Intrahospital Transport

CONSIDERATIONS FOR CRITICALLY ILL TRAUMA PATIENTS

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In the critical care setting, gains in patient well-being and hemodynamic stability are made with great effort on the part of medical and nursing staff and patient. Too often, the ground gained by resuscitation is lost to a variety of avoidable destabilizing influences during transport of critically ill patients out of the trauma intensive care unit (ICU):

- Inadequate temperature control with consequent hyperthermia or hypothermia
- Accidental derangement of precisely regulated infusions of vasoactive drugs
- Loss of ventilatory control settings that have been carefully matched to the needs of a particular patient
- Disturbances of fractures in close proximity to neurovascular structures, resulting in neurologic deficits or regional ischemia

All of these elements combine to increase patient morbidity and mortality through the common pathways of poor tissue perfusion and hypoxia.

To avoid transport-related morbidity and mortality, one must know what complications may occur during transport of patients outside the trauma ICU, how to anticipate and avoid those complications, and how to maximize the risk-benefit ratio of transports so that patients are exposed to the hazards of leaving the ICU environment for only justifiable causes.

A patient may need to leave the trauma ICU for surgery, diagnostic or therapeutic radiologic procedures, treatment in another specialty care area (for example, hyperbaric oxygen therapy), or an interhospital transfer. Support and monitoring begun in the ICU must be continued at the same levels during the transport. Support must be maintained to avoid deterioration in organ system function; monitoring must be continued so that such deteriorations, whether from changes in support or from worsening of intrinsic disease, are apparent to trained observers accompanying the patient. Simply monitoring vital signs during transport does not ensure the safety and continued stability of an ICU patient; electronic monitoring must be continued as well.

The focus of transport support and monitoring is to preserve the homeostasis achieved in the ICU; new therapies are not begun during patient transport except as needed to correct acute events occurring during that time. Neither should a patient be moved from the ICU during or soon after changes in ventilatory or hemodynamic support except as urgent circumstances dictate. In that event, the patient should be moved from the ICU only for diagnostic procedures that will guide major treatment decisions or for therapeutic procedures essential to recovery.

Finally, a cogent reason for complex electronic monitoring of patients during transport is to ensure the continued function of monitoring systems themselves. Vascular access lines, intracranial pressure monitors, and other invasive devices provide useful information but also expose the patient to numerous hazards, some fatal. Continual monitoring of such devices lessens their risks to the patient and, one hopes, increases their information-gathering potential.

The most critical systems to monitor and support during patient transport are respiratory, cardiovascular, neurologic, metabolic, and renal (see box below). Common complications in each of these systems are discussed (see box, page 381), together with some ways to antici-

Systemic parameters to monitor during transport

Respiratory
- Respiratory or ventilator rate
- Skin color, breath sounds
- Ventilator tidal volume (inspired and expired)
- Delivered PEEP, peak and mean airway pressures
- Arterial and venous O_2, CO_2 partial pressures
- Pulse oximetry readings of O_2 saturation

Cardiovascular
- Heart sounds, skin color, capillary refill time
- Blood pressure (mean, systolic and diastolic, pulse)
- Cardiac rate and rhythm; ischemic changes on ECG
- Central venous pressure, mean pulmonary arterial pressure, and mean arterial pressure by vascular catheters
- Pacemaker function

Neurologic
- Level of consciousness
- Cranial nerve function
- Extremity function, deep tendon reflexes
- Intracranial pressure
- Occurrence of seizures

Metabolic
- Acid-base balance
- Serum glucose, electrolytes

Renal
- Urine output
- Serum creatinine, BUN
- Output of continuous arteriovenous hemofiltration dialysis systems

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pate complications and deal with them should they arise.

Respiratory System

Numerous respiratory problems may occur during transport of critically ill patients outside the trauma ICU. These problems can be divided into difficulties with the patient's airway (natural or artificial), problems with mechanical ventilation, worsening of thoracic or parenchymal disease process (either spontaneously or as a consequence of transport), and loss of monitoring capability with which to assess adequacy of ventilation.

Airway

Airway problems are fairly common during transports. A patient not already receiving mechanical ventilation may suffer airway compromise as a result of neurologic deterioration (sedation, analgesia, spontaneous worsening of central nervous system [CNS] injury) and require emergent securing of the airway by endotracheal intubation or cricothyroidotomy while out of the critical care area. Expanding neck hematoma or a history of penetrating neck injury should also raise concern about the patient's airway before the transport. Doubt about airway integrity mandates intubation before the transport, when the tracheal tube can be placed in a controlled environment, with less patient risk.

Artificial airways (endotracheal and nasotracheal tubes, tracheostomies) are prone to problems as well. Loss of ventilation in a mechanically ventilated patient may be caused by tube malfunctions such as change in position (the tube may be dislodged upward, losing the airway entirely, or downward, generally into the right main stem bronchus) or obstruction by pulmonary secretions, clotted blood, or tissue. The pressurized cuff of the endotracheal tube may develop a leak, causing loss of the airway's effective seal. This situation is a particular problem in patients requiring high airway pressures to maintain ventilation, in whom cuff leaks can allow a substantial part of the ventilatory tidal volume to be lost to the upper airway. Effective positive end-expiratory pressure (PEEP) can be lost to cuff leaks also. Bronchospasm may develop in patients outside the ICU, either spontaneously or in reaction to drugs given any time in the hospital course.

Common problems with transport equipment

Respiratory
- Tracheal tube: dislodgment, obstruction, cuff leaks
- Ventilator unit failure, oxygen exhaustion, battery failure
- Dislodgment of chest tubes, interruption of water-seal drainage systems

Cardiovascular
- Alterations in drug infusion
- Pacemaker generator failure (usually battery or electrode placement)
- Failure of cardiac monitors
- Failure of defibrillator or external pacemaker

Neurologic
- Dislodgment of ICP monitors, failure of ICP monitoring equipment

Metabolic
- Alterations in drug infusion (insulin, bicarbonate, CAVHD dialysate)

Renal
- Catheter obstruction

Mechanical ventilation

Problems with mechanical ventilation during transport are legion. Some of these are a result of the fact that patients dependent on mechanical ventilation are inherently sicker than those not dependent. Also, ventilators are complex electropneumatic devices prone to a variety of malfunctions, most of which compromise the process of ventilation itself.

Ventilated patients commonly "fight the ventilator," that is, they attempt respiration on their own in a manner not synchronized with the machine. Such loss of synchrony will compromise air exchange and result in hypoxia and hypercarbia, reflected in the blood gas. Sedation, which may ensure adequate ventilation in the ICU, may not be adequate during transport, when patients may become agitated and hard to ventilate as a result of being moved from bed to stretcher or to a new environment or undergoing painful or frightening procedures. Sedation may need to be reinforced. Fighting the ventilator may lead to very high airway pressures and consequent barotrauma, resulting in pneumothorax in a situation in which detection is difficult until serious deterioration in respiratory function occurs. Excessive airway pressures may require neuromuscular blockade and a change to controlled ventilation if additional sedation does not produce acceptable ventilatory compliance.

Patients who have been adequately maintained with mechanical ventilation may suffer rapid and marked (and possibly irreversible) deterioration in respiration if, during transport, their ventilation is maintained by "bagging" instead of the mechanical ventilator. With bagging, it is very difficult to ensure adequate air exchange and stability of critical ventilatory parameters such as PEEP and respiratory rate in patients with restrictive lung disease, especially patients with adult respiratory distress syndrome (ARDS). If one has any doubt as to whether an artificially ventilated patient will do well with bagging, the benefit of that doubt must be given and the patient transported with a portable, self-contained transport ventilator, preferably of identical design to the patient's ICU ventilator. Most pa-

tients at the Shock Trauma Center of the Maryland Institute for Emergency Medical Services Systems are ventilated with a Siemens Servo 900C machine (Siemens-Elema Ventilator Systems, Schaumburg, Ill.); therefore, we use an identical ventilator equipped with battery pack and oxygen tanks to transport high-risk patients. Our criteria for moving patients on the transport ventilator include intrapulmonary shunting ($\dot{Q}s/\dot{Q}t$) >15%, compliance <35 ml/cm H_2O, PEEP >10 cm H_2O, use of a ventilator mode such as pressure control, or moderate to severe brain injury with poor intracranial compliance. A modified, nonparamagnetic version of this machine is used to transport ventilator-dependent patients for magnetic resonance imaging (MRI) studies. For transport to and ventilation in the hyperbaric oxygen chamber, we use a pneumatically powered ventilator capable of PEEP, which does not require electrical power.

The risk of inadequate ventilation with bagging during transport is especially high in patients with ARDS, who frequently depend on high levels of PEEP to ensure alveolar expansion and gas exchange or who require medical paralysis and controlled ventilation. Such patients should be transported only with a ventilator, one capable of the same support as the ICU ventilator. Frequent samples for blood gases must be drawn to ensure that oxygenation has not changed, because such patients generally have considerable ventilation-perfusion inequality and may deteriorate markedly simply from altered position.

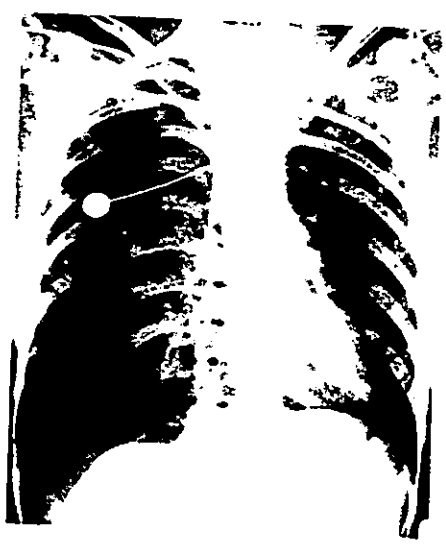

Fig. 1. *Tension pneumothorax. Anteroposterior chest radiograph in supine patient demonstrates a large pneumothorax on right with leftward shift of mediastinum indicating a tension component.*

Pleural cavity and chest wall

Pleural cavity or chest wall problems may arise during transport. Common problems are a spontaneous pneumothorax or worsening or recurrence of a previous pneumothorax thought to be stable or reduced. Frequently, patients have had previous pneumothoraces that responded well to tube thoracostomy and maintenance of suction through a water-seal device. Usually, the suction is discontinued at the beginning of the transport because maintaining suction with the patient out of the ICU and away from wall fixtures is quite difficult. Air may then reaccumulate in the pleural cavity, causing recurrent pneumothorax and loss of ventilation or increased ventilation to perfusion ratio ($\dot{V}/\dot{Q}$) inequality. Staff supervising the transport of a patient with tube thoracostomy suction must be alert to the possibility of reaccumulation of intrapleural air and emergent pleural decompression if the patient's ventilation deteriorates.

Pneumothorax may also occur de novo during transport, as a result of invasive chest procedures (computed tomography [CT] or sonographically guided aspirations or biopsies) or vascular access procedures performed shortly before or during transport. Thus obtaining a chest radiograph is mandatory after central venous access is attempted. If the need for transport is so acute that time does not permit a chest radiograph, then one should be obtained at the earliest possible opportunity and the transport personnel should be made aware of the possibility of an occult pneumothorax or hemothorax. Any deterioration in ventilation during transport that is not rapidly correctable by simple means strongly suggests the need for a chest film along with any other required diagnostic and therapeutic maneuvers, such as pleural decompression.

Frequently, patients undergo intubation and ventilation for multiple rib fractures, especially when a flail chest is present. PEEP and a mechanically supported tidal volume are commonly used in such patients to maintain alveolar expansion; these patients' conditions may deteriorate if they are transported without PEEP or ventilated by means that cannot deliver a consistent PEEP and tidal volume. Again, the solution is to transport these patients with a sophisticated ventilator capable of consistently delivering the needed support.

Monitoring during transport

Monitoring of ventilatory parameters during transport can be divided into:

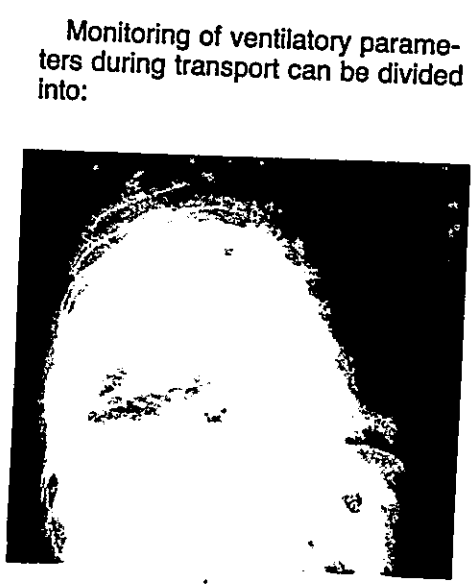

Fig. 2. *Hemothorax. Anteroposterior chest radiograph in supine patient obtained after trauma to right side of chest demonstrates large area of increased density over right side of chest. Mediastinum is displaced to left. Thoracostomy tube placed on right drained a large hemothorax consisting of pleural and extrapleural hemorrhage.*

- Physical examination and tracking of vital signs
- Evaluation of blood gas results
- Observance of serial readings from pulse oximeters and transcutaneous oxygen–carbon dioxide monitors
- Observance of displayed data from mechanical ventilators

Close assessment of skin color, respiratory rate (with and without the ventilator), breath sounds by chest auscultation and chest wall motion, neurologic status, and the presence or absence of air bubbling or leaking from the mouth and nose in intubated patients will provide much information about respiratory status and oxygenation. Increased heart rate or blood pressure may alert the clinician to developing hypoxia or hypercarbia; tachycardia and hypotension may herald a tension pneumothorax. Blood gases give additional evidence of ventilatory adequacy, especially if both arterial and mixed venous specimens are examined simultaneously. Pulse oximetry and transcutaneous oxygen monitoring can help the clinician recognize and evaluate trends in the patient's reaction to changes in ventilatory support, and, combined with other data, help to assess the respiratory picture. However, these modalities are often insensitive to moderate respiratory deterioration, which is more commonly elucidated by the arterial blood gas. Monitoring ventilator data is especially important; early airway obstruction may manifest as a rise in airway pressures, loss of ventilatory volume, or increased respiratory rate. Compliance can be calculated rapidly if the ventilator displays paired airway pressures and volume. Changes in compliance frequently herald a change in ventilatory function and may provide valuable information at the bedside.

Patients not maintained with mechanical ventilation who are transported out of the ICU are prey to certain unique risks. Painful diagnostic studies or therapies, or those requiring the patient to hold still for image quality, may require sedation or analgesia. Such sedation may, if administered in excess or to a patient with preexisting airway or neurologic compromise, cause sufficient deterioration in respiration to require intubation and assisted ventilation. This risk can be minimized by the judicious use of tranquilizing drugs, local anesthesia, careful dosing of drugs known to be respiratory depressants, and consideration of intubation and ventilation in the ICU before transport if ventilation is believed to be at all marginal.

Cardiovascular System

Trauma patients requiring transport are generally monitored for blood pressure by intraarterial cannula and transducer, for central venous pressure by subclavian or internal jugular vein catheter, and for cardiac alterations by continuous single-lead electrocardiography. Pulmonary artery occlusive pressures are not monitored routinely out of the trauma ICU except in the operating room. In patients with pulmonary artery catheters, mean pulmonary artery pressure is monitored instead of central venous pressure. Alterations in blood pressure and in cardiac rate and rhythm frequently occur in critically ill patients. These changes in hemodynamic state may reflect a worsening of disease processes independent of therapeutic interventions, inadvertent changes in medications delivered by mechanical means, noxious stimuli (or the relief of same), or worsening of pretrauma conditions such as ischemic heart disease.

Hypertension

Elevations in blood pressure during transport, diagnostic procedures, or therapeutic procedures may result from increased pain to the patient or inadvertent increase in the delivery rate of vasoactive drugs such dopamine, dobutamine, isoproterenol, epinephrine, or norepinephrine. Infusions of antihypertensive drugs such as nitroprusside

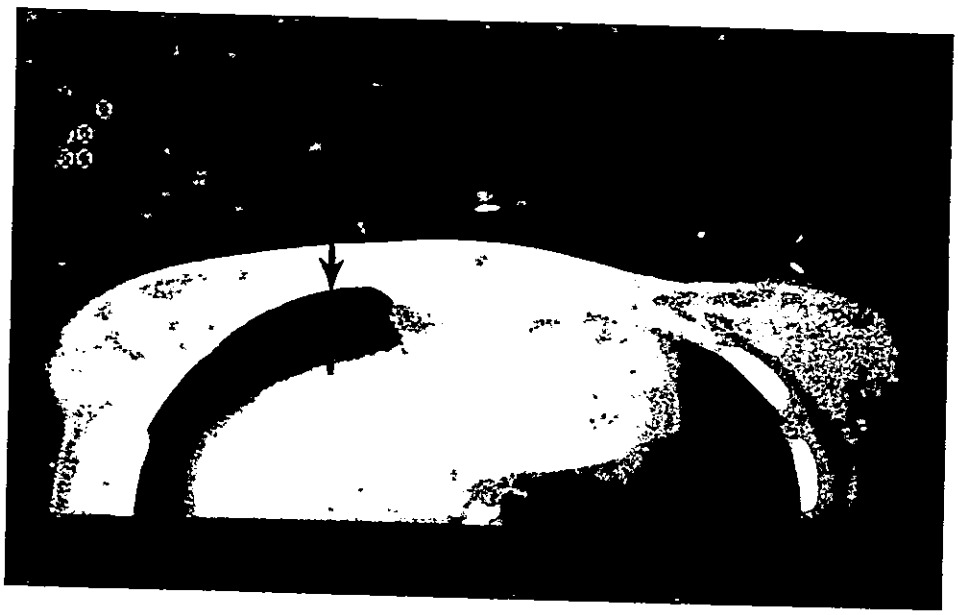

Fig. 3. *CT of pneumothorax. Close-up axial CT section obtained through lung base as part of abdominal CT study after blunt trauma revealed a small anterior pneumothorax on right anteriorly* (arrows). *The pneumothorax was not demonstrated on admission chest radiographs.*

or nitroglycerin may be interrupted by failure of infusion pumps, intravenous (IV) tubing bending or kinking while moving the patient, or dysfunction of a central venous access line. Lack of sedation during frightening or painful procedures may also raise the patient's blood pressure. Infusions of sedatives and analgesics such as morphine or fentanyl may be interrupted by pump, tubing, or line problems, causing increased discomfort and consequent elevation in blood pressure or heart rate. Ventilation disturbances causing hypoxemia or hypercarbia may raise blood pressure; therefore, the workup of unexplained hypertension in the patient should include evaluation of ventilatory status and arterial blood gases as needed. Finally, an unnoticed rise in intracranial pressure (ICP) in the patient with brain injury can elevate blood pressure as well.

Hypotension

Hypotension in the ICU trauma patient during transport can appear from a variety of causes. Chief among them is hypovolemia, either from sudden losses of intravascular volume caused by "third-spacing" of fluids, hemorrhage, or urinary losses caused by diabetes insipidus, or from relative losses of volume caused by progressive sepsis, drug side effects, and shock. For example, manipulating a vascular catheter associated with an unsuspected infection during patient preparation for transport may induce a sudden episode of hypotension soon after the patient leaves the ICU. Inadequate replacement of fluid losses suffered either before transport (hemorrhage, diabetes insipidus) or during transport (forced diuresis from contrast media, bleeding resulting from invasive procedures) may lead to hypovolemia requiring prompt and aggressive volume resuscitation while the patient is out of the trauma ICU.

Cardiac ventricular dysfunction may produce hypotension and reduce organ perfusion. Causes of such cardiac failure include myocardial contusion or infarction, valvular cardiac disease including papillary muscle rupture, or cardiac tamponade resulting from hemopericardium or effusion. Tension pneumothorax can cause hypotension as well, by shifting the mediastinum and preventing effective cardiac filling. Injury to the cervical and thoracic spinal cord may cause a sympathectomy (spinal shock) and hypotension.

Disturbances of infusions of vasoactive drugs may lead to hypotension by several paths: loss of infusion of inotropic or chronotropic drugs will lead to decreased blood pressure or heart rate, and excessive infusion of antihypertensives may lead to lowered blood pressure and compensatory tachycardia. Excess narcotic administration will produce hypotension. In either case, the remedy is simple: flow rates of all drugs infused must be observed carefully and recorded before, during, and after transport.

Alterations in cardiac rhythm

Alterations in cardiac rhythm, such as atrial or ventricular ectopy, junctional rhythms or tachycardias, bradycardia with various escape rhythms, ventricular tachycardia, fibrillation, and asystole, may occur during transport. The causes of such disturbances include:

- Reactions to various cardiovascular drugs (digoxin, β-blockers, or calcium entry blockers)
- The presence of central venous or pulmonary artery catheters, which may irritate the heart electrically
- Electrolyte imbalances
- Hypoxemia
- Failure of permanent or temporary pacemaker generators or dislodgment of pacing electrodes
- Spontaneous dysrhythmias as a result of primary myocardial damage such as infarction or contusion
- Brain injuries
- Ischemic heart disease or preexisting cardiac disease

Such rhythm disturbances are treated in proportion to their severity; a detailed discussion of the treatment of various cardiac dysrhythmias is beyond the scope of this article.

Dysrhythmias that compromise blood pressure or threaten tissue perfusion must be treated during transport. More benign alterations in normal rhythm may be observed during transport and exact cause and treatment sought when the patient returns to the ICU. Anticipation is helpful; for example, checking battery function in a temporary pacemaker generator before leaving the ICU (with spare battery on hand) may prevent a disaster during transport.

Patients with previous rhythm disturbances who are maintained with continuous infusions of antidysrhythmic drugs such as lidocaine, procainamide, or bretylium are prone to all the mechanical infusion-related problems noted above, and any new cardiac rhythm should be evaluated accordingly. Also, antidysrhythmic drugs are generally negative inotropes, and their excessive administration may cause hypotension not otherwise explained.

Monitoring during transport

Besides monitoring heart rate and rhythm, systemic blood pressure, central venous pressure, and mean pulmonary artery pressure, other information about the patient's cardiovascular state may be gained as follows. Clinical observation of skin color (pink, pale, or cyanotic) and temperature (cool, warm, damp, or dry), the character of the peripheral and apical pulses, and estimation of fluid loss (especially via hemorrhage) aid the physician and nurse in assessing the patient's hemodynamic state. Blood pressure measured by arterial line must be correlated with periodic cuff pressures. Careful attention should be

paid to the appearance of the pressure tracing from all vascular catheter recordings; a flattened arterial waveform, or one that contains substantial overringing, should alert the clinician to monitor cuff pressure. Alterations in central venous catheter waveform should prompt a search for changes in catheter position or occlusion of catheter lumens caused by infusion malfunction or catheter bending. With these observations, a reasonably accurate picture of the patient's volume status and ability to perfuse the tissues effectively can be drawn, permitting appropriate preventive or therapeutic measures to safeguard the patient's well-being.

Neurologic System

Causes of neurologic deterioration in ICU trauma patients during transport are many. A useful way to look at these causes is to divide them into those with global worsening of CNS function during transport and those developing focal neurologic deficits.

Global CNS dysfunction

Causes of global CNS dysfunction can include inadequate cerebral perfusion from cardiovascular failure, poor oxygenation of arterial blood from ventilatory compromise, or excessive sedation or analgesia given for transport or procedures. Seizures may also occur from preexisting seizure diatheses, toxicity of various drugs such as lidocaine or aminophylline, low serum glucose from excessive insulin infusion, or hypoxia. All of these problems will lessen the patient's level of consciousness and alter CNS function. Another common cause of altered CNS function in trauma patients is intracranial hypertension. This condition can result from brain swelling from primary traumatic injury (closed brain injury or penetrating injury to the brain substance), expanding mass lesions such as epidural or subdural hematomas, or direct hemorrhage into the brain substance (see O'Callaghan et al., page 389). Increased intrathoracic pressure, stemming from tension pneumothorax or ventilator-patient phasing problems, may elevate ICP and, in a patient with marginal cerebral perfusion, may cause a significant and rapid decrease in cerebral blood flow and function. Increased ICP leads to global depression of CNS function, and, if not checked, possible transtentorial herniation and death.

Focal neurologic deficits

Focal neurologic lesions occur in a wide range of locations and degrees of severity. The most serious of these are expanding mass lesions (hematomas), which may occur in the intracranial spaces (producing lateralizing signs such as hemiplegia or hemiparesis) or next to the spinal cord (producing central or peripheral lesions of varying degrees of lateralization depending on location and size of the hematoma). Compressive lesions of the cord can occur at any level; most commonly they occur in the cervical cord. Inadvertent manipulation of the spine during transport is a preventable cause of spinal cord lesions. Any patient whose spine has not been cleared of suspected injury by definitive radiologic diagnosis should be transported in appropriate immobilization devices until clearance is possible. Virtually all life-saving or diagnostic procedures can be accomplished with the patient in cervical immobilization; no occurrence is more tragic than a spinal cord injury in a patient who arrived at a trauma center neurologically intact.

Patients who have sustained a compressive spinal cord injury before arriving at a trauma center may suffer progressive cord impairment as a result of developing edema and worsening compression at the site of injury. Reducing bony injuries to the cervical and thoracic spine and maintenance of appropriate traction and positioning may prevent such occurrences. Progressive edema of the spinal cord in the quadriplegic or high-cord paraplegic patient may seriously compromise ventilation. A patient who was able to maintain adequate ventilation unassisted in the ICU may require intubation and mechanical ventilation for progressive respiratory failure a very short time later. Such hypoxia and hypercarbia may well worsen focal cord edema, thereby further compromising perfusion. Therefore, securing the airway early in such patients is advisable if any doubt exists about adequacy of ventilation.

Focal neurologic lesions in the periphery may develop as a result of compartment syndromes and nerve compression, or as a result of regional ischemia from vascular compromise. Generally patients are transported with suitable immobilization of injured extremities; however, loss of elevation, accidental destabilization of injuries, and expansion of focal hematomas may contribute to the appearance or worsening of focal deficits. Serial neurologic evaluation of extremity function will detect these developing abnormalities and permit treatment before irreversible loss of function occurs.

Monitoring during transport

Neurologic stability during patient transport is assessed by evaluating:

- Neural status before undertaking transport and comparing that baseline with serial assessments obtained during transport (including eye opening, level of consciousness, extremity function, pupillary size, and ability to follow commands)
- Changes in motor function with regard to either overall strength or focal impairment
- Changes in cranial nerve function, particularly relating to pupillary size and reactivity
- Changes in overall mental status

The patient's neurologic level before moving from the ICU dictates

which of these numerous parameters to watch. Patients who are lucid and readily follow commands are best followed with serial mental status examinations and tracking of command-following ability. Patients with a severely impaired level of consciousness may need to be followed for more basic functions such as pupillary reactivity, similarity, and size; response to painful stimuli; and local motor function, as well as basic vital signs. Patients with severe brain injuries, in which elevated ICP is a concern, frequently have invasive pressure-monitoring devices such as subarachnoid bolts, intraventricular cannulas, and fiber-optic pressure transducers; such devices should be monitored during transport. Staff transporting a patient equipped with such devices must be familiar with methods of zeroing the monitor and should be able to interpret the pressure tracing generated by such devices. If a patient is transported with an ICP monitor, the pharmacologic means of treating elevated ICP must be brought with the patient. Such patients are transported with a ventilator as well.

Metabolic System

The development of metabolic abnormalities during transport often reflects derangement in other vital systems as noted above. Although the means exist to monitor many aspects of a patient's metabolic state, in practice such monitoring consists chiefly of attending to acid-base status, arterial and venous oxygenation and carbon dioxide level, electrolyte levels, and control of blood glucose.

If at all possible, a patient should not be moved from the ICU until the metabolic state is stable. Stabilization is generally achieved by examining laboratory results shortly before transport. Changes in therapy that will affect metabolic state ought not to be instituted shortly before transport because monitoring the effects of those changes while the patient is out of the ICU will be difficult, if not impossible. For example, changes in insulin infusion rate or contents of total parenteral nutrition and IV solutions should not be made shortly before the patient is removed from a controlled environment. Similarly, major changes in ventilatory support should not occur before transport. The patient who requires significant increases in respiratory support possibly should not leave the ICU at all, if the reason for transport is deferrable. Certainly, if the patient is doing well and being weaned from the respirator, such changes should take place only in the ICU, against the possibility that they will not be well tolerated.

Data regarding metabolic stability can be obtained fairly rapidly with the patient outside the ICU; most useful will be determinations of blood gases, finger-stick glucose, serum glucose, and electrolytes. To a great extent, abnormalities in metabolism can be regulated by manipulating therapies in place before the patient leaves the ICU. Supplemental insulin, potassium, phosphate, magnesium, and the like can be brought to the patient outside the ICU and administered as needed. Problems in acid-base balance are generally dealt with by correcting ventilatory abnormalities or eliminating the causes of metabolic acidosis and alkalosis as they are uncovered.

Frequently, in patients requiring pharmacologic control of glucose levels, serum glucose determinations obtained with the patient out of the ICU will be either too low or too high. Common causes of hypoglycemia in such a situation include:

- Excessive insulin administration
- Interruption of parenteral nutrition solution flow (such solutions typically contain large amounts of glucose and often require exogenous insulin to maintain euglycemia)
- Substitution of low-glucose solutions for high in a situation where supplementary insulin is not then adjusted to allow for the decreased glucose load

Causes of hyperglycemia in these situations include:

- Interruption of insulin infusion
- Changes in IV solutions as noted
- Progressive global metabolic decompensation (for example, as a result of sepsis)

Generally, problems in blood glucose control can be treated adequately by adjusting insulin infusion rate or IV solution flow rate. Substituting parenteral nutrition solutions is difficult and expensive.

Renal System

While the patient is out of the ICU, the renal system is usually assessed by monitoring urine output. However, information about blood urea nitrogen (BUN), creatinine, and electrolyte levels may be obtained during patient transport when laboratory work is obtained for a decrease or increase in urine output or other reasons.

Decreased urine output

Urine output may be compromised by several factors; those chiefly responsible are lack of vascular volume and obstruction of urinary drainage systems.

Lack of vascular volume and consequent oliguria or anuria should not manifest as an isolated phenomenon. Vital sign abnormalities (reduced blood pressure, narrowed pulse pressure, tachycardia) accompany volume depletion and frequently will be noted along with a decrease in urine output. Augmentation of volume with infused colloid or crystalloid will generally improve the output and provide the diagnosis.

Another cause of decreased urine output is a decrease in the infusion rate of drugs that stimulate renal blood flow, the most common being dopamine. This decrease may manifest over a period of several hours; if dopamine infusion is

discontinued before transport, the consequent decrease in renal blood flow should be considered as a cause of a later drop in urine output.

Obstructed urinary catheters commonly cause apparent cessation of urine output. In a patient alert enough to complain of pain, bladder distension and discomfort may bring the clinician's attention to the problem. In a comatose or lethargic patient, tachycardia as a response to pain may be the only presenting sign. Flushing or replacing the urinary catheter will determine whether it is malfunctioning and should be done if any question exists.

Elevated intraabdominal pressure can compromise renal perfusion and cause decreased urine output. Severe cases may require laparotomy and the placement of a fascial bridge (e.g., Gore-Tex patch [Gore & Associates Inc., Elkton, Md.]) to decrease the abdominal hypertension.

Increased urine output

Causes of increased urine output are many; the chief dangers of this phenomenon are loss of intravascular volume and consequent tissue hypoperfusion and electrolyte imbalances. Polyuria may stem from:

- The osmotic or loop diuretics (mannitol, furosemide) commonly used in patients with brain injury
- Excessive serum glucose causing an osmotic diuresis from glycosuria
- The administration of intravenous contrast media causing brisk diuresis
- The development of diabetes insipidus

In trauma patients, diabetes insipidus most commonly develops in those with acute serious brain injury. Inasmuch as urine output in this condition can exceed several liters per hour, the threat of shock and vascular collapse is extremely serious, particularly if the patient is supported by inotropic drugs that may maintain blood pressure temporarily in the face of extreme volume depletion. Acutely, diabetes insipidus is diagnosed by comparing urine and serum electrolytes and osmolarity and evaluating the appropriateness of urine output and concentration with respect to the patient's volume status. Treatment in the acute situation consists of volume resuscitation with appropriate fluids and pharmacologic therapy with intravenous vasopressin. For a patient outside the ICU in whom diabetes insipidus and consequent volume depletion or shock develops, volume resuscitation will be the most immediate issue to address, with definitive diagnosis and treatment often begun during transport. Urine and serum chemistries are obtained immediately; intravascular fluids, vasopressin (Pitressin), or both are initiated; and chemistries are evaluated as soon as possible.

Transport Logistics

Transporting a critically ill trauma patient from the ICU, together with acquiring desired information or meeting intended therapeutic goals, can be accomplished safely if a few simple rules are followed. Although these guidelines do not guarantee safe patient transport, they certainly reduce the chance of additional morbidity and mortality as a complication of transport.

Staffing

Adequate and appropriate personnel must be at hand. To move a critically ill patient from one environment to another and back requires the skill and dedication of numerous individuals. For patients transported with a ventilator, a critical care nurse, respiratory therapist, transport orderly, and another person to bring resuscitation equipment if needed are the minimum personnel required. A physician may need to accompany the patient as well. If a physician is not along, one should be notified of the transport, the intended destination, and expected interval of absence from the ICU. A transport sheet should accompany the patient and be completed during the transport, showing relevant times, therapies, complications, and all procedures performed. Necessary drugs and resuscitation equipment must accompany the patient.

Anticipation of problems

A useful philosophy in dealing with the problems involved in moving ICU patients is that complications are the rule rather than the exception. Because critically ill patients are supported by complex mechanical means, and because such devices are commonly prone to failure, some type of breakdown is virtually unavoidable when a patient leaves the ICU supported by multiple infusion pumps; a respirator; and monitoring of arterial, central venous, or pulmonary artery and intracranial pressures.

Certain periods in transport seem to pose particular types of risks. For example, intravenous and vascular lines are most likely to be disconnected or pulled out when the patient is actually moved from bed to stretcher or procedure table. Such movements require adequate personnel, not only to lift the patient but also to watch for damage to monitoring equipment. Similarly, endotracheal tubes are most likely to be pulled out during movement of the patient and must be watched carefully to prevent extubation.

Inasmuch as mechanical devices such as pumps, ventilators, and monitors may fail in transport, spares should be readily available. Bringing spare pumps or a spare ventilator is not necessary if the means exists to replace malfunctioning equipment rapidly. However, the personnel to effect such a replacement must be readily available and the locations of spares must be known.

Certain accessories make transporting patients out of the ICU much easier and lessen the chance

of mechanical malfunction. For example, the use of a self-contained transport ventilator with its own battery pack and oxygen supply reduces the risk of ventilatory failure during transport and frees the respiratory therapist somewhat to help with moving the patient. A variety of self-contained beds with racks for infusion pumps and monitors have been marketed; however, at present we prefer to use a free-standing wheeled rack for all infusion pumps. This rack, which is fastened to the patient's bed with a trailer-hitch arrangement constructed from orthopedic hardware, adds flexibility and stability. We monitor the electrocardiogram (ECG), systemic arterial pressure, central venous pressure, mean pulmonary artery pressure, and ICP when necessary during transport; one monitor generally suffices for this. Currently we use a two-channel portable monitor on transports; one channel continuously monitors ECG, and the second accommodates two cables from pressure transducers and can be switched to display either waveform. This permits continuous monitoring of the arterial pressure with periodic monitoring of central venous or mean pulmonary artery pressure. The fiber-optic ICP monitor has its own display module and does not require connection to a central monitor.

Conclusion

Indeck et al.[1] studied 103 consecutive transports of seriously ill trauma patients (mean Acute Physiology and Chronic Health Evaluation [APACHE] II score, 19.4) over 3 months to assess risk, cost, and benefit of transporting ICU patients. (Our patient population is similar to this study group.) Serious physiologic changes occurred during 68% of transports; 113 changes required an increase in therapy during transport. Twenty-five of the transports resulted in a change in patient management within 48 hours. These findings underscore our belief that transport of critically ill trauma patients out of the ICU should be performed only for cogent reasons and after adequate preparation.

At the current state of the art of trauma intensive care, patients must be moved many times for reasons that are medically justifiable and have an acceptable ratio of benefit to risk. Key to ensuring that patients who must move from the ICU for diagnosis or treatment will do so with the greatest benefit and the least risk are

- Careful planning of patient transports
- Attention to simple and logical basics
- Adequate personnel and equipment to monitor and treat the patient
- Adequate patient preparation
- Maximum hemodynamic and ventilatory stability
- Foreseeing the common problems with both persons and equipment
- Knowing how to treat the complications that develop

Louis Pasteur stated, "Chance favors the prepared man." When moving critically ill patients out of the ICU, this dictum applies absolutely.

Reference

1. Indeck M, Peterson S, Smith J, Brotman S. Risk, cost, and benefit of transporting ICU patients for special studies. J Trauma 1988;28:1020-5.