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ABSTRACT

The majority of patients admitted to the intensive care unit (ICU) have a short stay of only a few days. However a small but significant number require prolonged intensive care. This is typically due to persisting, and sometimes complex, medical/surgical problems. Discharge of such ICU patients requires a comprehensive, multidisciplinary, verbal and written handover to the receiving ward team. As with any acutely ill adult in hospital, post-ICU patients should be carefully monitored with 'track and trigger' systems such as the Early Warning Score. Those with unexpected physiological deterioration should be promptly reviewed by senior clinicians and/or medical emergency/critical care outreach teams and considered for ICU re-admission where appropriate. Patients who have received prolonged organ support in the ICU are often affected by a number of specific medical problems such as ventilatory insufficiency, cardiac dysfunction, kidney injury, nutritional deficiency, ICU acquired weakness, and brain injury. They also frequently experience physical disability and psychosocial problems including delirium, anxiety, depression, post-traumatic stress disorder, cognitive dysfunction, and disturbed sleep. Structured rehabilitation programmes for post-ICU patients, tailored to individual needs, should be commenced on the ICU and continued through to and beyond hospital discharge. Care bundles, which are widely used on the ICU, are groups of interventions employed to optimise treatments or minimise complication rates. They may be additionally useful in the post-ICU ward setting by prompting clinicians to focus on, and address, commonly occurring medical and psychosocial problems in these patients.

INTRODUCTION

Prolonged intensive care (ICU) admission is associated with the development of a number of specific medical and psychosocial problems in addition to the acute critical illness and pre-existing comorbidities. The heterogeneous group of patients discharged from ICU with such problems has been the subject of little research or published opinion and yet presents considerable challenges for the multidisciplinary team adopting responsibility for their ongoing care. This review aims to address this deficiency: first, by considering the common experiences of ICU patients and how these may impact on their continuing management; secondly, by discussing generic issues faced by the teams looking after such patients; and thirdly by suggesting how they may best be managed. Where available, evidenced based interventions and existing national guidelines are detailed. Adept management of post-ICU patients in the ward setting requires a good understanding of these specific problems. It also requires close coordination with other clinicians and all members of the multidisciplinary team.

PLANNING THE DISCHARGE FROM ICU TO WARD CARE

The discharge of patients to ward care following a prolonged ICU admission should be a planned event that seamlessly continues to promote a patient's recovery. A timely discharge from ICU to a suitable ward environment is an ideal not always realised due to practical constraints. Ideally, timing should reflect the patient's ongoing needs, the practical considerations of daytime care, and hospital bed management, such that evening/night-time, and even weekend discharges are best avoided,[1 2] ICU bed pressures from emergency and major elective surgery admissions can force earlier than planned discharges, but should be resisted wherever possible. In fact, delaying ICU discharge of the most at-risk patients for 24–48 h may reduce mortality.[1]

Discharge planning from the ICU should start at the earliest opportunity and, whenever practical, the team assuming care should review the patient on the ICU before transfer. A verbal handover should be supplemented by a concise but comprehensive, multidisciplinary, written summary in the patient's notes (see box 1 for a suggested outline). A problem plan based approach is recommended. A clear plan should also be documented for any predictable deterioration post-ICU discharge, together with any decision regarding limitation of therapy, including the appropriateness of re-admission to ICU.

In addition to the medical/nursing therapy handover, the ward team should also have the opportunity to meet the patient and prepare him or her for the transition. Patients often have fears and anxieties about transferring to a ward environment and may perceive that they will receive less frequent and less expert care. Expectation management is an important part of easing the transition for the patient. Involving the patient's next of kin/frequent visitors, and where appropriate, familiarising them with the intended ward environment, is also recommended.

In published series, approximately 10% of patients discharged from ICU are re-admitted during the same hospital stay. Early re-admissions (usually taken as <48 h) are often predictable[3] and can be used as a quality measure of ICU discharge. Older, sicker patients with longer ICU stays are unsurprisingly at highest risk of re-admission. A recent study showed that earlier-than-planned discharges from a surgical ICU were associated

Box 1 Suggested outline of ICU discharge summary

- Date and duration of ICU admission.
- Reason for ICU admission.
- List of diagnoses/active problems. Include a brief chronological history (including important results/diagnostic procedures/interventions) of each and the plans for future management. Include a list of positive and outstanding microbiology investigations and the details of recent/ongoing antimicrobial therapy complete with planned stop/review date.
- Brief description of clinical state.
- Frequency of observations and triggers for calling for help.
- Age and plans for any lines and drains.
- List of current medications and any chronic drugs that may need to be reinstated.
- Plans for nutritional and fluid management, as appropriate.
- Summary of patient awareness of condition and communication with relatives and other clinical teams.
- Rehabilitation plan and key therapist coordinating care.
- Predictable risks post discharge and re-escalation plan.

with a fourfold increase in re-admission rate and substantial increased risk of mortality.[4]

TRACK AND TRIGGER SYSTEMS

The UK National Institute for Health and Clinical Excellence (NICE) guidance on management of acutely ill adults in hospital,[5] including patients discharged from ICU, advocates the use of physiological scoring systems such as the Early Warning Score[6] to track a patient's physiological status and trigger an appropriate response. The response should involve timely review by senior members of the patient's own medical or surgical team, and/or by additional staff competent in resuscitation as part of a medical emergency team or critical care outreach team. Use of such tools should empower ward nurses to alert medical staff to patients with deteriorating conditions. Notably, however, such scoring systems perform variably in different institutions and may lack sensitivity.[7] Hence, recognition of acutely unwell or deteriorating patients must also involve clinical acumen and the recognition of temporal changes in physiology rather than just the degree of physiological derangement at a single time point. Given that older patients have a higher risk of mortality post-ICU, age may be a useful additional parameter to consider.[8] In some institutions, calls to specific teams, activated by track and trigger systems, have been associated with a significant reduction in cardiac arrests and unexpected deaths.[9] A UK based single centre trial demonstrated that the introduction of their ICU outreach team was associated with reduced hospital mortality,[10] although this is not a universal finding.[11] In addition to this role, some institutions review recent ICU discharges. Such services can aid continuity of care, create informal education opportunities, and improve links between wards and the ICU.[12 13]

AIRWAY: TRACHEOSTOMIES

The majority of prolonged ICU admissions occur in patients who require weaning from ventilatory support. Contributory factors to the aetiology of prolonged ventilatory failure include slowly recovering acute pulmonary pathology, chronic cardiorespiratory disease, an impaired level of consciousness, and the syndrome of ICU acquired weakness causing neuromuscular ventilatory failure and inadequate volitional secretion clearance. Many of these patients require tracheostomies for optimal delivery of ventilatory support, patient comfort, secretion management, and airway protection. The basic principles of tracheostomy are reviewed here.[14 15] Patients with a tracheostomy are at potential risk of sudden airway obstruction, and thus should only be discharged from the ICU to a ward environment which is managed by staff experienced in tracheostomy care.

Tracheostomy tubes are simply curved hollow cylinders projecting from the front of the neck. Typically, they enter the trachea between the second and third tracheal rings and project downward. They are usually secured with a cloth and Velcro tie attached to a flange, but may be sutured. A cuff at the lower end, when inflated, isolates the distal from the proximal trachea. Hence, speech is not possible with an inflated cuff, and any oxygen therapy should administered via the tracheostomy. When the cuff is inflated, intra-cuff pressures should be regularly monitored to ensure the pressure does not exceed 32 cm H_2O to prevent tracheal mucosal injury. A need for excessive cuff pressure suggests the tracheostomy is malpositioned or too small.

Ideally, tracheostomies used outside the ICU should have an inner cannula within the tracheostomy tube. These can (and should) be regularly removed and cleaned. Tracheostomies are generally sized according to the internal diameter of the outer tracheostomy tube; however, there is no standard sizing and the outer diameter may vary considerably across manufacturers. It is important to note that the use of an inner cannula will reduce the functional internal diameter, thereby increasing resistance. A vast number of different types and brands of tracheostomy are available to suit particular needs. For patients requiring prolonged tracheostomy, planned tube exchange is necessary at 4–6 weekly intervals.

In order to minimise tracheal mucosal desiccation, oxygen therapy should be humidified. Suctioning should only be performed when necessary and insertion of the suction catheter beyond the length of the tracheostomy tube should be avoided where possible. Post-ICU patients may be able to cough their secretions to the proximal opening of the tube, precluding the need for any distal suctioning. Stoma hygiene should be maintained by daily inspection of the site and cleansing with saline before the reapplication of a slimline, keyhole dressing to absorb local secretions and reduce pressure injury to surrounding tissue. The use of a 1–2% chlorhexidine mouthwash or gel, as part of regular oral hygiene, may prove beneficial.[16] Further information on tracheostomy care can be found at http://www.stgeorges.nhs.uk/trachindex.asp.

Most post-ICU patients will not require long term tracheostomies, and a process of decreasing dependence on the tracheostomy (weaning) towards ultimate removal (decannulation) should occur. Involvement of a dedicated multidisciplinary team may be useful in expediting this process.[17] Weaning involves patients spending increasing periods of time with the cuff deflated to allow them to adapt to managing their saliva and tracheobronchial secretions effectively. In most patients, deflating the cuff will facilitate translaryngeal gas flow and may be sufficient to allow speech. However, it is common practice to place a one way valve (commonly known as a speaking valve) at the end of the tube to enhance translaryngeal flow, aid in the production of speech, and improve the effectiveness of their cough. Should there be poor translaryngeal airflow with the cuff deflated, downsizing the tube or using a fenestrated tube may help. The ability of patients to tolerate occlusion of the

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tracheostomy subsequently, usually trialled with a decannulation cap for 1–4 h (with the cuff deflated), predicts that they will manage without their tracheostomy and that decannulation can take place.[18]

External bleeding around a tracheostomy is rare but blood stained secretions are common, are usually the result of endotracheal suction trauma, and are rarely problematic. Essential equipment that should be at the bedside of all tracheostomised patients includes operational suction and appropriately sized catheters, spare tracheostomy tubes of the same size and one size smaller, tracheal dilators, and an assisted ventilation circuit (Ambubag, Mapleson C or Waters circuit). The immediate management of tracheostomy emergencies (blockage, displacement and bleeding) is briefly described in box 2. Note that an important initial step is to enlist immediate expert help of clinicians with advanced airway skills (eg, anaesthetists, intensivists).

Tracheal stenosis is a rare complication of both prolonged endotracheal intubation and of tracheostomy. Stenosis may occur at the site of the cuff, though this is now rare with the advent of high volume low pressure cuffed tubes. More commonly it occurs following tracheostomy at the site of stoma creation. The incidence of symptomatic stenosis in survivors of critical illness who had had endotracheal intubation followed by percutaneous tracheostomy is reported to be 6%.[19] Screening and follow-up for tracheal stenosis can and should be performed at the bedside using a spirometer, looking for a significant reduction in inspiratory flow. Management may require specialist referral for endoscopic dilatation, resection of granulation tissue or stent placement.

RESPIRATORY DYSFUNCTION

Respiratory problems are common causes of prolonged ICU admission. Such patients are usually at high risk of further respiratory complications, specifically hospital acquired pneumonia and pulmonary embolism. Ongoing pulmonary, cardiac and/or neuromuscular pathologies, whether resolving acute, or chronic, often require active management.

Physiotherapy is key to minimising the risks of complications and restoring lost respiratory muscle function in post-ICU patients.[20] A graduated programme of increasing general mobility and activity, akin to cardiopulmonary rehabilitation programmes, is probably the best approach. Specific manoeuvres that may be useful for patients with ventilatory insufficiency include: supported upright sitting (to maximise functional residual capacity and minimise atelectasis), supported standing, and repetitive, active limb exercises.

In patients with viscous secretions a trial of regular nebulised hypertonic saline 4–7% (as opposed to 0.9%) and/or enteral carbocysteine[21] can be useful as mucolytic agents. Consideration should also be given to nebulised antibiotics in patients with respiratory tract colonisation, as opposed to focal pulmonary infection. In ventilated ICU patients with purulent secretions, such a strategy has been shown to decrease the incidence of pneumonia, facilitate ventilatory weaning and decrease the need for systemic antibiotics.[22]

Recovering acute and chronic respiratory conditions may also benefit from nocturnal (and sometimes intermittent daytime) ventilatory support delivered by mask or tracheostomy, sometimes termed non- or minimally-invasive ventilation. The decision to transfer a patient from the ICU to a ward environment capable of delivering intermittent support (where this is available) is usually dependent upon the patient's ability to breathe comfortably unsupported for minutes to hours and to be able to communicate/attract the attention of nursing staff. There should be a written prescription for the use of any ventilatory support, together with a documented weaning plan (or chronic therapy plan) as well as a clear plan relating to treatment escalation, ceiling and failure.[23]

Box 2 Tracheostomy emergencies (adapted from Mackenzie *et al*[15])

Blocked tracheostomy
- Call for Help
- Ask a cooperative patient to give a vigorous cough

If blockage not relieved:
- Remove the inner cannula
- Pass a suction catheter down the tracheostomy tube to remove secretions
- If inflated, deflate the cuff to enable breathing around the blocked tracheostomy tube and administer oxygen via the mouth.
- If not breathing provide assisted ventilation with bag and mask via mouth

In the face of persisting obstruction:
- Remove the tracheostomy tube and pass a large bore Yankeur suction catheter into the trachea to remove secretions.
- Proceed as per displaced tracheostomy

Displaced tracheostomy
- Call for help

If airway patent:
- Apply oxygen to both nose/mouth and tracheostomy
- Ask a clinician experienced in tracheostomy management to reposition/replace tube

If airway non-patent:
- Remove tracheostomy tube to allow breathing through the nose/mouth and stoma.
- Apply oxygen to both nose/mouth and tracheostomy stoma

If there is a well formed tract
- Consider re-insertion of tracheostomy tube

If there is not a well formed tract:
- Occlude stoma with gauze. Administer oxygen ± ventilatory support via the mouth
- Undertake endotracheal intubation if necessary
- If required, re-insert tracheostomy tube in a controlled environment

Bleeding from a tracheostomy
- Call for help
- Ensure airway patency and give oxygen via tracheostomy
- Inflate cuff to minimise contamination between spaces

If bleeding from around stoma site:
- Apply manual pressure
- Correct any clotting abnormalities

If bleeding persists:
- Try to establish whether the bleeding is from a site distal to the tube or from around the tube
- Apply closed circuit oxygen if available, inflate tube cuff to 40–50 cm H_2O and lie the patient left side down (or right side down if believed to be bleeding from the right bronchial tree) to minimise bilateral contamination
- Ensure large bore intravenous access
- Urgently cross-match and transfuse blood/clotting products as required
- Ask an experienced surgeon to urgently explore

CARDIAC DYSFUNCTION

Congestive cardiac failure was the most common comorbidity in a study of American patients who were difficult to wean from ventilatory support.[24] Following ICU discharge, cardiovascular disease is the second most common cause of death, after malignancy.[25] Cardiac dysfunction is usually due to pre-existing coronary artery disease. However, cardiac injury is probably under-recognised during ICU admission, in part because of analgesia, sedation, and patients' limited ability to communicate ischaemic symptoms. A recent study of patients admitted to a general ICU showed 36% had a focal myocardial injury (defined as troponin T >0.04 μg/l associated with dynamic ischaemic ECG changes) and a further 15% had an isolated troponin rise without ECG changes.[26] Sepsis,[27] renal failure, pulmonary embolism and other acute illnesses, independent of coronary artery disease, can all cause myocardial necrosis and hence an elevated serum troponin. A recent meta-analysis[28] reported that elevated serum troponins occur in 43% of ICU patients, and are associated with an increased risk of death and length of ICU and hospital stay. Given the prognostic relevance of troponin, many ICUs now measure it routinely. The basis for troponin elevation is not always entirely clear, but if seen in the context of ischaemic symptoms, dynamic ECG changes, new regional wall abnormalities, or haemodynamic instability, may warrant specialist cardiology review and intervention.

The need to refine the management of new, worsening, or chronic systolic and/or diastolic cardiac dysfunction is common in the post-ICU patient. New onset atrial fibrillation is another frequent complication of acute illness that may persist post-ICU discharge. This may necessitate appropriate investigations, refinement of rate/rhythm control therapy, institution of long term anticoagulation, planning of future attempts at DC cardioversion, or consideration of an ablation procedure.

RENAL DYSFUNCTION AND THE ONGOING NEED FOR RENAL REPLACEMENT THERAPY

Acute kidney injury (AKI) is a common complication of critical illness.[29] Patients with AKI have higher ICU and hospital mortality rates.[30] Those with severe AKI who are discharged to the ward from the ICU also spend nearly twice as long in hospital.[29] AKI may develop in any acutely unwell (including post-ICU) ward patients. A recent UK National Confidential Enquiry into Patient Outcomes and Death (NCEPOD) report[31] on AKI outlined the need for closer monitoring of serum electrolytes and physiology of acutely unwell patients together with a greater recognition of risk factors for the development of AKI. Common causes for renal injury include hypovolaemia, sepsis, hypotension, major surgery and nephrotoxic agents/drugs (including radiological contrast).[32] For patients who require renal replacement therapy on ICU, around 10% of those surviving to hospital discharge remain dialysis dependent.[33]

Reduced urine output can be an important indicator of an acutely unwell patient. Oliguria is defined as a urine output <0.5 ml/kg/h for 2 or more consecutive hours. It most commonly represents intravascular volume depletion, but may also result from relative hypotension or activation of the renin–angiotensin–aldosterone system. Rarely, relative oligura may be attributable to prior cessation of diuretics. If such a patient is fluid replete, it may be appropriate to restart chronic diuretic therapy.

Polyuria in the post-ICU patient typically reflects mobilisation of iatrogenic excess body water in the context of recovery from critical illness. Patients will have often received large amounts of intravenous fluids during the initial resuscitation and stabilisation phase of an acute illness on ICU, and this iatrogenic salt and water load is excreted slowly, as the patient recovers and 'finds their own balance'. If a patient appears to be euvolaemic then this is likely to be the case. However, polyuria may represent the recovery phase of acute renal injury, and result in intravascular depletion. If present, this warrants active monitoring and replacement therapy to prevent further renal injury and electrolyte abnormalities, in particular, hypokalaemia, hypomagnesaemia and hypophosphataemia (box 3).

Contrast induced nephropathy is an important cause of hospital acquired AKI. Post-ICU patients who have had an AKI, or have chronic renal impairment and need radiological investigations with intravenous contrast, are at particular risk. Primary prevention is good hydration, usually administered intravenously, before the procedure. The use of 1.26% sodium bicarbonate with the addition of N-acetylcysteine may be of additional benefit.[34]

MANAGEMENT OF NUTRITIONAL AND HYDRATION NEEDS

Basic daily water, electrolyte, energy and protein requirements are listed in table 1, but may vary depending on the patient's clinical condition. The most useful monitoring tool may be regular assessments of a patient's weight—daily for fluid balance, weekly for anabolic versus catabolic state.

Box 3 Causes and clinical features of hypomagnesaemia and hypophosphataemia

Causes of hypomagnesaemia

- Dietary deficiency
- Gastrointestinal losses
  - malabsorption
  - diarrhoea
  - pancreatitis
- Renal losses
  - from a polyuric phase of acute tubular necrosis
  - use of thiazide/loop diuretics or aminoglycosides.

Clinical features of hypomagnesaemia

- Cramps
- Tetany
- Vertigo
- Psychiatric disturbance
- Seizures
- Increased sensitivity to digoxin
- Cardiac dysrhythmias including torsades de pointes.

Causes of hypophosphataemia

- Malnutrition
- Refeeding syndrome (following administration of large amounts of carbohydrates to malnourished patients)
- Respiratory alkalosis
- Diabetic ketoacidosis
- Alcoholism
- Hyperparathyroidism
- Diuretic use

Clinical features of (severe) hypophosphataemia

- Profound muscle weakness
- Cardiac failure
- Seizures
- Respiratory failure[40 41]

Table 1 Daily water, electrolyte, energy and protein requirements (from Hinds and Watson[35] and Leonard[36])

	Daily requirement
Water	30 ml/kg
Sodium	1–2 mmol/kg
Potassium	0.7–1 mmol/kg
Magnesium	0.1 mmol/kg
Calcium	0.1 mmol/kg
Phosphorus	0.4 mmol/kg
Glucose	2–4 g/kg
Energy	25–35 kcal/kg/day
Protein	1–1.25 g/kg/day

A recent UK study of fluid prescribing habits of junior doctors[37] found that fluid balance was poorly managed on surgical wards. There was no correlation between the rate of fluid prescription or electrolyte content and serum electrolyte concentrations and fluid losses. Consequently some patients developed sodium and water overload or hypokalaemia associated tachyarrhythmias. Accurate measures of input and output are notoriously poor. Fluid prescription is all too often left to the most junior/inexperienced member of the medical team, who is given little if any guidance or feedback. We suggest that fluid prescribing should be better taught and considered a higher priority than current practice.

Providing adequate maintenance ± replacement regimens for water and electrolytes while avoiding iatrogenic overload and injury is essential. Recently published national guidelines for fluid resuscitation and replacement[38] advocate avoiding 0.9% sodium chloride (normal saline) as routine intravenous maintenance; 0.9% saline can cause iatrogenic hyperchloraemia, a consequent metabolic acidosis, and both renal and gastrointestinal dysfunction, although the importance of this phenomenon is debated.[39] In addition, the use of 5% dextrose and dextrose/saline (4%/0.18%) solutions can precipitate hyponatraemia and should be used intelligently. So-called balanced solutions, such as Hartmann's or Ringer's lactate, are more physiological and therefore are recommended as maintenance intravenous fluids. It should be stressed that enteral maintenance regimens should be instituted at the earliest opportunity and intravenous fluids avoided whenever possible. Electrolyte deficiencies, such as hypomagnesaemia and hypophosphataemia, are common in ICU patients, and many persist or recur after ICU discharge. Causes and clinical features are outlined in box 3. Other common laboratory abnormalities, including mild anaemia, thrombocytosis and hypoalbuminaemia, rarely require active management and should correct spontaneously over days to weeks.

Malnutrition/undernutrition is common in all hospitalised patients and associated with an increased length of hospital stay, increased infective complications,[42] and the development of pressure ulcers.[43] Optimisation of nutrition, both calories and micronutrients, is therefore a priority in post-ICU patients. Enteral nutrition stimulates intestinal mucosa, thereby minimising atrophy. This reduces bacterial translocation into the bloodstream, and protects against gastric stress ulceration. Without effective enteral nutrition, atrophic intestinal mucosal may additionally cause a protein (in particular albumin) losing enteropathy.[44] Where not contraindicated, enteral nutrition is invariably preferable to parenteral nutrition, which is beset by problems of vascular access associated infection, electrolyte imbalance, hyperglycaemia, hepatic dysfunction, and gut mucosal atrophy. Obstacles which may impair establishing effective enteral nutrition in the post-ICU patient are outlined in box 4.

Expert speech and language therapist assessment may be needed in cases of unsafe or inadequate swallow. Options to ensure continuing adequate enteral nutrition include the use of an adapted diet, such as thickened liquid feed, or continuous or supplemental (nocturnal) nasogastric or percutaneous enteral gastrostomy (PEG) feeding. Amotivation and depression, common psychological sequelae in post-ICU patients, or a change in the sensation of taste may cause volitional reduced oral intake. Dietician involvement and use of appropriate low volume high calorie supplements together with favourite foods, patient reassurance and addressing any psychological issues will help optimise enteral intake.

HYPERGLYCAEMIA

Hyperglycaemia, defined as blood glucose >8 mmol/l, is associated with an increase in infective complications and a worse outcome following many acute illnesses.[48–50] Until recently, tight glycaemic control (defined as maintenance of blood glucose between 4.4 and 6.1 mmol/l) was widely advocated in intensive care, following demonstration of reduced mortality in cardiac surgical intensive care patients.[51] However, this effect was not reproducible in medical ICU patients,[52] and a recent large international multicentre randomised trial demonstrated increased mortality in the tight glycaemic control arm.[53] While tight glycaemic control may not confer a survival benefit, we advocate that a reasonable approach in post-ICU patients is to target normoglycaemia actively—that is, blood glucose <8 mmol/l—although there is currently no evidence to support this and no specific recommendations as how best to achieve it.

PHYSICAL COMPLICATIONS OF CRITICAL ILLNESS

Prolonged immobility on the ICU is associated with loss of muscle bulk, frozen shoulders, finger and ankle joint contractures.[54] These complications may be minimised/prevented by early, regular passive physiotherapy and for contractures, regular splintage. Heterotropic ossification,[55] the formation of ectopic

Box 4 Obstacles to effective enteral nutrition in the post-ICU patient

Poor appetite
- Multifactorial but fatigue/fatiguability and apathy/delirium should be considered

Dysphagia
- Following prolonged intubation, at least 20% of patients experience dysphagia post-extubation[45]
- Is common and multifactorial in tracheostomised patients. It may be improved with regular/continuous exposure to translaryngeal gas flow (cuff deflation ± a speaking valve[46])
- Oral or oesophageal candidiasis, especially following prolonged antibiotic treatment. Prevention strategies include minimising duration of antibiotic treatment, strict oral hygiene, prophylactic chlorhexidine mouthwash, oral intake (encourages saliva) and topical antifungal agents

Delayed gastric emptying, dysmotility,[47] and small bowel ileus
- Causes abdominal distension and vomiting
- Often precipitated/exacerbated by electrolyte imbalance, intercurrent illness and drugs, eg, opiates
- Sometimes treatable by addressing precipitants

bone around large joints, has been described in 5% of survivors of critical illness,[54] and is manifested by pain, joint swelling, and significant functional disability. Exposure keratitis is prevalent in ICU patients,[56] despite optimal eye care regimens, and can occasionally lead to infectious keratitis, corneal perforation, and blindness. Survivors of critical illness may also experience alopecia, entrapment neuropathies, and prolonged pain at insertion sites of previous catheters and drains.[54]

ICU ACQUIRED WEAKNESS, PRESSURE ULCERS, AND REHABILITATION

ICU acquired weakness,[57] also known as critical illness neuromyopathy, is a sensorimotor axonopathy and primary myopathy with selective loss of myosin filaments. It occurs in 25–60% of patients receiving mechanical ventilation for more than 7 days. It is a major cause of acute (and sometimes chronic) functional disability. It is the most common cause of physical disability in survivors of the acute respiratory distress syndrome.[54] A poorly mobile post-ICU patient with ICU acquired weakness is at high risk of contractures, atelectasis, nosocomial pneumonia, dependent oedema, and pressure ulcers.

The aetiology, clinical manifestations and therapeutic strategies for ICU acquired weakness are outlined in box 5. The best therapeutic strategy for ICU acquired weakness is likely to be early targeted mobilisation and exercise on the ICU. Early passive mobilisation of limbs may reduce muscle atrophy, which occurs most rapidly in the first 2–3 weeks following ICU admission.[58] Initiation of an early mobilisation strategy, even in patients receiving ventilatory or other organ support, is associated with improved functional status and exercise capacity at ICU discharge.[59] Continued physiotherapy led mobilisation and rehabilitation remains important for improving exercise capacity, even after hospital discharge.[60]

Box 5 ICU acquired weakness

Postulated aetiological factors
- Ischaemic/inflammatory neuronal injury during systemic inflammatory response syndrome (SIRS)
- Immobility
- Voluntary muscle inactivity and atrophy
- Hyperglycaemia[61]
- *Possibly* corticosteroid induced muscle atrophy *and* use of 'high' cumulative doses of muscle relaxants (especially steroid based)

Manifestations
- Reduced or absent deep tendon reflexes
- Glove/stocking sensory loss
- Painful hyperaesthesia
- Muscle atrophy
- Foot drop
- Quadri/paraplegia.

Therapeutic strategies
- Early mobilisation and exercise programmes (starting on ICU)[59 62]
- Minimisation of sedation
- Optimal nutrition
- Replacement of deficiencies of potassium, magnesium, and phosphate (reversible causes of muscle weakness)

Immobility, incontinence, malnutrition, and altered consciousness predispose to pressure ulcer development. Hence, post-ICU patients are a vulnerable population. UK national guidelines exist outlining optimal management.[63] Regular assessment and accurate recording are essential. Management priorities include wound cleansing, prompt management of infection, debridement of necrotic tissue, and maintenance of a moist environment for wound healing using specialist dressings (hydrocolloids, foams, alginates) rather than simple gauze or dressing pads.[64] Use of pressure relieving support surfaces and regular repositioning should be routine. When necessary, specialists in tissue viability should be consulted and (plastic) surgical referral considered. A similar degree of vigilance is necessary for postoperative wounds of surgical patients. New pain, erythema, swelling, discharge, or wound dehiscence should prompt urgent surgical review.

Recent UK NICE guidelines[65] outline optimal rehabilitation after critical care. Before ICU discharge a comprehensive multidisciplinary assessment should evaluate physical and psychological problems including ICU acquired weakness, sensory, communication, cognitive and functional disabilities. Patients with, or at risk of, such problems should receive early tailored

Box 6 Delirium

Predisposing factors
- Advanced age
- Cognitive impairment

Precipitating factors
- Acute illness
- Admission/transfer to an unfamiliar environment
- Withdrawal syndrome (alcohol, cigarettes, usual medication)
- Polypharmacy
- Use of psychoactive medication (eg, morphine, benzodiazepines)
- Isolation/inactivity/boredom
- Lack of/poor quality sleep
- Lack of exposure to daylight

Therapeutic strategies
- Minimisation of precipitating factors
- Treatment of underlying illness and physiological/biochemical abnormality
- Regular patient orientation and reassurance
- Establishing a day night cycle
- Mental (and physical) occupation
- Assured, good quality sleep

Rescue therapy
- Benzodiazepines
  - — may be useful as acute sedatives but can be ineffective or even exacerbate agitation
  - — their respiratory depressant actions may make them inappropriate
  - — their regular use is associated with an increased risk of delirium and the development of a dependency syndrome
- Antipsychotics
  - — typical (eg, haloperidol, chlorpromazine) or atypical (eg, olanzapine, risperidone, quietapine)
  - — have a myriad of undesirable side effects and complications including an increased risk of cardiovascular death
  - — there is some evidence to suggest that atypical agents are as efficacious and better tolerated[69]

Table 2 Proposed multidisciplinary daily checklist for patients transferred to ward care after a prolonged/complex intensive care unit (ICU) stay

Item	Issue	Question
Analgesia	Recovering injuries/wounds + immobility may require regular and as required analgesia, especially to optimise rehabilitation sessions.	Adequate pain control? ☐
Cognition, mood and motivation	Common deficits, easily neglected.	Is there evidence of acute/recovering delirium? ☐ depression? ☐ apathy? ☐ *If yes*, make/refine management plan ☐
Adequate nutrition and hydration	Essential to recovery	Oral intake possible? ☐ adequate? ☐ Supplements needed ? ☐ available? ☐ used? ☐ Overnight NG/gastrostomy tube feeding? ☐ OR Continuous tube feeding? ☐ OR Parenteral nutrition? ☐
Glycaemic control	Persistent (even mild) hyperglycaemia associated with an increased risk of complications.	Blood sugar maintained at <8 mmol/l? ☐ *If not*, make/refine plan ☐
Bowels	Constipation common due to immobility, reduced oral hydration and opiates.	Are they working? ☐ *If not*, use stool softeners and mild aperients. ☐ *Consider* rectal exam/suppositories/enema.
Sleep	Often poor with detrimental consequences	Patient (nursing staff) reports adequate 'restorative' sleep? ☐ *If not*, make/refine plan ☐
Wounds/pressure areas	Easily overlooked and potential source of complications	Surgical wounds present? ☐ Pressure injuries present? ☐ Deteriorating? ☐ Infected? ☐ Need refinements to care or review by surgeon/plastic surgeon/ tissue viability nurse? ☐
Mobility	Essential to maximise recovery and minimise complications	Daily mobilisation plan? ☐ Daily/weekly goals? ☐ Special needs/equipment? ☐
Thromboembolic prophylaxis	High risk patients	Daily subcutaneous high dose, prophylactic, low molecular weight heparin? ☐ ± Appropriate stockings/mechanical pumps? ☐
Tubes	All medical tubes/devices can malfunction and cause complications, in particular infection. They are also uncomfortable. Hence they should be removed as soon as practical.	For each tube/device: Still needed? ☐ *If not*, remove ☐ *If yes*, working? ☐ secure? ☐ exit site normal? ☐ *If no to any*, fix? ☐ *OR* replace? ☐ *If replacing*, use simpler or 'long term' alternative? ☐
Drug chart	Drug errors and complications are common.	Legible and clear? ☐ Appropriate dose/frequency? ☐ Missed doses? ☐ Restart chronic drugs? ☐ Stop anything? ☐ (in particular, antibiotics) Convert iv to enteral? ☐ Therapeutic monitoring required? ☐
Investigations	Are blood tests/x-rays needed? What is the specific question to be answered? Unnecessary tests are counterproductive and expensive. If performed, have results been reviewed and acted upon?	Haemoglobin Na^+ K^+ Mg^{2+} PO_4^{3-}

rehabilitation plans. These should start in the ICU and assume an increasing portion of the therapeutic plan for many patients once they arrive on a ward. Rehabilitation progress should be regularly reviewed on the ward, and after hospital discharge. At hospital discharge, key information should be provided to the patient, their carers and their primary care physician on physical recovery, how to manage activities of daily living, and plans for follow-up.

DIFFUSE AND FOCAL BRAIN INJURY

Brain injury, either as a direct result of a focal pathology such as trauma, stroke or subarachnoid haemorrhage, or a diffuse process such as global hypoxia (eg, post-cardiac arrest) or metabolic/drug induced encephalopathy, frequently results in acute and often chronic disability.[66] Such problems are common in post-ICU patients, with perhaps the most challenging being those patients who exhibit aggressive or disinhibited behaviour (see delirium section below). Rehabilitation to minimise disability together with the active prevention of complications of global and focal neurological deficit are well established. The cohorting of such patients, as is now common following stroke, has many advantages and outcome benefits. The most common limiting factor, however, remains the relative scarcity of resources. Where cohorting is not possible, the development of a multidisciplinary 'brain injury' team[67] and the identification of a key therapist for each patient has demonstrable benefits.

PSYCHOLOGICAL PROBLEMS AND SLEEP

Delirium is a fluctuating decline in attention, awareness and mental clarity, with disorganised thinking. Features include reduced activity, a subdued state and apparent withdrawal (so-called hypoactive delirium), which may fluctuate with periods of restlessness, agitation, paranoia and combativeness (hyperactive delirium). It may be precipitated by acute illness and exacerbated by ICU interventions. It is common in the ICU population, in whom its incidence has been described to be 20–80%.[68] Delirium probably remains common, though underappreciated, following ICU discharge; not least as transfer to an unfamiliar ward is a known precipitating factor. Other precipitating factors and therapeutic strategies are outlined in box 6.

Anxiety occurs frequently both during and after ICU admission. Patients may experience distressing delusions, hallucinations and nightmares after ICU discharge. In a recent

prevalence study, post-traumatic stress disorder (PTSD) was detected in 13% of post-ICU patients 1 month following discharge, although it may occur in additional patients at a later stage.[70] PTSD is an anxiety disorder which develops after exposure to a traumatic event that threatened or caused grave physical harm. Patients experience intrusive recollections of the event, often in the form of flashbacks or nightmares, which may be triggered by various stimuli they associate with the initial traumatic event. Psychologist led treatment approaches include desensitisation techniques, cognitive behavioural therapy, anxiolytics, and antidepressants.

Cognitive impairment and depression are common in post-ICU patients, each being present 6 months post-discharge in around a third of patients who were mechanically ventilated.[71] Recent NICE guidelines on rehabilitation from critical care[65 72] advocate identification of patients at risk of anxiety, PTSD, depression and cognitive impairment before hospital discharge. Existing care models for these disorders[73–75] should be followed. Finally, it should not be forgotten that considerable psychological stress is also experienced by many family members of post-ICU patients, who may also require appropriate interventions.

Persistent sleep disturbance is common in post-ICU patients, being present in up to 40% of patients 6 months following discharge,[76] and being more common in patients with concurrent disease. Post-ICU patients experience loss of circadian rhythm, difficulty falling asleep, poor sleep quality, and sleep for shorter periods than normal individuals. This impacts adversely on quality of life and general health. Therapeutic strategies to improve sleep include noise reduction, relaxation training, cognitive behavioural therapy, and sleep hygiene. Sleep hygiene[77] describes the process of optimising the sleep environment and includes: maintenance of a regular sleep-wake cycle with maximal daytime daylight exposure; avoidance of light and disruptive noises at night; minimisation of wakeful time spent in bed; avoidance of caffeine, nicotine and alcohol; avoidance of eating within 3 h of bedtime; and undertaking a relaxation routine before bed. The use of eye shades and foam ear plugs may also be beneficial.[78] Pharmacological treatment, in particular melatonin, has an undefined role although benzodiazepines are probably best avoided.

CARE BUNDLES AND CHECKLISTS

Care bundles are widely utilised on ICUs. A care bundle is a group of interventions employed to optimise treatment or minimise complication rates.[79 80] Bundling interventions aims to ensure that they are implemented uniformly and reliably. Complementary to the implementation of bundles, checklists are useful in minimising errors, especially those of omission. A good ICU example, known by its pneumonic, 'FASTHUG',[81] prompts clinicians to focus on **f**eeding, **a**nalgesia, **s**edation, **t**hromboembolic prophylaxis, **h**ead-of-the bed elevation, stress **u**lcer prophylaxis, and **g**lycaemic control. Evolution of a care bundle for post-ICU patients may be valuable though none currently exist. We propose an adapted form of an ICU checklist (care bundle) that might be of benefit in the management of the post-ICU patients (table 2).

PHARMACOLOGICAL CONSIDERATIONS

Attention to drug prescribing for a complicated post-ICU patient is imperative. Clinical pharmacists are invaluable in highlighting prescription errors and omissions,[82] and as a source of advice to clinicians to aid safe and appropriate prescribing. Levels of some antibiotics or anticonvulsants need serum monitoring and dose adjustments if patients have acute, recovering or chronic hepatic or renal dysfunction. Clinicians should be aware of nephrotoxic and hepatotoxic effects of commonly used medications and, when indicated, monitor renal and liver biochemistry. Consideration should be given to the re-introduction of any medications, such as antihypertensives, which may have been withheld on the ICU, and to review and rationalise any polypharmacy. The indication for, and intended duration of, any antimicrobial therapy should be clearly documented. Minimisation of antimicrobial exposure is crucial to minimising the acquisition of multiply resistant pathogens and *Clostridium difficile*.

Appropriate specific formulations of drugs are required for patients who are nil by mouth and receiving medications via nasogastric or PEG routes. Crushing of enteric coated medicines alters bioavailability, while crushing of modified release preparations can produce dangerous peaks and troughs in drug concentrations. Crushing some medicines to administer down nasogastric tubes can also cause mechanical blockage. Intravenous insulin infusions may need conversion to subcutaneous insulin regimens. The subcutaneous insulin requirement over 24 h should be 60–70% of the intravenous insulin requirement over the last 24 h.[83] Additional specialist diabetic prescribing advice may be needed during recovery and with step changes in diet. Before discharge from hospital, consideration should be also given to limiting medication to the minimum necessary to optimise compliance.

DISCHARGE FROM HOSPITAL

A prolonged ICU admission followed by a prolonged ward stay is likely to result in a 'complex' hospital discharge. As with the ICU to ward transfer, the hospital discharge will need to be considered and planned for at the earliest stage. This may need to involve a number of outpatient services as well as external resources from rehabilitation services or domiciliary palliative care. Specific ICU follow-up services are valued by many former ICU patients for their contribution to physical, emotional and psychological recovery.[84] However, currently such services only exist in a minority of centres,[85] and have yet to be demonstrated to be efficacious or cost-effective.[86]

Main messages

- Discharge of ICU patients after prolonged ICU admission requires meticulous planning, and a comprehensive multidisciplinary verbal and written handover to the receiving ward team.
- 'Track and trigger' systems such as the Early Warning Score should be used to monitor patients discharged from ICU. Unexpected physiological deterioration should trigger a response including acute resuscitative measures, and consideration of the need for ICU re-admission
- Patients who have received prolonged organ support in the ICU may be affected by ventilatory insufficiency, cardiac dysfunction, kidney injury, nutritional deficiency, ICU acquired weakness, and brain injury. They also frequently experience physical disability, and psychosocial problems including delirium, anxiety, depression, post-traumatic stress disorder, cognitive dysfunction and disturbed sleep.
- Structured rehabilitation programmes of post-ICU patients, tailored to individual physical and psychosocial needs, should be commenced on the ICU and continued through to and beyond hospital discharge.

Current research questions

- How effective are critical care outreach teams in reducing morbidity, mortality, and ICU re-admission in patients discharged from ICU?
- Is targeting normoglycaemia beneficial in acutely unwell and recovering patients?
- What is the pathophysiological basis for the association of elevated troponin with increased mortality and length of stay in ICU patients? How should we respond to elevated troponin that is unrelated to myocardial infarction in ICU patients?
- What is the pathogenic basis for ICU acquired weakness? What strategies on the ICU can reduce its development? What therapeutic strategies (physical and pharmacologic) might be useful in its treatment?
- How can psychological complications of critical illness be prevented or best treated?
- Are care bundles/daily (or weekly) goal sheets useful in managing patients discharged from ICU?
- What are the benefits of post-ICU follow-up clinics?

CONCLUSION

Patients discharged from ICU after critical illness exhibit a variety of generic problems. Structured and tailored rehabilitation programmes for such patients should commence before ICU discharge and continue through to, and beyond, hospital discharge. Comparatively lower staffing levels and reduced availability of resources on medical or surgical wards, compared to the ICU, can make looking after such patients challenging. An appreciation of the common complications/problems, and strategies for dealing with them, should help clinical staff in providing the best possible ward based care of complex and often difficult-to-manage post-ICU patients.

Key references

- **NICE clinical guideline 50**. National Institute for Health and Clinical Excellence. Acutely ill patients in hospital: Recognition and response to acute illness of adults in hospitals. 2007. http://www.nice.org.uk/CG050.
- **Mackenzie S**, Murphy P, Bodenham A, *et al*. Standards for the care of adult patients with a temporary tracheostomy. Intensive Care Society Standards and Guidelines. 2008. http://www.ics.ac.uk/intensive_care_professional/standards_and_guidelines/care_of_the_adult_patient_with_a_temporary_tracheostomy_2008.
- **Navaneethan SD**, Singh S, Appasamy S, *et al*. Sodium bicarbonate therapy for prevention of contrast-induced nephropathy: a systematic review and meta-analysis. *Am J Kidney Dis* 2009 April;**53**(4):617–27.
- **Powell-Tuck J**, Gosling P, Lobo DN, *et al*. NHS Evidence. British consensus guidelines on intravenous fluid therapy for adult surgical patients. Intensive Care Society 2008. http://www.ics.ac.uk/intensive_care_professional/standards_and_guidelines/british_consensus_guidelines_on_intravenous_fluid_therapy_for_adult_surgical_patients__giftasup__2008.
- **NICE clinical guideline 83**. National Institute for Health and Clinical Excellence. Rehabilitation after critical illness. London. 2009. http://www.nice.org.uk/CG83.

MULTIPLE CHOICE QUESTIONS (TRUE (T)/FALSE (F); ANSWERS AFTER THE REFERENCES)

1. Tracheostomy:

A. A tracheostomised post-ICU patient can be safely managed on any medical or surgical ward
B. Cuff pressures >32 cm H_2O can cause tracheal mucosal injury and should be avoided where possible
C. Cuff inflation is necessary to facilitate speech
D. Regular suctioning to the level of the carina should be undertaken to reduce secretion load
E. In the case of a blocked tracheostomy, both inner and outer cannulae should be immediately removed

2. Cardiac dysfunction:

A. Congestive cardiac failure is a common reason for prolonged need for ventilatory support on the ICU
B. Cardiac disease is one of the most common causes of death in patients discharged from ICU
C. Elevated troponins are common in ICU patients, but have no prognostic relevance
D. Elevated troponins in ICU patients are invariably due to coronary artery disease
E. Coronary angiography should be undertaken in every post-ICU patient who has had an elevated troponin

3. Renal dysfunction and electrolyte imbalance:

A. Polyuria is common in the recovery phase of critical illness
B. Oliguria is defined as urine output <1 ml/kg/h for 2 consecutive hours
C. Use of intravenous 0.9% saline as maintenance fluid can precipitate iatrogenic hyperchloraemic metabolic acidosis
D. Use of Hartmann's solution as maintenance intravenous fluid can precipitate iatrogenic hyperchloraemic metabolic acidosis
E. Use of intravenous 5% dextrose can precipitate hyponatraemia

4. ICU acquired weakness:

A. Is a purely motor neuropathy
B. Is predominantly associated with hyper-reflexia
C. Systemic inflammation, the use of corticosteroids and neuromuscular blocking agents are implicated in its development
D. Rapidly resolves once patients are weaned from organ support on ICU
E. Bed rest is the main therapeutic strategy

5. Delirium:

A. Advanced age and cognitive impairment are predisposing factors
B. Transfer to an unfamiliar ward is a precipitating factor
C. Withdrawal of alcohol or cigarettes are other common precipitating factors
D. Is characterised by a decline in awareness with disorganised thinking
E. Invariably needs to be treated with benzodiazepines or antipsychotics

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REFERENCES

1. **Daly K,** Beale R, Chang RW. Reduction in mortality after inappropriate early discharge from intensive care unit: logistic regression triage model. *BMJ* 2001;**322**:1274—6.
2. **Goldfrad C,** Rowan K. Consequences of discharges from intensive care at night. *Lancet* 2000;**355**:1138—42.
3. **Frost SA,** Alexandrou E, Bogdanovski T, *et al*. Severity of illness and risk of readmission to intensive care: a meta-analysis. *Resuscitation* 2009;**80**:505—10.
4. **Utzolino S,** Kaffarnik M, Keck T, *et al*. Unplanned discharges from a surgical intensive care unit: Readmissions and mortality. *J Crit Care*. Published Online First 2009. doi:10.1016/j.jcrc.2009.09.009.
5. **NICE clinical guideline 50.** National Institute for Health and Clinical Excellence. Acutely ill patients in hospital: recognition and response to acute illness of adults in hospitals. 2007. http://www.nice.org.uk/CG050.
6. **Morgan RM,** Williams F, Wright MM. An early warning scoring system for detecting developing critical illness. *Clin Intensive Care* 1997;**8**:100.
7. **Smith GB,** Prytherch DR, Schmidt PE, *et al*. A review, and performance evaluation, of single-parameter "track and trigger" systems. *Resuscitation* 2008;**79**:11—21.
8. **Smith GB,** Prytherch DR, Schmidt PE, *et al*. Should age be included as a component of track and trigger systems used to identify sick adult patients? *Resuscitation* 2008;**78**:109—15.
9. **Chen J,** Bellomo R, Flabouris A, *et al*. The relationship between early emergency team calls and serious adverse events. *Crit Care Med* 2009;**37**:148—53.
10. **Priestley G,** Watson W, Rashidian A, *et al*. Introducing critical care outreach: a ward-randomised trial of phased introduction in a general hospital. *Intensive Care Med* 2004;**30**:1398—404.
11. **McGaughey J,** Alderdice F, Fowler R, *et al*. (EWS) for the prevention of intensive care admission and death of critically ill adult patients on general hospital wards. *Cochrane Database Syst Rev* 2007;(3):CD005529.
12. **Ball C,** Kirkby M, Williams S. Effect of the critical care outreach team on patient survival to discharge from hospital and readmission to critical care: non-randomised population based study. *BMJ* 2003;**327**:1014.
13. **Endacott R,** Chaboyer W, Edington J, *et al*. Impact of an ICU Liaison Nurse Service on major adverse events in patients recently discharged from ICU. *Resuscitation* 2010;**81**:198—201.
14. **De LP,** Bedert L, Delcroix M, *et al*. Tracheotomy: clinical review and guidelines. *Eur J Cardiothorac Surg* 2007;**32**:412—21.
15. **Mackenzie S,** Murphy P, Bodenham A, *et al*. Standards for the care of adult patients with a temporary tracheostomy. *Intensive Care Soc Stand Guidel* 2008. http://www.ics.ac.uk/intensive_care_professional/standards_and_guidelines/care_of_the_adult_patient_with_a_temporary_tracheostomy_2008.
16. **National Institute of Health and Clinical Excellence, National Patient Safety Agency.** Technical patient safety solutions for ventilator-associated pneumonia in adults. *NICE/NPSA/2008/PSG002*;2008. http://www.nice.org.uk/nicemedia/pdf/PSG002VAPGuidance.pdf.
17. **Garrubba M,** Turner T, Grieveson C. Multidisciplinary care for tracheostomy patients: a systematic review. *Crit Care* 2009;**13**:R177.
18. **Rumbak MJ,** Graves AE, Scott MP, *et al*. Tracheostomy tube occlusion protocol predicts significant tracheal obstruction to air flow in patients requiring prolonged mechanical ventilation. *Crit Care Med* 1997;**25**:413—17.
19. **Norwood S,** Vallina VL, Short K, *et al*. Incidence of tracheal stenosis and other late complications after percutaneous tracheostomy. *Ann Surg* 2000;**232**:233—41.
20. **Gosselink R,** Bott J, Johnson M, *et al*. Physiotherapy for adult patients with critical illness: recommendations of the European Respiratory Society and European Society of Intensive Care Medicine Task Force on Physiotherapy for Critically Ill Patients. *Intensive Care Med* 2008;**34**:1188—99.
21. **Braga PC,** Allegra L, Rampoldi C, *et al*. Long-lasting effects on rheology and clearance of bronchial mucus after short-term administration of high doses of carbocysteine-lysine to patients with chronic bronchitis. *Respiration* 1990;**57**:353—8.
22. **Palmer LB,** Smaldone GC, Chen JJ, *et al*. Aerosolized antibiotics and ventilator-associated tracheobronchitis in the intensive care unit. *Crit Care Med* 2008;**36**:2008—13.
23. **Roberts CM,** Brown JL, Reinhardt AK, *et al*. Non-invasive ventilation in chronic obstructive pulmonary disease: management of acute type 2 respiratory failure. *Clin Med* 2008;**8**:517—21.
24. **Scheinhorn DJ,** Hassenpflug MS, Votto JJ, *et al*. Post-ICU mechanical ventilation at 23 long-term care hospitals: a multicenter outcomes study. *Chest* 2007;**131**:85—93.
25. **Mayr VD,** Dunser MW, Greil V, *et al*. Causes of death and determinants of outcome in critically ill patients. *Crit Care* 2006;**10**:R154.
26. **Lim W,** Holinski P, Devereaux PJ, *et al*. Detecting myocardial infarction in critical illness using screening troponin measurements and ECG recordings. *Crit Care* 2008;**12**:R36.
27. **Rudiger A,** Singer M. Mechanisms of sepsis-induced cardiac dysfunction. *Crit Care Med* 2007;**35**:1599—608.
28. **Lim W,** Qushmaq I, Devereaux PJ, *et al*. Elevated cardiac troponin measurements in critically ill patients. *Arch Intern Med* 2006;**166**:2446—54.
29. **Kolhe NV,** Stevens PE, Crowe AV, *et al*. Case mix, outcome and activity for patients with severe acute kidney injury during the first 24 hours after admission to an adult, general critical care unit: application of predictive models from a secondary analysis of the ICNARC Case Mix Programme Database. *Crit Care* 2008;**12**(Suppl 1):S2.
30. **Harrison DA,** Brady AR, Rowan K. Case mix, outcome and length of stay for admissions to adult, general critical care units in England, Wales and Northern Ireland: the Intensive Care National Audit & Research Centre Case Mix Programme Database. *Crit Care* 2004;**8**:R99—111.
31. **National Confidential Enquiry into Patient Outcome and Death.** Adding Insult to Injury. A review of the care of patients who died in hospital with a primary diagnosis of acute kidney injury (acute renal failure). 2009. http://www.ncepod.org.uk/2009report1/Downloads/AKI_report.pdf.
32. **Prescott GJ,** Metcalfe W, Baharani J, *et al*. A prospective national study of acute renal failure treated with RRT: incidence, aetiology and outcomes. *Nephrol Dial Transplant* 2007;**22**:2513—19.
33. **Wald R,** Deshpande R, Bell CM, *et al*. Survival to discharge among patients treated with continuous renal replacement therapy. *Hemodial Int* 2006;**10**:82—7.
34. **Navaneethan SD,** Singh S, Appasamy S, *et al*. Sodium bicarbonate therapy for prevention of contrast-induced nephropathy: a systematic review and meta-analysis. *Am J Kidney Dis* 2009;**53**:617—27.
35. **Hinds CJ,** Watson D. *General aspects of managing critically ill patients. Intensive care, a concise textbook*. 3rd edn. London, UK: Saunders Elsevier, 2008:291—327.
36. **Leonard R.** Enteral and parenteral nutrition. In: Bersten AD, Soni N, Oh TE, eds. *Oh's intensive care manual*. 5th edn. Oxford, UK: Butterworth Heinmann, 2007:903—12.
37. **Walsh SR,** Walsh CJ. Intravenous fluid-associated morbidity in postoperative patients. *Ann R Coll Surg Engl* 2005;**87**:126—30.
38. **Powell-Tuck J,** Gosling P, Lobo DN, *et al*. NHS Evidence. British consensus guidelines on intravenous fluid therapy for adult surgical patients. *Intensive Care Soc* 2008. http://www.ics.ac.uk/intensive_care_professional/standards_and_guidelines/british_consensus_guidelines_on_intravenous_fluid_therapy_for_adult_surgical_patients__giftasup__2008.
39. **Handy JM,** Soni N. Physiological effects of hyperchloraemia and acidosis. *Br J Anaesth* 2008;**101**:141—50.
40. **Oud L.** Transient hypoxic respiratory failure in a patient with severe hypophosphatemia. *Med Sci Monit* 2009;**15**:CS49—53.
41. **Varsano S,** Shapiro M, Taragan R, *et al*. Hypophosphatemia as a reversible cause of refractory ventilatory failure. *Crit Care Med* 1983;**11**:908—9.
42. **Deane A,** Chapman MJ, Fraser RJ, *et al*. Mechanisms underlying feed intolerance in the critically ill: implications for treatment. *World J Gastroenterol* 2007;**13**:3909—17.
43. **Banks M,** Bauer J, Graves N, *et al*. Malnutrition and pressure ulcer risk in adults in Australian health care facilities. *Nutrition.* Published Online First 2009. doi:10.1016/j.nut.2009.09.024.
44. **Redelmeier D.** New thinking about postoperative hypoalbuminemia: a hypothesis of occult protein-losing enteropathy. *Open Med* 2009;**3**:E215—19. http://www.openmedicine.ca/article/view/343.
45. **Barker J,** Martino R, Reichardt B, *et al*. Incidence and impact of dysphagia in patients receiving prolonged endotracheal intubation after cardiac surgery. *Can J Surg* 2009;**52**:119—24.
46. **Dettelbach MA,** Gross RD, Mahlmann J, *et al*. Effect of the Passy-Muir Valve on aspiration in patients with tracheostomy. *Head Neck* 1995;**17**:297—302.
47. **Quigley EM.** Critical care dysmotility: abnormal foregut motor function in the ICU/ITU patient. *Gut* 2005;**54**:1351—2.
48. **Brennan AL,** Gyi KM, Wood DM, *et al*. Airway glucose concentrations and effect on growth of respiratory pathogens in cystic fibrosis. *J Cyst Fibros* 2007;**6**:101—9.
49. **McAlister FA,** Majumdar SR, Blitz S, *et al*. The relation between hyperglycemia and outcomes in 2,471 patients admitted to the hospital with community-acquired pneumonia. *Diabetes Care* 2005;**28**:810—15.
50. **Chakrabarti B,** Angus RM, Agarwal S, *et al*. Hyperglycaemia as a predictor of outcome during non-invasive ventilation in decompensated COPD. *Thorax* 2009;**64**:857—62.
51. **Van den Berghe G,** Wouters P, Weekers F, *et al*. Intensive insulin therapy in the critically ill patients. *N Engl J Med* 2001;**345**:1359—67.
52. **Van den Berghe G,** Wilmer A, Hermans G, *et al*. Intensive insulin therapy in the medical ICU. *N Engl J Med* 2006;**354**:449—61.
53. **Finfer S,** Chittock DR, Su SY, *et al*. Intensive versus conventional glucose control in critically ill patients. *N Engl J Med* 2009;**360**:1283—97.
54. **Herridge MS,** Cheung AM, Tansey CM, *et al*. One-year outcomes in survivors of the acute respiratory distress syndrome. *N Engl J Med* 2003;**348**:683—93.
55. **Hudson SJ,** Brett SJ. Heterotopic ossification—a long-term consequence of prolonged immobility. *Crit Care* 2006;**10**:174.
56. **McHugh J,** Alexander P, Kalhoro A, *et al*. Screening for ocular surface disease in the intensive care unit. *Eye (Lond)* 2008;**22**:1465—8.
57. **Schweickert WD,** Hall J. ICU-acquired weakness. *Chest* 2007;**131**:1541—9.
58. **Gruther W,** Benesch T, Zorn C, *et al*. Muscle wasting in intensive care patients: ultrasound observation of the M. quadriceps femoris muscle layer. *J Rehabil Med* 2008;**40**:185—9.
59. **Schweickert WD,** Pohlman MC, Pohlman AS, *et al*. Early physical and occupational therapy in mechanically ventilated, critically ill patients: a randomised controlled trial. *Lancet* 2009;**373**:1874—82.
60. **McWilliams DJ,** Atkinson D, Carter A, *et al*. Feasibility and impact of a structured, exercise-based rehabilitation programme for intensive care survivors. *Physiother Theory Pract* 2009;**25**:566—71.
61. **Hermans G,** De JB, Bruyninckx F, *et al*. Interventions for preventing critical illness polyneuropathy and critical illness myopathy. *Cochrane Database Syst Rev* 2009;(1):CD006832.
62. **Perme C,** Chandrashekar R. Early mobility and walking program for patients in intensive care units: creating a standard of care. *Am J Crit Care* 2009;**18**:212—21.
63. **NICE clinical guideline 29.** National Institute for Health and Clinical Excellence. The prevention and treatment of pressure ulcers. 2005. http:\\www.nice.org.uk/CG029.

64. **Bluestein D,** Javaheri A. Pressure ulcers: prevention, evaluation, and management. *Am Fam Physician* 2008;**78**:1186—94.
65. **NICE clinical guideline 83.** National Institute for Health and Clinical Excellence. Rehabilitation after critical illness. 2009. http:\\www.nice.org.uk/CG83.
66. **Hopkins RO,** Jackson JC. Long-term neurocognitive function after critical illness. *Chest* 2006;**130**:869—78.
67. **Turner-Stokes L,** Disler PB, Nair A, *et al*. Multi-disciplinary rehabilitation for acquired brain injury in adults of working age. *Cochrane Database Syst Rev* 2005;(3): CD004170.
68. **Girard TD,** Pandharipande PP, Ely EW. Delirium in the intensive care unit. *Crit Care* 2008;**12**(Suppl 3):S3.
69. **Rea RS,** Battistone S, Fong JJ, *et al*. Atypical antipsychotics versus haloperidol for treatment of delirium in acutely ill patients. *Pharmacotherapy* 2007;**27**:588—94.
70. **Wallen K,** Chaboyer W, Thalib L, *et al*. Symptoms of acute posttraumatic stress disorder after intensive care. *Am J Crit Care* 2008;**17**:534—43.
71. **Jackson JC,** Hart RP, Gordon SM, *et al*. Six-month neuropsychological outcome of medical intensive care unit patients. *Crit Care Med* 2003;**31**:1226—34.
72. **Tan T,** Brett SJ, Stokes T. Rehabilitation after critical illness: summary of NICE guidance. *BMJ* 2009;**338**:b822.
73. **NICE clinical guideline 26.** National Institute for Health and Clinical Excellence. Post-traumatic stress disorder (PTSD): the management of PTSD in adults and children in primary and secondary care. 2005. http://www.nice.org.uk/CG26.
74. **NICE clinical guideline 22.** National Institute for Health and Clinical Excellence. Anxiety: management of anxiety (panic disorder, with or without agoraphobia, and generalised anxiety disorder) in adults in primary, secondary and community care. 2007. http://www.nice.org.uk/CG22.
75. **NICE clinical guideline 23.** National Institute for Health and Clinical Excellence. Depression: management of depression in primary and secondary care. 2007. http://www.nice.org.uk/CG23.
76. **Orwelius L,** Nordlund A, Nordlund P, *et al*. Prevalence of sleep disturbances and long-term reduced health-related quality of life after critical care: a prospective multicenter cohort study. *Crit Care* 2008;**12**:R97.
77. **Neubauer DN.** Current and new thinking in the management of comorbid insomnia. *Am J Manag Care* 2009;**15**(Suppl):S24—32.
78. **Richardson A,** Allsop M, Coghill E, *et al*. Earplugs and eye masks: do they improve critical care patients' sleep? *Nurs Crit Care* 2007;**12**:278—86.
79. **Galpern D,** Guerrero A, Tu A, *et al*. Effectiveness of a central line bundle campaign on line-associated infections in the intensive care unit. *Surgery* 2008;**144**:492—5.
80. **Papadimos TJ,** Hensley SJ, Duggan JM, *et al*. Implementation of the "FASTHUG" concept decreases the incidence of ventilator-associated pneumonia in a surgical intensive care unit. *Patient Saf Surg* 2008;**2**:3.
81. **Vincent JL.** Give your patient a fast hug (at least) once a day. *Crit Care Med* 2005;**33**:1225—9.
82. **Ryan C,** O'Mahony D, Byrne S. Application of STOPP and START criteria: interrater reliability among pharmacists. *Ann Pharmacother* 2009;**43**:1239—44.
83. **Weant KA,** Ladha A. Conversion from continuous insulin infusions to subcutaneous insulin in critically ill patients. *Ann Pharmacother* 2009;**43**:629—34.
84. **Prinjha S,** Field K, Rowan K. What patients think about ICU follow-up services: a qualitative study. *Crit Care* 2009;**13**:R46.
85. **Griffiths JA,** Barber VS, Cuthbertson BH, *et al*. A national survey of intensive care follow-up clinics. *Anaesthesia* 2006;**61**:950—5.
86. **Cuthbertson BH,** Rattray J, Campbell MK, *et al*. The PRaCTICaL study of nurse led, intensive care follow-up programmes for improving long term outcomes from critical illness: a pragmatic randomised controlled trial. *BMJ* 2009;**339**:b3723.

ANSWERS

1. A (F); B (T); C (F); D (F); E (F)
2. A (T); B (T); C (F); D (F); E (F)
3. A (T); B (F); C (T); D (F); E (T)
4. A (F); B (F); C (T); D (F); E (F)
5. A (T); B (T); C (T); D (T); E (F)