one study (10). Of the newest category of pressure ulcers as defined by the National Pressure Ulcer Advisory Panel (NPUAP) (1), suspected deep tissue injury is surprisingly more commonly found on the heels (41%) rather than on the sacrum (19%) (11). Once these ulcers are formed, they may become chronic wounds with physiological impairments that prevent the stimulation of wound healing even further (12-14). The blood supply to the sacral region defines an angiosome. Compromised blood supply from pressure, hypothermia and vasopressors can both contribute to the development of sacral pressure ulcers, as well as impair the angiogenic response during wound healing. Sharp surgical debridement promotes wound bed preparation for closure. The angiogenic response is dependent upon where the region is anatomically and that the response is significantly skewed away from the lower dorsum (15,16). This decreased angiogenic response contributes to the slower healing in the lumbosacral area (17), which increases the likelihood of the need for surgical intervention in a sacral pressure ulcer. A sacral pressure ulcer is in close proximity to the rectum, which increases the likelihood of fecal contamination from incontinence and can hinder healing in addition to making the wound larger (18,19). According to a protocol for the comprehensive treatment of pressure ulcers (2), these aspects make sacral pressure ulcers a prime candidate for operative debridement to allow tissue regeneration to start (20,21). Sharp debridement using a scalpel is the preferred method used to eliminate the necrotic tissue, which hinders the healing process and possibly masks other factors such as infection that impede wound closure as well (2). The National Pressure Ulcer Advisory Panel (NPUAP)/ European Pressure Ulcer Advisory Panel (EPUAP) Clinical Guidelines recommends that the vascular status and stability of eschar heel ulcers be determined prior to sharp debridement (1). The vital aspect in the pain management and treatment of chronic wounds is first making the decision to perform the surgical debridement then finding the most effective mixture of topical, subcutaneous and systemic anaesthesia (22).

The role of surgery in treating sacral pressure ulcers

Surgery has inherent risks. Many patients, family members and attending physicians believe that taking patients to the operating room for surgery and anaesthesia is excessively risky compared with medical therapy. The wound patient has more comorbidities and is at greater risk than a normal average patient as evident by the fact that wound patients have an average score of 3·09 on the American Society of Anaesthesiologists (ASA) physical status classification scale (Table 1) compared with the overall average of 2·03 value for anaesthesia patients at a major university hospital (23). The ASA score does increase with severity of disease and the impact on function. The ordinal scale is based on an assessment of the entire present health condition of the patient during the preoperative evaluation. The number of comorbidities does combine to increase the score, but it is not linear. For example, a single disease with end-organ damage (like end-stage renal disease requiring dialysis), which impairs function places a patient at higher perioperative risk than six relatively benign problems such as a lipoma or pin worms.

The incidence of complications and mortality for patients who undergo anaesthesia is greater among patients with a higher ASA score (24). However, sharp surgical

Table 1 American Society of Anaesthesiologists physical status classification

Class	Patient Status
I	Healthy patient with no comorbidities
II	A patient with mild systemic disease that should not affect anaesthesia and surgery
III	A patient with a severe systemic disease that requires special care during surgery
IV	A patient with a severe systemic disease or end-organ damage that is life threatening and which must be addressed preoperatively, intraoperatively and postoperatively by the anaesthesia team
V	A moribund patient whose life expectancy does not exceed 24 hours irrespective of surgery

Adapted from American Society of Anaesthesiologists. ASA physical status classification system. Available at: http://www.asahq.org/clinical/physicalstatus.htm

debridement is necessary to remove sources of infection and sepsis from the wound that will increase mortality rates or prevent wound closure (10). This procedure has been described in detail and has been shown to be safe and have low-mortality rates even in patients with multiplecomorbidities (10).

The formation of new pressure ulcers is also something to consider when evaluating the risks of surgery. Between 4·7% (25) and 45% (26) of surgical patients are at risk of developing pressure ulcers, where the only reliable indicator was the length of surgery. This is because of the fact that patients are immobile and cannot feel pain caused by an extended period of pressure and shearing forces (27). In addition, an awake patient can position themselves comfortably on the operating room table, but anaesthetised patients lack the motor control to adjust their position to relieve these forces and prevent pressure injuries (27). Therefore, a lengthy procedure makes the operating table an ideal place to develop new ulcers especially in the presence of elderly frail skin, hypothermia and haemodynamic instability requiring vasopressors (27). Special techniques for patient positioning are used including padding to decrease the risk of skin breakdown. Because debridement procedures are usually brief, anaesthesia protocols that minimise duration and depth of sedation are preferred. The operating room provides a safe environment for patient care. The surgeons have the benefit of highly skilled nursing and technical staff, modern equipment, adequate lighting, sterile surgical field and professional anaesthetic care. Although many may claim that sacral ulcers can be debrided 'at the bedside' to minimise the use of operating room resources, patients may develop sepsis from inadequately treated infections including osteomyelitis and myositis. This surgical infection diagnostic challenge is analogous to differentiating cellulitis which can be treated medically and necrotising fasciitis which needs to be treated surgically. The 'conservative' strategy of 'benign neglect' by avoiding surgical intervention may be 'cost effective' for policy makers who tolerate attrition like hospice rather than promoting wound healing.

Financial concerns

The financial burden for both the patient and the hospital to bear for a surgical procedure is significant. Perioperative costs account for 5-6% of total hospitalisation costs (28,29). These costs result from expensive intraoperative procedures, medications, staffing and physiological monitors, which places them at the forefront of intense health care industry scrutiny (30). One method to cut costs to both anaesthesia departments and hospitals would be to decrease Postanaesthesia Care Unit (PACU) and postoperative admissions by investigating the types of anaesthetic techniques (31).

The post-anesthesia care unit (PACU) or recovery room is essential for stabilisation and management of most surgical patients. Decreased PACU stay and postoperative admissions have been shown to be benefits of increased use of regional techniques versus general anaesthesia techniques (31). When spinal anaesthesia was used as an alternative to general anaesthesia in knee arthroscopies (an outpatient procedure), there was no difference in the readiness for discharge (32). The spinal anaesthesia group had lower pain scores and no nausea and vomiting compared with a 19% incidence of these symptoms in the patients who received general anaesthesia (32). Another study on anterior cruciate ligament repairs found an increase in PACU bypassing after the use of peripheral nerve blocks for postoperative pain management as opposed to general anaesthesia (33). Therefore PACU bypassing and shorter PACU duration can be accomplished by reducing postoperative pain incidence, nausea, vomiting and other postoperative complications, especially with regional anaesthesia (32). We describe general anaesthesia in some detail because many surgeons erroneously believe that all sedated patients are under general anaesthesia. In addition, general anaesthesia is required when regional blocks fail to provide adequate antinociception or are unable to provide the operating conditions needed for surgical success. When a local infiltration block is administered for sacral debridement, the recovery would primarily be dependent upon the duration of action of the intravenous (IV) anaesthetics. When spinal anaesthesia is used, for example, in a spinal cord patient to decrease the risk of autonomic hyperreflexia, the sensory and motor function of the spinal cord should return to the baseline state. Knowledge of complete or incomplete spinal cord function would be necessary to attain preoperatively. The duration of action of the intrathecal sodium channel blocker may

Key Points

- sharp surgical debridement is necessary to remove sources of infection and sepsis from the wound that will increase mortality rates or prevent wound closure
- the operating room provides a safe environment for patient care
- we describe general anaesthesia in some detail because many surgeons erroneously believe that all sedated patients are under general anaesthesia

Key Points

- on the basis of the literature surrounding the benefits of regional anaesthesia, it is the conclusion of this wound healing and regenerative medicine team, that a protocol of a regional block with sedation as needed is the most effective anaesthesia regimen for sharp surgical debridement
- this literature review also examined financial data that looked at the costs of regional compared with general anaesthesia techniques by looking at several cost drivers in the preoperative, intra operative and postoperative costs

vary from 40 to 50 minutes for plain procaine from 180 to 240 minutes for dibucaine with epinephrine. During the duration of residual neuroaxial blockade, special additional care needs to be maintained to protect injury especially to the skin and peripheral nerves of the lower extremities. The Bromage scale is used commonly to grade block recovery.

Anaesthesia for pressure ulcer patients

The sparse literature about anaesthesia protocols for wound care, especially for pressure ulcers, provides limited guidance as to the best method given the wide variation in techniques available (34). Sacral pressure ulcers can be debrided in the lateral or prone position with local, regional or general anaesthesia with various degrees of sedation and airway management (34). Sacral pressure ulcers demand significantly different anaesthesia regimens than other pressure ulcers, because they could be outside the scope of certain anaesthesia treatments that could be applied to other pressure ulcers (34). The anaesthesia technique is usually determined and applied based on the preference of the anaesthesiologist after completing the preoperative anaesthesia evaluation. This practice often leads to a common misconception that anaesthesia refers to only general anaesthesia even when regional blocks can be and are used (35). On the basis of the literature surrounding the benefits of regional anaesthesia, it is the conclusion of this wound healing and regenerative medicine team, that a protocol of a regional block with sedation as needed is the most effective anaesthesia regimen for sharp surgical debridement (35). This is because it limits complications, reduces postoperative pain and decreases the economic burden placed on both the health care stakeholders and the patient. This protocol acknowledges the limitations of regional anaesthesia and realises that pre-existing conditions or failed blocks require the anaesthesiologist to re-evaluate the anaesthetic technique (35).

METHODS

Literature review

As a result of a comprehensive study of the literature pertaining to both regional and general anaesthesia, we compiled this protocol, which functions to give guidelines on proper application and management of the care from the preoperative evaluation through the postoperative care. This literature review also examined financial data that looked at the costs of regional compared with general anaesthesia techniques by looking at several cost drivers in the preoperative, intraoperative and postoperative costs.

Protocol for the administration of an anaesthesia regimen to a sharp debridement candidate

It must be emphasised that our protocol for preventing the appearance of new pressure ulcers and promoting healing of existing sacral pressure ulcers using sharp, surgical debridement and anaesthesia (Table 2) is designed to maintain homeostasis during times that would otherwise be traumatic for both the patient as well as the patient's family. The protocol is initiated when the surgeon decides to schedule a surgical procedure in the operating room.

Acknowledge that every patient with pressure ulcers has comorbidities, which increase the risk associated with anaesthesia

Pressure ulcers are caused by unrelieved pressure, which obstructs blood flow creating a deficiency of oxygenation and nutrients creating tissue destruction (2,3). Therefore, certain comorbidities are associated with the formation of these pressure ulcers. These comorbidities must be accounted for, and they comprise risk factors for anaesthesia complications. A frequent risk factor associated with the formation of a new pressure ulcer is limited mobility, which results from amputations, paralysis, neurological disorders (multiple sclerosis, Alzheimer's, dementia, etc.), coma or sedation, and so forth (36). These might not be direct risk factors for anaesthesia, but they do influence certain factors of the overall treatment and pain management with analgesia.

Pressure ulcers are also associated with malnutrition, which could be caused by eating disorders, dehydration or dietary restrictions (36). Indices of nutritional status include albumin, prealbumin and cholesterol. Malnutrition is important for the anaesthesiologist to take into account including drug dosing given the changes in volume of distribution, protein binding to α–1 glucoprotein and pharmacodynamic effects to decrease the risk of problems. One study found electrolyte imbalances and significantly slower heart rates

Table 2 Protocol for anaesthesia administration for surgical debridement of sacral pressure ulcer © O'Neill & Ayello 2012

1	Acknowledge that every patient with pressure ulcers has comorbidities which increase the risk associated with anaesthesia.
2	The preoperative assessment begins with the medical history, physical examination, laboratory analysis and consultation of medical specialists as indicated.
3	The expectation of the consultant is to characterise the disease, medically optimise and risk stratify the patient prior to surgery and anaesthesia.
4	All laboratory results and diagnostic studies are evaluated for abnormalities and are corrected as indicated.
5	All patients with cardiac rhythmic management devices (CRMD) are interrogated to ensure proper functioning.
6	To decrease the risk of aspiration pneumonia from gastric regurgitation, Nothing Per Os (or mouth) orders are enforced.
7	Intravenous (IV) fluids and medications are administered preoperatively for inpatients, diabetics and those who need medications.
8	All patients are monitored intraoperatively according to American Society of Anaesthesiology (ASA) standards.
9	Regional anaesthesia (local, peripheral and central) with sedation (none, light or deep) is administered as the default regimen with general IV or inhalational agents used when blocks are not carried out.
10	Goals of airway management are to maintain airway patency, oxygenation and ventilation using the minimum invasiveness of airway devices.
11	All patients are monitored postoperatively for duration appropriate to their acuity.

during preoperative assessments compared with healthy patients (37).

Pressure ulcers are also linked with many other conditions that can severely complicate an anaesthesia regimen. These include diabetes mellitus, vasculitis and other vascular collagen disorders, immunodeficiency, corticosteroid therapy, congestive heart failure, malignancies, end-stage renal disease and chronic obstructive pulmonary disease (36). All of these conditions have implications for surgery and wound healing.

The preoperative assessment begins with the medical history, physical examination, laboratory analysis and consultation of medical specialists as indicated

The preoperative assessment has become crucial, as the role of the anaesthesiologist has expanded outside of the operating room and an increased number of ambulatory procedures, such as surgical debridement, are performed (38). It is also vital in securing the safety of the patient during the admission of an anaesthesia regimen. The Australian Incident Monitoring Society concluded that 3·1% of complications were directly attributed to deficient, and 11% to inadequate preoperative assessments (38). Davis determined that 53 of 135 patient deaths (39%) linked to anaesthesia resulted from inadequate preoperative assessments and suboptimal monitoring of existing medical conditions (39).

The preoperative assessment needs to be thorough and complete to ensure the maximum protection for both the patient and the institution. The evaluation begins with the medical history then proceeds to physical examination, laboratory analysis and finally to consultation of medical specialists as indicated by the attending anaesthesiologist. The physical examination takes vital signs and confirms the presence of any conditions that could compromise the patient intraoperatively. Laboratory tests are ordered as necessary to discover any underlying conditions that could complicate the procedure and explain any abnormalities observed during the physical examination. History of malignant hyperthermia (MH) by questionnaire or interview requires avoidance of triggering agents such as succinylcholine and potent inhalational anaesthetics such as isoflurane, sevoflurane or desflurane. Family history of MH should prompt a call to the MH hotline and diagnosis by muscle biopsy. A questionnaire or muscle biopsy about anaesthesia history in the patient and family is also useful for diagnosing the risk of MH. Furthermore, a consultation is usually necessary and beneficial to the operative care of a patient and is usually ordered to clarify any existing medical issues.

The expectation of the consultant is to characterise the disease, medically optimise and risk stratify the patient prior to surgery and anaesthesia

Preoperative consultations should be initiated for the diagnosis, evaluation and treatment of a newly or poorly managed condition and

Key Point

- the preoperative assessment has become crucial, as the role of the anaesthesiologist has expanded outside of the operating room and an increased number of ambulatory procedures, such as surgical debridement, are performed

for the creation of a risk evaluation that the patient, anaesthesiologist and surgeon can use to make patient care decisions (38). The person requesting the consultation should ask specific questions about the patient health status and the tasks that need to be performed prior to the procedure. The result of the consultation should be a descriptive letter or progress note summarising the patient's medical conditions, along with the results of the diagnostic tests (38). Omitted data about the patient's condition could delay surgery and increase costs. Occasionally, an anaesthesiologist will receive a handwritten note from a medical consultant which reads 'Patient is cleared for surgery'. This substitute for a proper evaluation and assessment is not only useless, but is insulting to the anaesthesiologist who is responsible for clearing the patient for the operating room. For a pressure ulcer, one consultant must be the wound care specialist or operating surgeon. Other specialists could include internist, cardiologist, pulmonologist, nephrologist, haematologist, neurologist and/or rheumatologist. The diagnostic results that accompany the letter are vital for the anaesthesiologist's ability to make an independent, unbiased decision about patient risk and to plan the anaesthesia regimen accordingly.

All laboratory results and diagnostic studies are evaluated for abnormalities and are corrected as indicated

Laboratory examinations should only be prescribed to patients who present an increased risk of complications based on medical history or the physical examination. This will help to keep costs down and preserve valuable materials (28–31,40). Each institution should develop standards which are in alignment with national guidelines and individual patient needs. When preoperative test results are abnormal, correcting the abnormality reduces the risk of developing perioperative complications. Testing is most efficient when the most sensitive and specific tests are used to confirm an existing abnormality that was uncovered during the initial medical history and physical examination (40). The following tests should be ordered for the preanaesthesia evaluation when appropriate: electrocardiogram (ECG), echocardiography, stress tests, angiography, chest X-rays, pulmonary function tests, complete blood count, type and screen (T&S), coagulation studies [prothrombin time (PT), international normalized ratio (INR), activated partial thromboplastin time (APTT)], serum chemistries, urinalysis and pregnancy tests (41). Surgical patients can bleed to a point of requiring blood products. Therefore, a T&S would be indicated to exclude the risk of antibodies and antigen–antibody incompatibility which may delay blood product volume resuscitation during haemorrhage. Electronic cross matching has recently been introduced to improve the efficiency and effectiveness of the process. During surgery, the haemostatic balance between bleeding and clotting needs to be addressed to minimise the risk of haemorrhage, stroke, deep vein thrombosis (DVT) and pulmonary embolism. The coagulation cascade and platelet function are both important to evaluate in the preoperative assessment of risk. Most surgeons are fine with INR below 2·2, but platelets may need to be ordered. All substances that increase bleeding risk that a patient is taking including anticoagulants, antiplatelets and some herbals need to be considered. For example, some patients may not report that they are taking herbals that have haemostatic impact like garlic. Clinical decision support should be available for staff because the list of agents influencing the haemostatic system continues to grow.

All patients with CRMD are interrogated to ensure proper functioning

A cardiac rhythm management device (CRMD) is an implanted pacemaker (PM) or an implanted cardioverter-defibrillator (ICD) (42). Patients with these devices are at risk of developing complications during surgery because of the malfunction of the device (42–45). For example, the electrocautery can be sensed by an ICD which would trigger inappropriate electrical therapy (defibrillation) which would not only be painful or hazardous for the patient, but could prematurely deplete the generator power supply (45).

Successful perioperative procedures with CRMD patients depend on the preoperative evaluation (45). After acknowledging that the patient has a CRMD, device testing consists of determining the type and manufacturer of the device, determining if the patient is PM dependent for antibradycardia function

and understanding the function of the device in the perioperative patient (42–47). Direct interrogation of the CRMD by a device expert like a cardiac electrophysiologist or corporate representative remains the 'gold standard' for determining the battery status, quality of the leads and appropriateness of the settings at the time of interrogation (48). It is imperative that this device interrogation data be acquired and a therapeutic plan is established in a timely manner for optimal perioperative care (45).

The preoperative evaluation should be based on the ASA guidelines. This includes (1) determining the extent of electromagnetic interference (EMI) during the debridement, (2) determining any adjustments to the CRMD programming that are needed, (3) suspending antitachyarrhythmia functions if present, (4) advising the surgeon of the use of a bipolar electrocautery system or ultrasonic scalpel to limit EMI on generators and leads, (5) assuring the operating room has temporary pacing and defibrillation machines available and (6) evaluating the possible impact of anaesthesia on CRMD function in the patient (44,46). In general, PM function should be maintained but sensing function can be temporarily disabled.

During the postoperative care the device should be reset to the preoperative settings, re-enabled if necessary and re-interrogated to ensure proper function for discharge (48). Failure to re-establish tachyarrhymia therapy after the procedure defeats the purpose of protecting the patient from lethal ventricular tachycardias (48).

To decrease the risk of aspiration pneumonia from gastric regurgitation, NPO (or by mouth orders) are enforced

Nothing Per Os (NPO) is a precaution that anaesthesiologists use to decrease the risks of morbidity or mortality. NPO is prescribed to lower stomach acidity and gastric fluid volume before the operation in order to decrease the risks of aspiration pneumonia, which is a cause of morbidity from Acute Respiratory Distress Syndrome and mortality from multiple organ failure (49). Poor oral hygiene especially in debilitated patients who can not care for their own mouths has also been associated with aspiration pneumonia (50,51). Patients with dysphagia should have swallowing studies to evaluate the need for feeding tubes (50,51). However, the risk of pulmonary aspiration needs to be balanced with the risks of excessive NPO status leading to dehydration and starvation.

Recent studies have linked prolonged preoperative fasting periods to problems such as irritability, headache, emesis, hypotension, hypovolaemia and hypoglycaemia (52–55). Therefore, NPO orders should be according to the ASA guidelines which serve as recommendations that can be adopted to fit clinical needs and constraints (56). What one eats or drinks makes a difference in regards to NPO orders and anaesthesia. Therefore, ASA guidelines call for a fasting period for clear liquids (water, fruit juices without pulp, carbonated beverage, clear tea, black coffee, etc.) of at least 2 hours and non human milk and or a light meal (toast and clear liquids) of at least 6 hours prior to anaesthesia (56). The content and make-up of a heavier meal needs to be accounted for because it increases the gastric emptying time (56). Emergency cases with full stomachs or patients with abnormal gastric emptying or lower oesophageal sphincter function require special approaches to airway management which include rapid sequence induction with intubation, awake intubation or regional anaesthesia without sedation.

IV fluids and medications are administered preoperatively for inpatients, diabetics and those who need medications

Wound patients typically have comorbidities which need to be addressed prior to the start of the debridement, many requiring hospital admission for medical optimisation. Oral medications should be converted to the IV equivalent unless an 'NPO except Meds' guideline is appropriate for the patient and is consistent with institutional policies.

These medications can include orally administered antibiotics, antihypertensives, anticoagulants, antiplatelets, oral hypoglycaemics analgesics, antidepressants, antivirals and others that may change the risk of developing perioperative complications. Patients with difficult vascular access can undergo special procedures preoperatively like ultrasound guided peripherally inserted central catheter (PICC) line insertion which can save operating room time.

Key Point

- wound patients typically have comorbidities which need to be addressed prior to the start of the debridement, many requiring hospital admission for medical optimization

All patients are monitored intraoperatively according to ASA standards

All cases of sharp, surgical debridement in the operating room use anaesthesia services which apply the ASA standards for monitoring. In contrast to a 'local case' where 'local anaesthesia' is administered by the surgeon and monitoring is by non anaesthesia personnel who may or may not have other procedural responsibilities. The ASA has two standards for basic anaesthesia monitoring. One standard is based mainly on physical examination by a certified anaesthesia provider. That individual's sole responsibility is observing the mucosal colour, movement of the chest wall, rate and depth of respirations, presence of shivering or diaphoresis and response to painful stimuli of the patient for the duration of the procedure and anaesthesia care (57). The other standard is the continuous monitoring of oxygenation, ventilation, circulation and temperature (57). When the ASA standards are not followed, there is a 17·7% rate of complications (58).

Quantitative oxygenation assessment and continuous monitoring with pulse oximetry is based on the strong conviction that oxygen delivery and aerobic metabolism is most beneficial to the patient (59). Because of its ease of interpretation and continuous nature, pulse oximetry is used almost universally (57). Pulse oximetry does not replace monitoring spontaneous ventilation using observation of chest movement, auscultation of breath sounds and/or capnography (60). Capnography is effective in assessing airway patency and respiratory rate. The change of an end tidal carbon dioxide concentration waveform can indicate an abnormality such as an airway obstruction and/or apnoea (61).

Proper intraoperative monitoring standards of circulation require a continuous electrocardiogram (ECG) display, heart rate, pulse and arterial blood pressure measurements recorded at least every 5 minutes. The ECG Lead II can best detect atrial dysrhythmias (62) and in combination with V_5 provide the best sensitivity for ischaemia recognition short of transoesophageal echocardiography (63,64).

Pulse can be assessed by physical examination (palpitation, auscultation) or by technological monitoring (oximetry or invasive pressure measurement) (59). Heart rate measured by the ECG (voltage) can display a normal number during pulseless electrical activity (PEA) or electrical mechanical dissociation. Causes of PEA include hypovolaemia, hypoxaemia, hyperkalaemia, acidosis, myocardial infarction, pneumothorax, pericardial tamponade and pulmonary embolism. Therefore, pulse should be routinely measured by plethesmography with pulse oximetry. The pleth signal is reassuring when present, but may be absent for a variety of reasons ranging from artefact to cardiac arrest. In general, adequate perfusion to the finger tips or ear lobes suggests adequate perfusion to the vessel rich group including brain, heart and liver.

ASA standards recommend temperature to be monitored (57). Although MH is rare, it is potentially fatal. Fever associated with systemic inflammatory response syndrome (SIRS) or sepsis can be seen in the occasional infected patient. Hypothermia is a daily risk in the operating room because the cold physical environment and heat loss factors including conduction, convection, radiation and evaporation. Neuroaxial blocks and general anaesthesia impair the patient's ability to maintain central autonomic thermoregulatory control and peripheral thermogenesis (65). Sedatives administered such as propofol (66,67), midazolam (68,69) and opioids (70,71) reduce vasoconstriction and decrease shivering thresholds, furthering hampering thermogenesis.

This protocol also recommends glucose management to prevent and treat hypoglycaemia or hyperglycaemia which has negative consequences for wound infections (72) and a linear correlation with death rates (73). Even though BIS (bispectral EEG) and EMG monitoring are not required, the use of brain monitors can help optimise hypnotic drug titration for deep sedation and general anaesthesia. Given the risk of awareness during surgery when using neuromuscular blockade, BIS monitoring is highly recommended for general anaesthetics. BIS values less than 70 are associated with a chance of recall less than 1 in 1 million. Extremely low BIS values with high suppression ratios are undesirable and can increase risk of mortality (74). The question has been raised as to whether we use regional anaesthesia with sedation? If not, how do patients tolerate hearing the events in the OR? Patient-centred care challenges us to be concerned about patient satisfaction and emotional needs in the operating room. Sedation should be titrated to the patient's needs. Some patients prefer to be 'wide awake' as

long as they do not feel pain. The sense of control gives them comfort. Other patients have fears of mutilation and aversion to the smell of burning flesh from the cautery so they prefer to be 'asleep'. Other patients simply want to enjoy the good feeling of IV anaesthetics administered in a safe environment. The preoperative assessment provides the strategy for sedation when using regional anaesthesia. The 'events' in the OR can be more pleasant for the awake patient with a warm, nurturing environment cultivated by empathetic staff. In most operating rooms, the 'events' are routine for staff, but unusual for most patients except those who have frequent surgical procedures. Catastrophic events are uncommon so 'drama' is usually unnecessary.

Regional anaesthesia (local, peripheral or central) with sedation (none, light or deep) is administered as the default regimen with general IV and/or inhalational agents used when blocks are not carried out

The approach to the anaesthesia technique is influenced by the patient position during surgery. For example, sacral ulcers can be debrided in the lateral position or the prone position. The prone position requires an extra level of assessment and concern about airway management and ventilation. What about the issues of airway management of the prone patient needing debridement of a sacral pressure ulcer? The priorities for airway management include maintenance of airway patency, oxygenation and ventilation. The more sedation required for the case, the greater the likelihood of apnoea and/or airway obstruction. Paraplegics or patients with spinal anaesthesia can be positioned prone while awake to ensure comfort and adequate ventilation because positioning can be problematic. When antinociception is adequate, minimal sedation is required. Many anaesthesiologist will use endotracheal intubation for all prone cases to avoid the challenges of airway management without an endotracheal tube (ET). We hypothesise that neuromuscular blockade commonly used for intubation presents unnecessary problems with muscular weakness in debilitated patients which could lead to respiratory failure. This is a topic for a separate paper.

Patients with spinal cord injury always need individualised assessment of their anaesthetic need. The unique pattern of impairment including spasticity, may determine the strategy chosen to maintain comfort and homeostatsis. In our practice, neuropathic patients especially paraplegics and quadriplegics as well as those with regional blocks such as local or spinal require little or no sedation and can breathe spontaneously. Chest rolls are used to allow diaphragmatic offloading to prevent restricted ventilation. Deep sedation or general anaesthesia prompts most practitioners to intubate patients for the prone position. In contrast, the lateral position provides better access to the airway and more favourable mechanics for spontaneous ventilation compared with the prone position. Sedating a patient for sacral debridement in the lateral position is similar to colonoscopy assuming there is antinociception.

In the absence of complete central neuropathy like paraplegia, antinociception would be needed pharmacologically using sodium channel blockade. Regional blocks can be categorised as local infiltration blocks, peripheral blocks or central blocks (Figure 1). One regional anaesthesia regimen uses a local infiltration block with sedation as needed for amnesia and hypnosis. If no open wound is present, a transcutaneous injection of sodium channel blocker like 1% lidocaine is performed prior to the incision. In the presence of an open wound, local anaesthesia can be administered topically or subcutaneously. The subcutaneous technique for local anaesthesia injection into the wound edge is less painful compared with transcutaneous injection (75). When injecting local anaesthesia to the skin, it is important

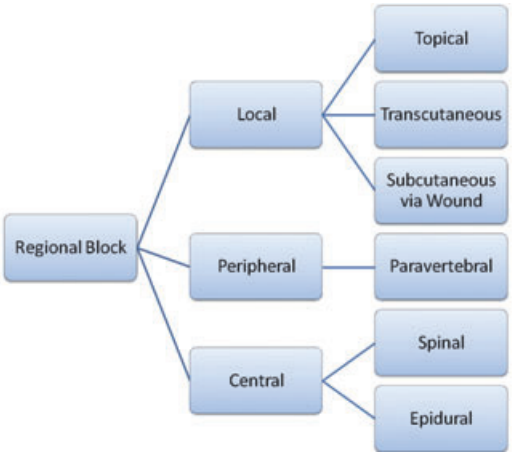

Figure 1. Regional anaesthesia hierarchy for sacral pressure ulcers. © O'Neill & Ayello 2012

to deposit the anaesthetic in or barely under the dermis layer, otherwise the small, unmyelinated nociceptive fibres that populate the epidermis will not be anaesthetised (76). It is difficult to fully anaesthetise infected tissue with Lidocaine. Topical agents do not penetrate devitalised tissues. Locally infiltrated sodium channel blockers require adequate distribution of the drug to the afferent nerves. Infected and devitalised tissues may be difficult to 'numb' using local techniques. Regional techniques including peripheral and central block or general anaesthesia are alternatives to local infiltration when drug tissue distribution factors exist.

The total dose of local anaesthetic adjusted for body weight should be below the limit for toxicity. Local anaesthetic toxicity needs to be accounted for as high plasma concentrations of these drugs can cause seizures, respiratory failure and cardiovascular collapse (77). The systemic effects are noted because sodium channels exist in both nerves and muscles including the heart. The total dose of sodium channel blocker combined with the rate of administration, absorption and elimination could influence the risk of complications (24).

Peripheral blocks or central blocks can also be used for pressure ulcers. Paravertebral blocks are more commonly used for trochanteric or ischial pressure ulcers compared with sacral pressure ulcers. Central blocks for sacral procedures include spinal or epidural blocks for antinociception with sedation as needed. Lidocaine, bupivacaine and tetracaine are the three most common spinal blockade drugs. Bupivacaine has almost replaced the use of lidocaine because less incidence of transient neurological symptoms postoperatively (78–80). Intrathecal opioids have little effect on haemodynamics, but can cause respiratory depression. Epidural blocks require higher drug dosages and volumes compared with spinal blocks because the dura mater limits the drug distribution to the cerebral spinal fluid. Lidocaine is the most common epidural anaesthetic but bupivacaine and ropivacaine can also be used as longer lasting local anaesthetics. However, central blocks with sodium channel blockade can lead to sympathectomy, bradycardia, decreased preload, decreased afterload, decreased cardiac output and hypotensison (24). Therefore, central blockades require adequate vascular access, fluid administration and vasopressors (phenylephrine, ephedrine or norepinephrine) as needed. The location of drug delivery influences the efficacy and complication rate (81,82).

Regional anaesthesia with sedation as needed is the default regimen of this protocol with general anaesthesia as a second choice. Sedation should be used for amnesia or hypnosis if the patient has anxiety that would prevent a safe procedure. Light sedation using midazolam (0·01 mg kg IV PRN) and fentanyl (0·5 μg/kg IV PRN) for analgesia is the suggested combination. For a patient with sleep apnoea or a difficult airway, a dexmedetomidine (0·2–0·6 μg/kg/minute) (83) infusion could be used because it produces less respiratory depression (84). When deep sedation and hypnosis become necessary, the light sedative drugs should be used in combination with propofol (25–200 μg/kg/minute) plus or minus ketamine (20–50 μg/kg IV PRN).

The Pro-Etom Infusion™ (30 ml total at 0·6–1·2 ml/kg/hour) is used by some anaesthesia providers to blend the benefits of propofol (200 mg in 20 ml) and etomidate (20 mg in 10 ml). Methohexothal infusion (25–200 μg/kg/minutes) can be used when propofol is not desirable for deep sedation (85). Opioid and non opioid (ketorolac or acetaminophen) analgesic agents have been used to supplement sedative anaesthetics to increase patient comfort during surgery (86).

When antinociception fails to be achieved with underlying neuropathy or a sodium channel blockade, the anaesthesiologist should use general anaesthetics. Total intravenous anaesthesia (TIVA) with propofol (25–200 μg/kg/minutes), fentanyl (0·5 μg/kg IV PRN) or remifentanil (25–200 ng/kg/minutes) plus or minus ketamine (20–50 μg/kg IV PRN) is one option. Another option is to balance general IV agents with the inhalational agents (isoflurane, sevoflurane or desflurane). This technique may be preferred when the patient presents to the operating room with an ET or tracheostomy. Inhalational agents could be used with or without blocks or IV administration. However, this technique using sevoflurane and nitrous oxide has a slower induction time compared with TIVA (87). In a pure inhalational anaesthesia technique, there is no background IV analgesia for postoperative pain control (88). As commonly performed with paediatric patients, inhalational induction with sevoflurane with nitrous oxide can be used to obtain vascular

access if the adult patient presents with excessive anxiety or needle phobia (89).

Neuromuscular blockade should only be used as a last resort as a part of general anaesthesia. Succinycholine is extremely useful for rapid sequence induction and treatment of laryngospasm. Short acting succcinylcholine, a depolarising muscle relaxant, can have severe side effects like hyperkalaemia, especially in patients with extrajunctional nicotinic receptors from disability. Although uncommon, succinylcholine can trigger MH leading to hypercarbia, hypoxia, hyperthermia, hyperkalaemia and death even if treated with dantrolene upon recognition of the symptoms (90). In addition, non depolarising blockers such as rocuronium, vecuronium and *cis*-atracurium can be associated with residual neuromuscular weakness which can increase risks of respiratory failure in debilitated patients with sarcopenia.

Goals of airway management are to maintain airway patency, oxygenation and ventilation using the minimum invasiveness of airway devices

This protocol recommends that airway management without tracheal intubation be considered whenever possible on the premise that complications associated with intubation are worth minimising. These airway management techniques include simple face mask ventilation, oral and nasal airways and supraglottic devices like laryngeal mask airways (LMA). However, this protocol acknowledges that ETs or surgical airways (existing tracheostomies or rarely, emergency cricothyroidotomy) may be necessary occasionally.

Lets review respiratory physiology and the difference between oxygenation and ventilation (Figure 2). Catabolism, which is half of metabolism, involves oxidation of fuels such as glucose, amino acids and fatty acids to produce chemical energy in the form of adenosine tri-phosphate (ATP). The metabolic rate can be estimated by oxygen consumption (VO_2) and/or carbon dioxide production (VCO_2). Oxygen delivery represents the transport of inhaled oxygen to the cellular mitochondria for oxidative phosphorylation. The carbon dioxide from the decarboxylation steps in the Kreb's tricarboxylic acid cycle gets transported to the alveoli via the venous blood for diffusion, dilution and exhalation. This 'fuel exhaust' can be measured by infrared spectroscopy and other techniques at the airway as an index of airway patency and quality of ventilation without measurement of an arterial blood gas.

Continuous assessment of oxygenation and ventilation is extremely important when sedation is given. Vigilance is essential to allow early detection, early intervention and avoiding catastrophes such as hypoxic brain injury from airway obstruction, apnoea and/or cardiac arrest.

Ventilation methods are classified into three categories (Figure 3). Unsupported negative pressure spontaneous ventilation is most natural which can be maintained with any airway device or circuit. Positive pressure ventilation requires a breathing system that generates a pressure above atmospheric pressure to overcome resistance and compliance factors. Manual ventilation using the circle system with anaesthesia bag, the ambu bag (manual resuscitator), or Jackson Rees circuit bag provides positive pressure by the hand of the anaesthesia provider. Controlled mechanical ventilation provides positive pressure ventilation using either volume control or pressure control using either an ICU ventilator or standard anaesthesia machine. The breaths can be either mandatory (set number machine triggered) or spontaneous (patient triggered). Synchronisation modes are designed to prevent patient-ventilator asynchrony which can present as the patient 'fighting the ventilator' and/or as ventilator associated lung injury like barotrauma. Newer anaesthesia machines have sensors and other features which allow them to function like sophisticated ICU ventilators.

Room air light sedation techniques allow pulse oximetry to be an index of hypoventilation as well as hypoxaemia based on the oxygen-haemoglobin saturation curve. Hypercarbia will cause desaturation when the alveoli contain 78% nitrogen (room air), but may not when alveolar oxygen is higher (91). When supplemental oxygen is used, capnography should be monitored to ensure airway patency and respiration rate (Figure 4 – capnography). An abnormality in the CO_2 wave can be artefact, central apnoea or airway obstruction (61) (Figure 4B – capnography). The disruption of the waveform should prompt immediate action for assessment and intervention.

Simple face masks and nasal cannula can promote small increases in the fraction

Figure 2. Review of respiratory physiology. © O'Neill & Ayello 2012

of inspired oxygen (FiO_2) which helps to alleviate hypoxaemia (92,93). Simple face masks provide supplemental oxygen to both the nose and mouth while providing the opportunity for superior gas analysis compared with nasal cannula (Figure 5 – simple face mask with gas sampling). Mouth breathers perform better with simple face masks. Exhaled water vapour accumulated on the face mask during ventilation can be detected, but this protocol still suggests digital and graphical display of ventilation via capnography. This protocol recommends clear-plastic masks with large volume, low-pressure cushions which provide an easy seal while simultaneously allowing for easy observation of condensation and evaporation from ventilation or the unexpected vomited stomach contents. A mask with a strap can be used to achieve a tighter seal to prevent room air dilutions of 100% O_2 and permitting positive pressure ventilation to treat airway obstructions, apnoea and/or hypoventilation. Using the circle system and anaesthesia bag, one can measure the expired tidal volumes and airway pressure associated with manual or controlled ventilation. An ambu bag is required to be present in all procedure rooms for all cases as a low-tech resuscitation device.

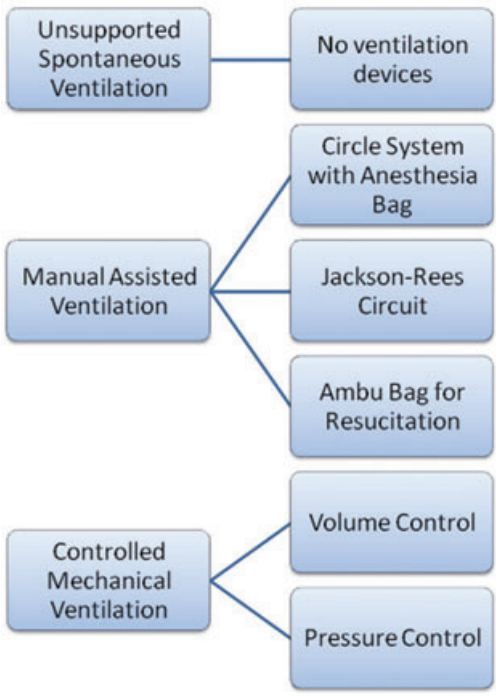

Figure 3. Classification of ventilation techniques. © O'Neill & Ayello 2012

If a seal between the mask and the face cannot be properly achieved alternative methods must be implemented. Failed seals can occur as a result of patient intolerance (cannot stand the mask on their face) which can lead to extended periods of time with the mask removed or poorly positioned to increase comfort (94). Nasopharyngeal oxygen via nasal cannula positioned at the exterior nostrils is a safe and comfortable alternative to face masks (94). Newer nasal cannula models have gas exchange analysis ports in their design which makes them more desirable than older models which had limitations like monitoring CO_2.

The evolution of supraglottic airway devices including the LMA and I-Gel have been an advancement in anaesthesia (Figure 6 – airway devices). Supraglottic airway devices keep the tongue and pharyngeal soft tissue away from the lumen of the trachea to maintain airway patency. This is important as the role of the anaesthesiologist is dynamic management and multitasking. As the anaesthesiologist no longer has to manually maintain the adequate seal and mask fit with chin lift, this frees up the anaesthesiologist's hands which can then be used to maintain IVs, preparation and administration of drugs and even fulfil other responsibilities including contemporaneous documentation (95).

Why use an LMA over an ET? Supraglottic airway can be inserted without using neuromuscular blockade which is an advantage compared with tracheal intubation (96). In addition, non anaesthesiologists are typically more successful at placing LMAs compared with performing tracheal intubations (97). The absence of neuromuscular blockade can allow spontaneous ventilation which is desirable, especially in an outpatient surgical setting for optimal drug titration and faster emergence (95). Patients receiving general anaesthesia in the outpatient setting for LMAs compared with ETs have been shown to have shorter PACU stays because the drug concentrations are more favourable with fewer side effects (96).

ETs with general anaesthetics using either spontaneous or positive pressure ventilation add a level of safety because respiratory depression and loss of reflexes are expected.

In typical patients, usually the anaesthesiologist will induce anaesthesia prior to intubation which leads to amnesia, apnoea and areflexia. In high risk airway patients, where intubation may be challenging or difficult, spontaneous ventilation is maintained until ET placement is confirmed to decrease risk of hypoxaemia. Therefore in high risk airway patients, the intubation plan is opposite that the usual plan (Table 3). ETs are commonly inserted using direct laryngoscopy for placement below the vocal cords and above the tracheal carina. Video scopes such as the glide scope or fibreoptic bronchoscopes may be necessary for some challenging intubations. Like the LMA, ETs maintain airway patency while freeing the hands of the anaesthesiologist to attend to other responsibilities. In addition to reliably preventing pharyngeal collapse and laryngospasm, the ET with the inflatable cuff decreases the risk of pulmonary aspiration of the gastric contents and saliva (Figure 6 – LMA versus ET).

Surgical airways remain the last resort. Elective patients with respiratory failure or severe dysphagia may require tracheostomy. Emergency cricothyroidotomy is the treatment of choice for the 'unable to intubate–unable to ventilate' scenario which the ASA difficult airway algorithm was designed to avoid.

Induction of general anaesthesia for intubation results in amnesia, apnoea and areflexia.

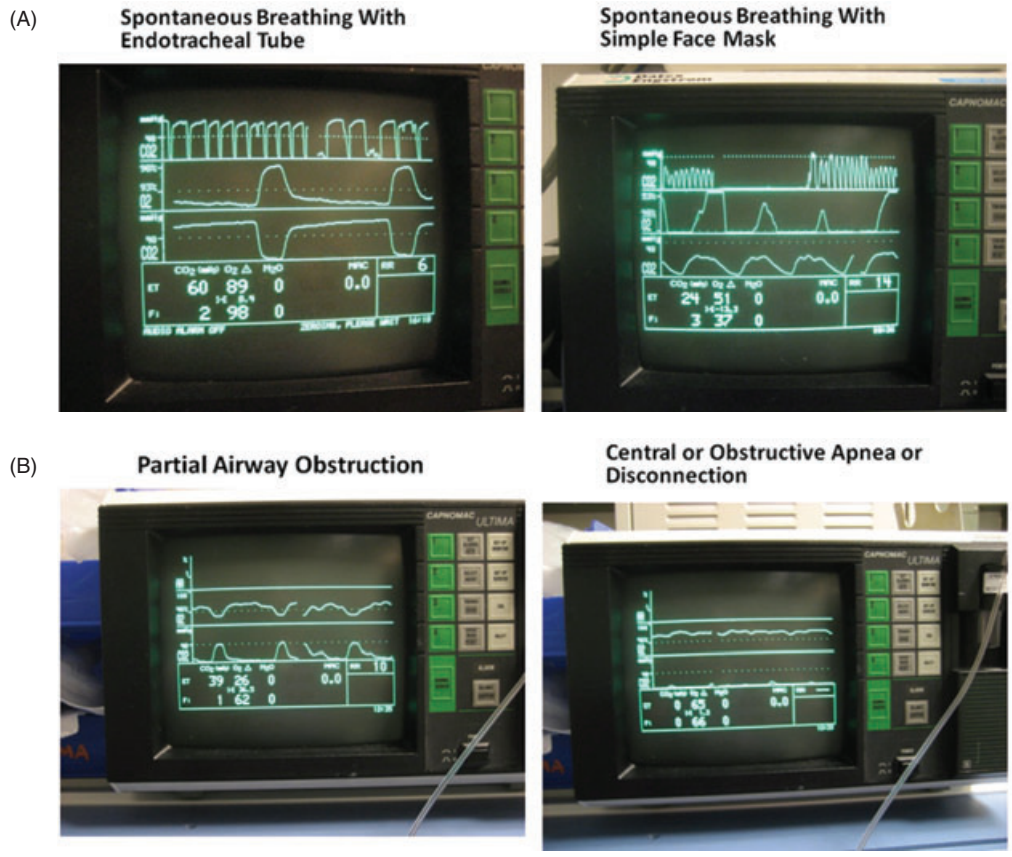

Figure 4. (A) Capnography part 1, endotracheal tube mask and (B) capnography with abnormalities. © O'Neill & Ayello 2012

Figure 5. Face mask gas sampling and oxygen delivery. © O'Neill & Ayello 2012

Consequently, there is a risk of airway obstruction, hypoxaemia and hypercarbia (respiratory acidosis) if mask ventilation with oxygen and/or intubation are not successful. There are two major approaches to endotracheal intubation (Table 3): plan A is induction of anaesthesia followed by intubation; Plan B is intubation followed by induction of anaesthesia. Plan A is most commonly used because most patients are NPO and are likely to be easy to mask ventilate and intubate easily using direct laryngoscopy. Plan B is used for the challenging or difficult intubation because spontaneous ventilation with maintenance of airway reflexes minimises the risk of hypoxaemia. The spontaneous breathing or so-called 'awake' technique usually involves airway topicalisation with sodium channel blockers such as lidocaine and sedation (midazolam, fentanyl, droperidol, dexmedetomine, ketamine and/or propofol) for comfort and cooperation. Once the ET placement is confirmed by the usual methods, induction of anaesthesia can proceed without the risk of airway obstruction.

All patients are monitored postoperatively for the duration appropriate to their acuity

Generally, patients are transferred to the PACU postoperatively. The PACU should be staffed with the attending anaesthesiologist, trained recovery room nurses and other support personnel, who have a comprehensive understanding of possible complications

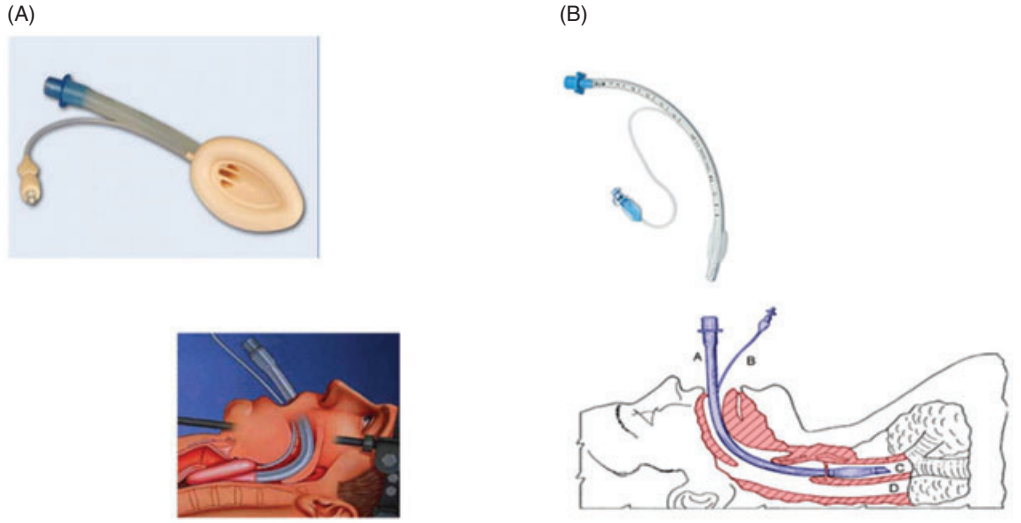

Figure 6. Airway devices: Head to head comparison. (A) Supraglotic laryngeal mask. (B) Endotracheal tube.

Table 3 Airway management strategies for intubation © O'Neill & Ayello 2012

Plan A	Induce anaesthesia, then intubate
Plan B	Intubate, then induce anaesthesia

Table 4 Basis of assessment for discharge from anaesthesia (Aldrete) © O'Neill & Ayello 2012

1	Haemodynamic stability achieved
2	Low risk of blood loss from surgical site
3	Baseline neurological status satisfactory
4	Low risk of respiratory depression
5	Comfortable

and treatment protocols (89,90). Patients are monitored until they meet the discharge criteria (Table 4). The ASA guidelines as summarised by the Aldrete score state that prior to PACU discharge, patients should be at baseline neurological status (awake and alert, if so preop) with vital signs within acceptable limits (98). Patients need to meet an acceptable hospital specific score of a set of criteria defined by where the procedure was performed. For outpatients, a responsible adult should be present to take the patient home if acceptable and a written set of instructions should be provided with the patient (98). Although the anaesthesiologist may occasionally choose to bypass the PACU, patients from the operating room usually go to the PACU prior to transfer to the floor, ICU or discharged home.

When the anaesthesiologist decides, it may be possible to 'fast track' the recovery patients by bypassing the PACU (Table 4) (99). This approach is usually used for patients who had ambulatory surgery or inpatients with local anaesthesia with minimal sedation or prolonged PACU hold times. PACU bypass is a cost effective, safe and efficient alternative to the PACU for these patients (99–102). This technique should only be used when the anaesthesiologist feels that the patient is at minimal risk for developing any postoperative complications.

DISCUSSION

Patients with pressure ulcers are typically quite ill and have multiple comorbidities making wound management and healing a challenge. Debridement is an important component of wound bed preparation. As debridement removes non functioning keratinocytes and bioburden, it must be extensive enough to provide the scaffold for tissue repair and stimulation of wound healing. Adequacy of debridement to the 'margin of response' is based on the biological understanding of the impairments to healing of a chronic wound (103). Assuring adequacy of debridement requires probing and manipulation of the wound that is painful. Pressure ulcer pain may preclude using some methods of debridement. Management of pain so that enough debridement of the wound can be undertaken is essential.

Advances in anaesthesiology over the last two decades including pulse oximetry,

Key Point

- patients with pressure ulcers are typically quite ill and have multiple comorbidities making wound management and healing a challenge

Key Points

- management of pain so that enough debridement of the wound can be undertaken is essential
- significantly lower mortality rates for wound patients who had debridements compared with those who did not suggest this surgery to be life saving
- as no literature exists on using a protocol emphasising regional anaesthesia with sedation as needed for operative debridement, it is the recommendation that through this protocol a retrospective chart review on outcomes of sacral pressure ulcer debridements with regional anaesthesia be performed
- a randomised, stratified prospective clinical trial with the use of this protocol is also suggested for the future

capnography, advanced airway management, as well as, ultrasound guided vascular access and regional block techniques have improved patient safety in the operating room. Although there are inherent risks with anaesthesia, fear of the operating room, especially receiving anaesthesia, should not prevent a wound patient from obtaining the surgical debridement they desperately need to stimulate wound closure. Significantly lower mortality rates for wound patients who had debridements compared with those who did not (2,10,104) suggest this surgery to be life saving. The use of this anaesthesia protocol will undoubtedly assist the attending physicians of wound patients in securing consent from the patient and performing a safe, successful debridement with minimal complications.

This anaesthesia protocol promotes the premise that subspeciality training in wound anaesthesia is advantageous for patients with non healing wounds like sacral pressure ulcers. As no literature exists on using a protocol emphasising regional anaesthesia with sedation as needed for operative debridement, it is the recommendation that through this protocol a retrospective chart review on outcomes of sacral pressure ulcer debridements with regional anaesthesia be performed. A randomised, stratified prospective clinical trial with the use of this protocol is also suggested for the future.

ACKNOWLEDGEMENT

This work was supported by NIH Grant RO1 LM008443-04.

REFERENCES

1 National Pressure Ulcer Advisory Panel (NPUAP) and European Pressure Ulcer Advisory Panel (EPUAP). Prevention and treatment of pressure ulcers: clinical practice guideline. Washington, DC: National Pressure Ulcer Advisory Panel, 2009.
2 Brem H, Lyder CH. Protocol for the successful treatment of pressure ulcers. Am Surg 2004;188 Suppl:9S–17S
3 Lyder CH. Pressure ulcer prevention and management. JAMA 2003;289:223–6.
4 Russo CA, Steiner C, Spector W. Hospitalizations related to pressure ulcers among adults 18 years and older 2006. Healthcare Cost and Utilization Project (HCUP) Statistical Brief 64. Rockville, MD: Agency for Healthcare Research and Quality, 2008. URL http://www.hcup-us.ahrq.gov/reports/statbriefs/sb64.pdf [accessed on 21 June 2010]
5 Allman RM, Goode PS, Burst N, Bartolucci AA, Thomas DR. Pressure ulcers, hospital complications, and disease severity: impact on hospital costs and length of stay. Adv Wound Care 1999;12:22–30.
6 Landi F, Onder G, Russo A, Bernabei R. Pressure ulcer and mortality in frail elderly people living in community. Arch Gerontol Geriatr 2007;44 Suppl 1:217–23.
7 Redelings MD, Lee NE, Sorvillo F. Pressure ulcers: more lethal than we thought? Adv Skin Wound Care 2005;18:367–72.
8 Black JM, Cuddigan JE, Walko MA, Didier LA, Lander MJ, Kelpe MR. Medical device related pressure ulcers in hospitalized patients. Int Wound J 2010 [Epub].
9 VanGilder C, Amlung S, Harrision S, Meyer S. Results of the 2008–2009 international pressure ulcer prevalence survey and a 3 year, acute care, unit-specific analysis. Ostomy Wound Manage 2009;55:39–45.
10 Schiffman J, Golinko MS, Yan A, Flattau A, Tomic-Canic M, Brem H. Operative debridement of pressure ulcers. World J Surg 2009;33:1396–402.
11 VanGilder C, MacFarlane GD, Harrison P, Lachenbruch C, Meyer S. The demographics of suspected deep tissue injury in the United States: an analysis of the International Pressure Ulcer Prevalence Survey 2006–2009. Adv Skin Wound Care 2010;3:254–61.
12 Lazarus GS, Cooper DM, Knighton DR, Margolis DJ, Pecoraro RE, Rodeheaver G, Robson MC. Definitions and guidelines for assessment of wounds and evaluation of healing. Arch Dermatol 1994;130:489–93.
13 Mostow EN. Diagnosis and classification of chronic wounds. Clin Dermatol 1994;12:3–9.
14 Stojadinovic O, Brem H, Vouthounis C, Lee B, Fellon J, Stallcup M, Merchant A, Galiano RD, Tomic-Canic M. Molecular pathogenesis of chronic wounds: the role of beta-catenin and c-myc in the inhibition of epithelialization and wound healing. Am J Pathol 2005;167:59–69.
15 Auerbach R, Auerbach W. Regional differences in the growth of normal and neoplastic cells. Science 1982;215:127–34.
16 Auerbach R, Morrissey LW, Kubai L, Sidky YA. Regional differences in tumor growth: studies of the vascualr system. Int J Cancer 1978;22:40–6.
17 Kubai L, Auerbach R. Regional differences in the growth of skin transplants. Transplantation 1980;30:128–31.
18 Ger R. Fecal diversion in management of large infected perianal lesions. Dis Colon Rectum 1996;39:1327–9.
19 Beldon P. Problems encountered managing pressure ulceration of the sacrum. Br J Community Nurs 2008;13;S6, S8, 10passim.
20 Longe RL. Current concepts in clinical therapeutics: pressure sores. Clin Pharm 1986;5:669–81.
21 Brem H, Nierman DM, Nelson JE. Pressure ulcers in the chronically critically ill patient. Crit Care Clin 2002;18:683–94.
22 Freedman G, Entero H, Brem H. Practical treatment of pain in patients with chronic wounds:

pathogenesis-guided management. Am J Surg 2004;188 Suppl:31S–5S.
23 O'Neill DK. Operative anesthesia for stage 3 and 4 pressure ulcers, July 2010, Lecture, NYU Kimmel Wound Center Pressure Ulcer Course Lecture. New York University Langone Medical Center.
24 Faccenda KA, Finucane BT. Complications of regional anesthesia: incidence and prevention. Drug Saf 2001;24:413–42.
25 Lewicki LJ, Mion L, Splane KG, Samstag D, Secic M. Patient risk factors for pressure ulcers during cardiac surgery. AORN J 1997;65:933–42.
26 Grous CA, Reilly NJ, Gift AG. Skin integrity in patients undergoing prolonged operations. J Wound Ostomy Continence Nurs 1997;24: 86–91.
27 Schoonhoven L, Defloor T, van der Tweel I, Buskens E, Grypdonck MH. Risk indicators for pressure ulcers during surgery. Appl Nurs Res 2002;15:163–73.
28 Orkin FK. Meaningful cost reduction. Penny wise, pound foolish. Anesthesiology 1995;83:1135–7.
29 White PF, White LD. Cost containment in the operating room: who is responsible? J Clin Anesth 1994;6:351–6.
30 Vogel WA, Manecke GR Jr, Poppers PJ. The ethics of cost containment from the anesthesiologist's perspective. J Clin Anesth 1999;11:73–7.
31 Schuster M, Standl T. Cost drivers in anesthesia: manpower, technique and other factors. J Curr Opin Anaesthesiol 2006;19:177–84.
32 Korhonen AM, Valanne JV, Jokela RM, Ravaska P, Kortitila KT. A comparison of selective spinal anesthesia with hyperbaric bupivacaine and general anesthesia with desflurane for outpatient knee arthroscopy. Anesth Analg 2004; 99: 1668–73.
33 Williams BA, Kentor ML, Vogt MT, Vogt WB, Coley KC, Wiliams JP, Roberts MS, Chelly JE, Harner CD, Fu FH. Economics of nerve block pain management after anterior cruciate ligament reconstruction: potential hospital cost savings via associated postanesthesia care unit bypass and same-day discharge. Anesthesiology 2004; 100:697–706.
34 O'Neill DK, Maggi J. Anesthetic care for patients with skin breakdown. J Clin Anesth 2009;27: 599–603.
35 O'Neill DK, Tsui S, Ayello EA, Cuff G, Brem H. Anesthesia protocol for heel pressure ulcer debridement. Adv Skin Wound Care 2012;25: 209–19.
36 Sweitzer Bobbie J. Overview of preoperative assessment and management. Chapter 4. In: Longnecker DE, Brown DL, Newman MF, Zapol WM, editors. Anesthesiology. New York: McGraw Hill Medical, 2008.
37 Hanci V, Ayoglu H, Yurtlu S, Yildirim N, Okyay RD, Erdogan G, Sayin E, Turan IO. An evaluation of P wave dispersion, QT, corrected QT and corrected QT dispersion intervals on the electrocardiograms of malnourished adults. Anaesth Intensive Care 2010;38:122–7.
38 Kluger MT, Tham EJ, Coleman NA, Runciman WB, Bullock MF. Inadequate pre-operative evaluation and preparation: a review of 197 reports from the Australian incident monitoring study. Anaesthesia 2000;55:1173–8.
39 Davis NJ, ed. Anaesthesia-related mortality in Australia 1994–1996. Melbourne, Australia: Capital Press, 1999.
40 Morgan GE Jr, Mikhail MS, Murray MJ. The practice of anesthesiology. Chapter 1. In: Morgan GE Jr, Mikhail MS, Murray MJ, editors. Clinical anesthesiology, 4rd edition. McGraw-Hill Companies Inc, 2006.
41 Pasternak LR, Arens JF, Caplan RA, Connis RT. Practice advisory for preanesthesia evaluation. J Anesth 2002;96:485–96.
42 Zaidan JR, Atlee JL, Belott P, Briesacher KS. Practice advisory for the perioperative management of patients with cardiac rhythm management devices: pacemakers and implantable cardiover ter-defibrillators. J Anesth 2005;103:186–98.
43 Atlee JL, Bernstein AD. Cardiac rhythm management devices (Part I): indications, device, selection and function. J Anesth 2001;95:1265–80.
44 Atlee JL, Bernstein AD. Cardiac rhythm management devices (Part II): perioperative management. J Anesth 2001;95:1492–506.
45 Buchanan K, Bernstein N, Ayello EA, O'Neill DK. Cardiac device interrogation for safer care of surgical wound patients. Adv Skin Wound Care 2011; 24:507–14.
46 Crossley GH, Poole JE, Rozner MA. The Heart Rhythm Society Expert Consensus Statement on the perioperative management of patients with implantable defibrillators, pacemakers and arrhythmia monitors: facilities and patient management, Heart Rhythm Society, May 2011.
47 Jacob S, Shahzad MA, Maheshwari R, Panaich SS, Aravindhakshan R. Cardiac rhythm device identification algorithm using X-Rays: CaRDIA-X. Heart Rhythm 2011; 8:915–22.
48 Rozner MA. The patient with a cardiac pacemaker or implanted defibrillator and management during anaesthesia. Curr Opin Anaesthesiol 2007;20:261–8.
49 Anderson M, Comrie R. Adopting preoperative fasting guidelines. AORN 2009;90:73–80.
50 Yoneyama T, Yoshida M, Ohrui T, Mukaiyama H, Hoshiba K, Ihara S. Oral care reduces pneumonia in older patients in nursing homes. JAGS 2002; 50:430–3.
51 Scannapieco FA. Pneumonia in nonambulatory patients: the role of oral bacteria and oral hygiene JADA 2006; 137:21S–5S.
52 Crenshaw JT, Winslow EH. Preoperative fasting: old habits die hard. Am J Nurs 2002;102:36–44; quiz 45.
53 Green CR, Pandit SK, Schork MA. Preoperative fasting time: is the traditional policy changing? Results of a national survey. Anesth Analg 1996; 83:123–8.
54 Shime N, Ono A, Chihara E, Tanaka Y. Current practice of preoperative fasting: a nationwide survey in Japanese anesthesia-teaching hospitals. J Anesth 2005;19:187–92.

55 Diks J, van Hoorn DE, Nijveldt RJ, Boelens PG, Hofman Z, Bouritius H, van Norren K, van Leeuwen PA. Preoperative fasting: an outdated concept? JPEN 2005;29:298–304.
56 Warner MA, Caplan RA, Epstein BS, Gibbs CP, Keller CE, Leak JA, Maltby R, Nickinovich DG, Schreiner MS, Weinlander CM. Practice guidleines for preoperative fasting and the use of pharmacologic agents to reduce the risk of pulmonary aspiration: application to healthy patients undergoing elective procedures. J Anesth 1999;90:896–905.
57 Anonymous. Standards for basic anesthetic monitoring. ASA Directory of Members, 2004. http://www.asahq.org/For-Members/Standards-Guidelines-and-Statements.aspx
58 Tinker JH, Dull DL, Caplan RA, Ward RJ, Cheney FW. Role of monitoring devices in prevention of anesthetic mishaps: a closed claims analysis. J Anesth 1989;71:541–6.
59 Eichhorn JH. Pulse oximetry as a standard of practice in anesthesia. J Anesth 1993;78:423–6.
60 Harris RS, Sebel PS. Monitored anesthesia care and conscious sedation. Chapter 68. In: Longnecker DE, Brown DL, Newman MF, Zapol WM, editors. Anesthesiology. New York: McGraw Hill Medical, 2008.
61 Cheifetz IM, Myers TR. Should every mechanically ventilated patient be monitored with capnography from intubation to extubation? Respir Care 2007;52:423–42.
62 Kistner JR, Miller ED, Epstein RM. More than V_5 needed (letter). Anesthesiology 1977;47:75–6.
63 London MJ, Hollenberg M, Wong MG. Intraoperative myocardial ischemia: localization by continuous 12-lead electrocardiography. J Anesth 1988;69:232–41.
64 Blackburn H, Katigbak R. What electrocardiographic leads to take after exercise? Am Heart J 1964;67:184–5.
65 Sessler DI, Ponte J. Shivering during epidural anesthesia. Anesthesiology 1990;72:816–21.
66 Leslie K, Sessler DI, Bjorksten AR, Ozaki M, Matsukawa T, Schroeder M, Lin S. Propofol causes a dose-dependent decrease in the thermoregulatory threshold for vasoconstriction but has little effect on sweating. Anesthesiology 1994;81:353–60.
67 Matsukawa T, Kurz A, Sessler DI, Bjorksten AR, Merrifield B, Cheng C. Propofol linearly reduces the vasoconstriction and shivering thresholds. Anesthesiology 1995;82:1169–80.
68 Kurz A, Sessler DI, Annadata R, Dechert M, Christensen R, Bjorksten AR. Midazolam minimally impairs thermoregulatory control. Anesth Analg 1995;81:393–8.
69 Toyota K, Sakura S, Saito Y, Ozasa H, Uchida H. The effect of pre-operative administration of midazolam on the development of intraoperative hypothermia. Anaesthesia 2004;59:116–21.
70 Kurz A, Ikeda T, Sessler DI, Larson MD, Bjorksten AR, Dechert M, Christensen R. Meperidine decreases the shivering threshold twice as much as the vasoconstriction threshold. Anesthesiology 1997;86:1046–54.
71 Kurz A, Go JC, Sessler DI, Kaer K, Larson MD, Bjorksten AR. Alfentanil slightly increases the sweating threshold and markedly reduces the vasoconstriction and shivering thresholds. Anesthesiology 1995;83:293–9.
72 Zerr KJ, Furnary AP, Grunkemeier GL, Bookin S, Kanhere V, Starr A. Glucose control lowers the risk of wound infection in diabetics after open heart operations. Ann Thorac Surg 1997;63:356–61.
73 van den Berghe G, Wouters P, Weekers F, Verwaest C, Bruyninckx F, Schetz M, Vlasselaers D, Ferdinande P, Lauwers P, Bouillon R. Intensive insulin therapy in critically Ill patients. N Engl J Med 2001;345:1359–67.
74 Monk TG, Saini V, Weldon C, Sigl JC. Anesthetic management and one-year mortality after noncardiac surgery. Anesth Analg 2005;100:4–10.
75 Bartfield JM, Sokaris SJ, Raccio-Robak N. Local anesthesia for lacerations: pain of infiltration inside vs outside the wound. Acad Emerg Med 1998;5:100–4.
76 Hilliges M, Wang L, Johansson O. Ultrastructural evidence for nerve fibers within all vital layers of the human epidermis. J Invest Dermatol 1995;104:134–7.
77 Lambert LA, Lambert DH, Strichartz GR. Irreversible conduction block in isolated nerve by high concentrations of local anesthetics. J Anesth 1994;80:1082–93.
78 Hampl KL, Schneider MC, Ummenhofer W, Drewe J. Transient neurologic symptoms after spinal anesthesia. Anesth Analg 1995;81:1148–53.
79 Hampl KR, Heinzmann-Wiedmer S, Luginbuehl I, Harms C, Seeberger M, Schneider MC, Drasner K. Transient neurologic symptoms after spinal anesthesia: a lower incidence with prilocaine and bupivacaine than with lidocaine. J Anesth 1998;88:629–33.
80 Keld DB, Hein L, Dalgaard M, Krogh L, Rodt SA. The incidence of transient neurologic symptoms (TNS) after spinal anaesthesia in patients undergoing surgery in the supine position. Hyperbaric lidocaine 5% versus hyperbaric bupivacaine 0.5%. Acta Anaesthesiol Scand 2000;44:285–90.
81 Magnusdottir H, Kimo K, Ricksten S. High thoracic epidural analgesia does not inhibit sympathetic nerve activity in the lower extremities. Anesthesiology 1999;91:1299–304.
82 Basse L, Werner M, Kehlet H. Is urinary drainage necessary during continuous epidural anesthesia after colonic resection. Reg Anesthes Pain Med 2000;25:498–501.
83 Arain SR, Ebert TJ. The efficacy, side effects, and recovery characteristics of dexmedetomidine versus propofol when used for intraoperative sedation. Anesth Analg 2002;95:461–6.
84 Ramsay MA, Luterman DL. Dexmedetomidine as a total intravenous anesthetic agent. J Anesth 2004;101:787–90.
85 Sa Rego MM, Inagaki Y, White PF. Cost-effectiveness of methohexital versus propofol

for sedation during monitored anesthesia care. Anesth Analg 1999;88:723–8.
86 Gesztesi Z, Sa Rego MM, White PF. The comparative effectiveness of fentanyl and its newer analogs during extracorporeal shock wave lithotripsy under monitored anesthesia care. Anesth Analg 2000;90:567–70.
87 Hong JY, Kang YS, Kil HK. Anaesthesia for day case excisional breast biopsy: propofol-remifentanil compared with sevoflurane-nitrous oxide. Eur J Anaesthesiol 2008; 25:460–7.
88 Moore JK, Elliott RA, Payne K, Moore EW, St Leger AS, Harper NJ, Pollard BJ, Kerr J. The effect of anaesthetic agents on induction, recovery and patient preferences in adult day case surgery: a 7-day follow-up randomized controlled trial. Eur J Anaesthesiol 2008; 25:876–83.
89 Joshi GP. Inhalational techniques in ambulatory anesthesia. Anesthesiol Clin N Am 2003;21: 263–72.
90 Halliday NJ. Malignant hyperthermia. J Craniofac Surg 2003;14:800–2.
91 Sivilotti ML, Messenger DW, van Vlymen J, Dungery PE, Murray HE. A comparative evaluation of capnometry versus pulse oximetry during procedural sedation and analgesia on room air. CJEM 2010; 12:397–404.
92 Oh TE, Duncan AW. Oxygen therapy. Med J Aust 1988;149:141–6.
93 Considine J. The reliability of clinical indicators of oxygenation: a literature review. Contemp Nurse 2005; 18:258–67.
94 Eastwood GM, Dennis MJ. Nasopharyngeal (NPO) oxygen as a safe and comfortable alternative to face mask oxygen therapy. Aust Crit Care 2006;19:22–4.
95 Joshi GP, Inagaki Y, White PF, Taylor-Kennedy L, Wat LI, Gevirtz C, McCraney JM, McCulloch DA. Use of the laryngeal mask airway as an alternative to tracheal tube during ambulatory anesthesia. Anesth Analg 1997;85:573–7.
96 Luba K, Cutter TW. Supraglottic airway devices in the ambulatory setting. Anesthesiol Clin 2010;28:295–314.
97 Martin PD, Cyna AM, Hunter WA, Henry J, Ramayya GP. Training nursing staff in airway management for resuscitation. A clinical comparison of the face mask and laryngeal mask. Anaesthesia 1993;48:33–7.
98 American Society of Anesthesiologists Task Force on Postanesthetic Care. Practice guidelines for postanesthetic care: a report by the American Society of Anesthesiologists Task Force on Postanesthetic Care. J Anesth 2002;96:742–52.
99 White PF, Rawal S, Nguyen J, Watkins A. PACU fast-tracking: an alternative to ''bypassing'' the PACU for facilitating the recovery process after ambulatory surgery. J Perianesth Nurs 2003; 18:247–53.
100 Kiekkas P, Poulopoulou M, Papahatzi A, Androutsopoulou C, Maliouki M, Prinou A. Workload of postanaesthesia care unit nurses and intensive care overflow. Br J Nurs 2005;14:434–8.
101 Kluger MT, Bullock MF. Recovery room incidents: a review of 419 reports from the Anaesthetic Incident Monitoring Study (AIMS). Anaesthesia 2002;57:1060–6.
102 Marshall SI, Chung F. Discharge criteria and complications after ambulatory surgery. Anesth Analg 1999;88:508–17.
103 Tomic-Canic M, Ayello EA, Stojadinovic O, Golinko MS, Brem H. Using gene transcription patterns (Bar coding scans) to guide wound debridement and healing. Adv Skin Wound Care 2008;21:487–92.
104 Dellinger RP, Levy MM, Carlet JM, Bion J, Parker MM, Jaeschke R. Surviving sepsis campaign: international guidelines for management of severe sepsis and septic shock: 2008 Intensive Care Med 2008;34:17–60.