Postoperative care and prevention of postoperative complications for the multiple-injured

VIRGINIA M. GRABER, RN, MSN
Peoria, Illinois

Drawing from her own experience the author provides both an overview and practical in-depth analysis of the considerations necessary during the critical postoperative period and how to prevent postoperative complications. This article is based on Ms. Graber’s presentation at the AANA’s 45th Annual Meeting and Professional Sessions which was held in Detroit in September, 1978.

An interesting observation can be made from reading about trauma and the care of the multiple-injured patient: the focus is most always on prehospital care, care in the Emergency Room and the importance of early diagnosis and treatment. Up until now there seems to have been little thought about what happens to these patients after they leave the Emergency Room and Operating Room and are sent to the Intensive Care Unit (ICU).

Nurse anesthetists often administer to the patient when he or she is taken from the Emergency Room to surgery. Frequently a nurse anesthetist will be asked to intubate or reintubate patients in the ICU or may be called upon to anesthetize these patients when they are taken to surgery for a second or third time. Therefore, it is important to know what transpires in the ICU, to be aware of some of the care required in nursing the multiple-injured patient, as well as the knowledge, skill and techniques required to render that care.

The term trauma or multiple injury encompasses a wide range of insults to the body. Even individual wounds take on a variety of forms. Some injuries have diffuse systemic effects and others involve one or two organ systems.

Statistics show that the number of accidents is increasing while the number of deaths is decreasing. The average age of the multiple-injured person is 24-25 years, and males suffer more injuries than females.

Critically injured patients who, in the past may have died soon after injury, now because of improved methods of transport, care and observation either survive or are kept alive for a much longer period of time by sophisticated life support systems.

Several factors serve to intensify the difficulties of caring for the multiple-injured:

1. The patient is critically ill.
2. More than one organ system is involved.
3. Frequently, decisions and therapy must be hastily accomplished.
4. More than one discipline is involved in the care, which may present contradictions in the therapy.

As the reader is well aware, there are a multitude of complications which can develop in any individual following surgery. To appreciate the magnitude of the problem of caring for the multiple-injured patient, compound those complications by a catastrophic event of multiple organ system injury and failure. Let us focus, however, on four of the major complications found in trauma patients:

Sepsis or wound infection is a local or generalized bacterial invasion of the body.

Shock is a severe pathophysiologic syndrome associated with abnormal cellular metabolism which in most instances is due to inadequate tissue perfusion.

Acute respiratory failure is a pathophysiologic condition in which there is severe impairment of gas exchange at the alveolar level in patients with previously healthy lungs and intact control of ventilation.

Renal failure is a progressive azo-
temia in a patient who previously had normal renal function.

Sepsis
As surgeons have become increasingly successful in the treatment of the trauma patient, the incidence of sepsis and its related complications has risen sharply. Why? In accidental wounds, for example, there is a high degree of contamination with bacterial organisms and foreign bodies because there frequently is a delay in treatment due to the fact that other things take greater priority or because the patient is not found for several hours after injury. There are other reasons for sepsis such as severe damage to tissue and loss of tissue, complicated by the fact that blood flow to injured tissue may be reduced by the trauma, vascular damage or by previous cardiovascular disease.

Given these overall problems, it would seem that prevention would be easier to accomplish than treating an infection once it is established. While preventative measures are physician-oriented initially, there are implications for nursing. First and foremost, the nurse must remember that sepsis or wound infection is most likely to occur in the trauma patient.

There must be a vigilance for the early signs and symptoms of infection: elevated temperature, erythema, swelling, tenderness or pain, the amount, odor, and character of exudate from wounds, sump and penrose drains, and chest tubes should be noted. Wounds should be inspected at least three or four times during a shift for crepitus as well as skin changes, such as blebs and blanching.

Sterile technique should be employed when cleansing wounds and changing wound dressings. Sterile technique should also be employed when changing the dressings on the cardiovascular pressure line (CVP) the arterial line, and the Swan-Ganz® insertion sites. These dressings, according to the protocol at the St. Francis Hospital in Peoria—the designated regional trauma center, should be changed every other day, while peripheral intravenous site dressings should be changed daily.

Intravenous tubing and filters should also be changed daily or after each bottle of hyperalimentation. Sump drains, meant to drain exudate, blood, serum, or bowel contents, should be watched so that they do not become occluded, and that air vents are not covered. While antibiotics may be administered as a preventative measure, it is paramount that they be given on schedule. This will often require adjusting other therapies and may even require a second or third intravenous line.

Special preventative precautions are vital in caring for the multiple-injured patient, as infection is one of the most frequent causes of morbidity and mortality in the injured person who survives more than 48 hours.

Shock
Shock may be classified as hypovolemic, cardiogenic, septic, and neurogenic. Since shock is an immediately life-threatening disorder, successful treatment of the injured patient in shock requires rapid and correct assessment, the establishment of priorities, and the correction of the disorder.

**Hypovolemic shock** is caused by a decrease in the intravascular volume and is generally associated with a blood volume deficit of at least 15-25%, and a much greater interstitial fluid deficit. These patients almost invariably have a low cardiac output, increased peripheral vascular resistance, increased pulse, decreased filling pressure in the right and/or left heart, and generally cool and clammy skin.

Trauma victims, in addition to the hypovolemia, have significant amounts of tissue damage. This tissue damage, in addition to sequestering large amounts of fluid in and around the injured cells, may release large quantities of vasoactive substances and toxins, which may significantly alter the patient's cardiovascular response.
Cardiogenic shock is caused by impaired function of the heart as the myocardium fails. Cardiogenic shock in the trauma patient may be due to an acute myocardial infarction, direct cardiac injury at the time of the accident, or cardiac tamponade.

In any case, these patients have a low cardiac output, increased peripheral vascular resistance, increased pulse rate, and normal or increased filling pressures in the right and/or left heart, and the skin is cool and clammy.

Septic shock is caused by or associated with severe sepsis and is now generally divided into hyperdynamic and hypodynamic shock. Hyperdynamic septic shock is associated with impaired cell metabolism, which prevents the tissues from properly utilizing the glucose, oxygen and so on brought to them. There is normal or increased cardiac output.

Hypodynamic septic shock patients generally have hypovolemia, due largely to an increased capillary permeability and a pooling in interstitial fluid throughout the body, particularly in the infected area. These changes are apparently a result of the effects of various toxic substances and vasoactive substances liberated or activated by endotoxins and/or lysosomal enzymes from damaged, ischemic or infected tissue.

Neurogenic shock occasionally results without any loss of blood volume. Instead, the vascular capacity increases because of loss of the vasomotor tone due to damage of the sympathetic nervous system. Because of the loss of vasomotor tone the patient develops a hypovolemia. The skin however, is warm and dry. This type of shock may be caused by anesthesia, injury to the upper cord, or damage to the basalar regions of the brain.

Accurate assessment and early diagnosis is imperative. Signs of shock are systolic blood pressure usually less than 80-90 mmHg, severe oliguria, metabolic acidosis and poor tissue perfusion.

Blood pressure. In the trauma victim suffering from hypovolemic shock, major decreases in stroke volume and pulse pressure often occur long before there is any significant fall in systolic pressure. Diastolic pressure generally rises initially with hemorrhage, due to sympathoadrenal stimulation; however, since vasoconstriction can only increase to a certain maximum, continued blood loss will eventually result in a rapid fall of both the systolic and diastolic pressure.

Accuracy in obtaining blood pressure readings is essential in assessing a patient for shock. However, relying only on a sphygmanometer for accurate pressure readings in trauma victims can result in severe errors. A useful instrument—which should be available in every ICU—for measuring blood pressures in hypovolemic and hypotensive patients is the Doppler, which works on the ultrasound principle, and continues to give accurate systolic pressures long after Korotkoff's sounds are inaudible.

If there is any difficulty obtaining a consistent and clear pressure and if the patient's condition is not improving, an intra-arterial catheter should be introduced and connected to a transducer for monitoring pressures. Because of its accessibility, the site most frequently used for the arterial line is the radial artery.

In many of our trauma patients, however, the catheter is left in the femoral artery, following arteriography, for monitoring purposes. This same line should be equipped with a three-way stop-cock to provide an access for obtaining blood samples. It should be remembered when monitoring pressures that changes and trends are more important than isolated readings, and therefore, continuous monitoring is essential.

Urine output correlates with cardiac output. With a sudden decrease in renal blood flow or pressure, there is generally a prompt reduction in the urine output. However, if the fall in flow or pressure is more gradual, urine sodium concentration will often drop and the urine
osmolality may rise significantly before there are any changes in the urine output. Because of the decreased amount of filtrate presented to the tubules, the amount of sodium taken up is increased and the urine becomes very concentrated. Hypovolemia promotes increased release of anti-diuretic hormone which then increases the absorption of water.

As a result of these changes the urine output may fall long before other signs of impaired tissue perfusion become evident. If the urine sodium falls rapidly or is less than 10-20 Meq. per liter, it is an indication that the kidneys are functioning well, but are not being satisfactorily perfused. In most instances this change is due to hypovolemia.

**Acid base changes.** The classic acid base abnormality in established shock had been considered due to metabolic acidosis. However, it is now recognized that early shock is characterized by a respiratory alkalosis and occasionally a metabolic alkalosis. This is because trauma and shock are extremely powerful stimuli to ventilation. Patients with trauma and shock tend to hyperventilate and have minute volumes of 1½ to 2 times normal. Therefore, the initial blood gases of a patient going into shock generally reveal a low pCO₂, a normal bicarbonate level, and an elevated pH.

It should be stressed that initial respiratory alkalosis is not a compensatory mechanism, but is described as a non-specific response. If the effects of trauma or shock are immediately corrected, a metabolic acidosis develops, causing the patient to hyperventilate even more. Tachypnea is often a valuable sign that an otherwise stable patient is deteriorating. If a patient with known shock is not hyperventilating, the incidence of later respiratory failure is increased, therefore one should be alert to an abnormality in the central nervous system, the airway, lungs, chest wall or diaphragm.

Metabolic alkalosis is noted in increasing numbers of critically injured patients. The cause is not always clear; however, some of the more frequently recognized causes are hypokalemia, due to excessive diuresis and metabolism of citrate from the multiple transfusions which are required, or lactate from the Ringer's lactate solution. When metabolic alkalosis is present, the seriousness of the patient's condition is often underestimated, because the most severe metabolic and perfusion defects are usually associated with a lactic acidosis and a resultant decrease in bicarbonate levels.

Furthermore, as the patient with metabolic alkalosis goes into shock, the bicarbonate level will first fall to normal and may be erroneously interpreted as an improvement in the patient's condition. As shock progresses, local changes in cellular metabolism eventually result in the development of metabolic acidosis. If a combined metabolic and respiratory acidosis is allowed to develop, the chances for survival become extremely poor even if the pH level can be restored to normal.

**Tissue perfusion.** It is generally agreed that blood flow and tissue perfusion are more important indicators than are blood pressure readings to determine if a patient is in shock. Changes in pulse pressure often accurately reflect changes in stroke volume and for this reason are a better indication of blood flow than the systolic pressure. Another change which points to poor tissue perfusion is the skin, which is cold and clammy. A cloudy sensorium or increasing lethargy may also be considered signs of poor tissue perfusion.

So, what are the implications for nursing?

1. Close observation for trends and responses are needed to determine which patients are beginning to deteriorate.
2. Multiple-injured patients must be monitored very closely. This should include frequent blood pressure readings, particularly by arterial line; cardioscopic monitoring of the pulse; and careful observation and recording of respirations.

If possible, continuous monitoring...
of arterial pressure is ideal. In any case, the vital signs must be monitored and recorded hourly at minimum.

The simplest most effective treatment for shock following trauma is aggressive replacement of fluids. Two or three liters of Ringer's lactate solution is usually given initially in the Emergency Room over a period of 30-40 minutes. If blood pressure does not respond, then colloidal solutions such as albumin, fresh frozen plasma, and blood are given. Blood transfusions are used liberally in an attempt to keep the hemoglobin at 12.5-14.0Gm. Rapid infusions of fluid require several intravenous lines, and our patients usually have a minimum of two lines: one central and one peripheral. While most of this fluid replacement is accomplished in the Emergency Room or surgery, because of the severity of injury or extensive losses, corrective replacement continues in the ICU.

**Acute respiratory failure**

Generally, when there is a fall in the pO₂ to less than 55-60 mmHg or a rise in the pCO₂ to 45-50 mmHg, acute respiratory failure is said to be present. Many terms are used to describe respiratory failure: acute respiratory distress syndrome (ARDS), shock lung, and traumatic wet lung. In virtually all types of respiratory failure, the same pathophysiology exists, that is, increased lung water and decreased functional residual capacity due to a diffuse microatelectasis and atroventricular shunting.

Some of the most frequent causes of acute respiratory failure in trauma patients include: fat and/or pulmonary emboli, fluid overload, lung contusion, pneumothorax, flail chest or aspiration. Usually two or more of the causative factors are present in multiple-injured patients, thereby putting them at very high risk level.

**Fat embolism.** The classic fat embolism syndrome is generally described as cerebral and respiratory dysfunction developing 24 to 72 hours after an injury usually of the long bones. The fat originates from contused soft tissue fat or the marrow. Some investigators feel that fat may also be present in the lungs from poorly filtered blood transfusions. Others note that the fat emboli can develop after trauma without fractures and the cause is felt to be release of catecholamines.

Massive transfusions are more often given than not in multiple-injured patients, therefore it is imperative that blood filters be used, and they should be changed after the administration of every two units of blood.

**Fluid overload** increases the pulmonary edema that develops in critically injured patients. However, even without fluid overload an increase in capillary permeability causes increased fluid in the lungs, making them less elastic, atelectatic, and less efficient in oxygenation of the blood. If colloid pressure is reduced due to excessive amounts of crystalloids, fluid overload may result. Given the legal problems associated with the administration of type specific blood to combat shock, many patients receive 4-6 liters of crystalloid fluid to replace blood volume.

Other causative factors of acute respiratory failure are aspiration of gastric contents, saliva or blood which frequently occurs at the time of injury. Flail chest, ruptured diaphragm, pneumothorax or lung contusion are other causative factors.

In assessing the patient for respiratory failure it is essential to recognize the clinical manifestations which may occur within hours of the injury and consist initially of tachypnea and hyperpnea. Typically, the lungs are dry and secretions are minimal. Central venous pressure (CVP) is usually not elevated. As the onset occurs, if the patient is being bagged or is on a ventilator, it will be noted that increased pressures are required to bag the patient or to maintain a given tidal volume.

Serial blood gases reveal a progressive decline in pO₂, a rise in pCO₂,
and the pH will fall. Careful observation and assessment is essential because by the time the classic signs of respiratory failure are evident, the syndrome is usually far advanced and difficult to reverse. Preventative nursing care for the patient who may develop acute respiratory failure is essential.

Frequent assessment of breath sounds; change of patient position; elevation of head and chest; maintenance of a patent airway; and reduction of abdominal distention are some factors to be considered in preventative nursing care.

The cardinal rule when assessing restlessness should be: that it is considered due to hypoxia until proven otherwise. Narcotics and sedatives are contraindicated in these patients until it has been proven that the patient's ventilation and oxygenation are adequate.

Since there is usually pulmonary edema in acute respiratory failure, fluid therapy is critical. Once the patient has been adequately resuscitated, gradual dehydration is initiated. This requires accuracy in monitoring intake and output, CVP, and wedge pressures. Dehydration is carried out cautiously so that vital signs and urine output are well maintained.

Some conditions, such as flail chest, require ventilatory assistance. A wide variety of ventilators are available, however, a volume cycled machine is the ventilator of choice. Points to consider when ventilator therapy is indicated include:

1. High tidal volumes of 1000 to 1200 or higher should be utilized if needed. High tidal volumes reduce venous return, therefore a hypovolemic patient may have a severely reduced cardiac output. The patient with acute respiratory failure is placed on the ventilator primarily to obtain optimal alveolar distention; achieving normal blood gases is a secondary consideration. Serial blood gas readings, at least every one to two hours, are mandatory to determine patient response and optimal therapy.

2. Ventilation pressures should not exceed 30 to 40 cm H₂O, if possible in an effort to avoid pneumothorax, however, in some instances pressure of 50 to 60 may be required to maintain adequate ventilation. Pressures must be monitored on the ventilator since changes or increases may indicate obstruction of flow from the ventilator to the patient, either from kinking of tubing, malpositioning of the endotracheal tube, excessive secretions, a pneumothorax or the possibility that the lung is becoming less resilient.

3. Respirations should be controlled at a rate of about 12 to 14 per minute. This enhances improved venous return and prevents the development of respiratory alkalosis.

4. Oxygen should be administered as indicated with a concerted effort toward keeping it as low as possible to prevent oxygen toxicity.

5. Dead space may be indicated to control respiratory alkalosis.

6. Patients should be sighed 6 to 12 times an hour with a volume approximately one-and a-half to twice the tidal volume.

7. PEEP may be indicated in cases where the pulmonary status does not improve with other methods of therapy. When caring for the patient on PEEP the following should be done: a monitoring of ventilator pressures hourly noting increases, auscultate for breath sounds since potential for pneumothorax is greatly increased, and a monitoring of vital signs remembering that with the use of PEEP, venous return can be severely impaired. We feel that once PEEP is initiated it should be interrupted as infrequently as possible, therefore the connector is fitted with a fiberoptic adapter through which we slide the suction catheter. No cuff pressures are taken since this requires deflation of the cuff. No bagging is done. However, the patient is manually sighed prior to each
suctioning. All of these things are done to maintain a continuous PEEP effect.

For those patients in acute respiratory failure who do not require a ventilator, but do need to improve expansion of alveoli, CPAP is used. With this technique the patient exhales against a water pressure of 10-20 cm. The system involves pressurized oxygen which is delivered to a reservoir bag. The gas is delivered to the patient via a one-way valve. Positive pressure is maintained by attaching the expiratory portion of the valve to a water valve. Since we are again attempting to keep the lungs expanded, bagging is done with the same pressure as the CPAP. When the patient is transported to another area of the hospital CPAP and PEEP are maintained by a portable system.

We do use SIMV more frequently now when the patient is first placed on the ventilator if his or her condition allows it. Weaning from the ventilator is accomplished by utilizing SIMV.

All care and treatment of the patient in acute respiratory failure should be directed toward restoring the function of respiration to maximum efficiency.

Renal failure

Initially, oliguria in the trauma patient may represent an appropriate defense mechanism of conservation of water and salt. However, if it is marked or persistent, it may increase the risk of developing acute renal failure. A kidney which is producing concentrated urine at low flow rates is very vulnerable to nephrotoxins and is more likely to develop acute renal failure.

Causes of renal failure are usually classified as prerenal, and postrenal. Prerenal causes of oliguria are reduction in plasma volume, cardiac output or blood pressure, or serum electrolyte imbalance, such as hyponatremia or hypoproteinemia. Postrenal causes are obstruction of urinary flow from the bladder or kidneys, or occlusion of the renal arteries.

The most frequent cause of impaired renal perfusion in the multiple-injured patient is hypovolemia. Other causes include cardiac failure, hypotension, catecholamine release with excessive vasoconstriction, and reduced renal perfusion which occurs as part of the physiologic response to trauma. Fluid deficits in the multiple-injured patient are frequently greatly under-estimated, thereby producing hypovolemia.

Hypovolemia, even when it is mild, will greatly reduce renal perfusion and urine output. What makes this a difficult problem is that there are increased capillary permeability and third-space losses which increase fluid needs, however, weight gain and elevated CVP may make it appear as though the patient is already fluid overloaded. Diuretics should be avoided until other treatable courses have been explored, since further hypovolemia may be its only effect.

Myoglobin must be considered as a cause of renal failure especially when extensive muscle damage has occurred.

Since hypovolemia is the most frequent cause of oliguria and renal failure in the multiple injured, one of the most important aspects of care should be hemodynamic monitoring which includes CVP, arterial line, and the use of Swan-Ganz catheter.

Laboratory data are helpful in confirming a diagnosis of renal failure. In the patient who has suffered severe trauma, accelerated tissue destruction will lead to a greater rise in BUN than creatinine. A rapidly rising BUN with little or no change in the creatinine suggests upper GI bleeding, accelerated tissue breakdown or a blood volume deficit.

Specific gravity may be helpful in determining patient status, however substances such as mannitol, dextran and dyes used for arteriography all increase the gravity well over 1.020.

Urine osmolality and urine sodium are additional measures used to differentiate renal failure from hypovolemia. A low urine osmolality with oliguria

*CPAP—Continuous Positive Airway Pressure.
*SIMV—Synchronized Intermittant Mandatory Ventilation.
and/or a urine sodium of 40 or higher in patients with oliguria suggests renal failure.

Care should be directed toward avoiding oliguria or correcting it by:

1. Regulation of fluids. In the multiple-injured patient there is extensive tissue destruction, volume shifts and massive blood loss, which make it difficult to determine the proper amount of fluid to administer. Therefore, it is important to accurately record the fluid balance, body weight, and closely monitor the vital signs to note changes indicative of fluid deficit and/or overload.

Initially our goal is to keep the patient in a negative or even balance. To do this all losses: urine, stool, drainage from chest tubes, sumps, drains, wound drainage and Levine drainage are replaced cc for cc on an hourly to every 4 hour basis. All fluids given to the patient inclusive of hyperalimentation, IV, blood, plasma and albumin as well as tube feedings and liquid medications are included as replacement fluid. In an effort to avoid over-loading the patient, medications are administered intravenously in the amount of fluid allowed for that hour, or if additional fluid is required this must be included as part of the replacement. Fluids used to keep open arterial lines and Swan-Ganz® catheters are also included as intake. It is also important for us to know how much fluid is given in the operating room.

2. Ideally daily weights should be done. In the event that this is not possible, particular attention must be given to signs of edema formation in dependent areas, and neck vein distention.

3. Monitoring of vital signs, CVP, and Swan-Ganz® pressures should be done hourly and significant changes reported.

4. Close monitoring of laboratory values is essential with particular attention to the BUN, potassium and creatinine.

Conclusion

Nursing care of the multiple injured patient in the ICU is complex. Not only must the physical condition of the patient be considered, but the psychological condition as well. The multiple-injured patient who is physically and psychologically prepared will stand a better chance for recovery, particularly in cases where repeated surgical procedures must be performed. Accurate documentation of the patient’s condition is essential for proper treatment.

REFERENCES


August/1979
AUTHOR

Virginia M. Graber, RN, MSN received a nursing diploma from St. Francis Hospital School of Nursing, Peoria, Illinois, a BS from Bradley University, Peoria, and an MSN from the University of Illinois Medical Center, Peoria. Ms. Graber is a member of the American Nurses’ Association and the American Association of Critical Care Nurses and also serves on the Heart Association Advisory Board for Professional Education and the American Cancer Society’s Nurses Education Committee. Currently, she is employed as head nurse, intensive care unit, St. Francis Hospital Medical Center, Peoria.