IMPERIAL COUNTY PUBLIC HEALTH DEPARTMENT
EMERGENCY MEDICAL SERVICES AGENCY
935 Broadway, El Centro, CA 92243
(760) 482-4468
Fax (760) 482-4519
E-mail: johnpritting@imperialcounty.net

Bruce Haynes, M.D.
EMS Medical Director

John Pritting, M.B.A.
EMS Manager

January 14, 2002

Richard E. Watson, Interim Director
California EMS Authority
1930 9th Street
Sacramento, CA 95814-7043

Dear Mr. Watson:

Thank-you for your letter of December 10, 2001 suggesting changes to our trial study of intravenous fluid administration by EMTs in Imperial County. We appreciate the expert review by your office and the EMDAC Scope of Practice committee.

We are making the following changes in response to items 1-4 in your letter.

1) That this does not include external jugular veins will be clarified.

2) We have extensively discussed the recommendation for standardized boluses of fluids in the protocols. We would ask that this be left as it is for the time being, possibly for the first 18 months of the trial. These amounts currently exist for other providers (paramedics and EMT-IIs) and it would be confusing in the system to have one "out of sync." In addition, a change would require re-writing all protocols and re-educating all proposed EMT-intermediates. We will be glad to evaluate whether this differing amounts are workable, and to change it at the first major evaluation if indicated. We hope you can give this flexibility.

3) The training materials have been included for your review.

4) Our data collection tool will be modified to the form now used by Northern California EMS. We have obtained a copy from NorCal and will be making the necessary changes.

Thank-you again for your assistance with this project, and please let us know if we can provide you with any additional information.

Sincerely,

Bruce E. Haynes, M.D.
EMS Medical Director

John Pritting, EMT-P, MBA
EMS Manager

SCOPE OF PRACTICE OF ADVANCED EMT's IN IMPERIAL COUNTY

An Advanced EMT (AEMT) student or a currently certified AEMT affiliated with an approved AEMT service provider may:

1. while caring for patients in a hospital as part of his/her training or continuing education under the direct supervision of a physician, registered nurse, or physician assistant; or,
2. while at the scene of a medical emergency; or,
3. during transport; or,
4. during interfacility transfer;

in accordance with Imperial County EMS Policies, Procedures, and Protocols:

1. Perform any activity identified in the California Administrative Code, Division 9, Section 100063 (Scope of Practice of an EMT-I).

2. Perform the following procedures:

   - Blood glucose measurement by venous blood/finger stick
   - Defibrillation
   - Oral intubation with Combitube
   - Injections (SC/IM)
   - Intravenous catheter establishment (peripheral IV, excluding external jugular)
   - Intravenous medication and solution administration
   - Oral medication administration (including sublingual)

3. Administer the following medications:

   - Activated charcoal
   - Albuterol, nebulized
   - Aspirin
   - Dextrose 50%
   - Epinephrine (1:1,000)
   - Glucagon
   - Naloxone
   - Nitroglycerine (sublingual tablets or spray)
   - Normal Saline
   - Oxygen

APPROVAL:

Bruce E. Haynes, M.D.
EMS Medical Director

IMPERIAL COUNTY
EMT-II MODULAR TRIAL STUDY PROGRAM

SECTION 10
LECTURE PLAN
ALTERED NEUROLOGIC FUNCTION
(Non-traumatic)
(allow approximately 4 hours)

LESSON OBJECTIVES

At the end of this session, the student will be able to:

1. Identify causes of altered neurologic function to include coma and decreased level of consciousness.

2. Identify how to use "BRIM" as an assessment tool.

3. Identify the various levels of consciousness.

4. Identify the pertinent special questions and physical exam to be elicited from a patient with an altered level of consciousness.

5. Identify the field treatment of a patient with altered neurologic function.

6. Explain the causes, signs and symptoms, field assessment and treatment for hypoglycemia.

7. Explain the pertinent drug information for 50% Dextrose and Glucagon

COMA/DECREASED LEVEL OF CONSCIOUSNESS

Level of Consciousness

The unconscious state relies on a network of cells and fibers in the brain stem, called the reticular formation, which sends impulses to the cerebral cortex, keeping the person awake and perceptive. Injuries to the brain, certain hormones, drugs, and levels of oxygen, glucose, and many other substances affect the reticular formation and its ability to send impulses to the cortex. These especially sensitive cells may forewarn of pathophysiology in alterations of consciousness long before it becomes manifest elsewhere in the body.

Coma, from whatever cause, is a state of unconsciousness from which the pateint cannot be aroused, even by powerful stimulation. The level of consciousness (LOC) is routinely assessed on all patients, but in the pateint whose function is depressed, it assumes increased importance.

-Avoid labels such as "stuporous", "obtunded", "semicomatose". Rather, describe the patient's response to specific stimuli, such as voice or pain.

-Serial monitoring of LOC is the most important measurement to be made; interpretations of other neurologic findings and data are made in relation to this parameter. Consciousness is lost in specific order. By serial determination of specific responses, one can determine whether the patient is improving or worsening.

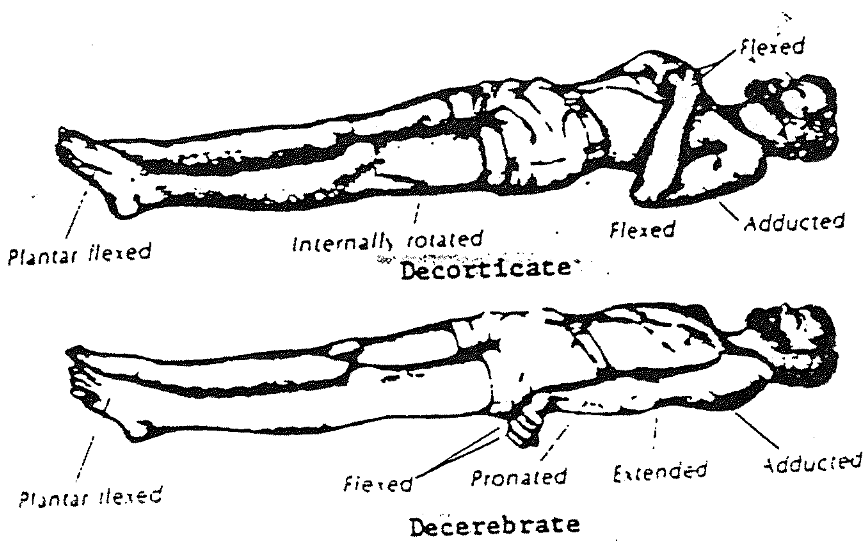
Assessing Level of Consciousness

Assessment of the LOC includes the following:

1. Ask questions to assess the patient's orientation to:
   a. events - does he remember what happened, what the incident was that caused the present problem?
   b. time - what day of the week is it - morning, afternoon, evening?
   c. Place - where is he at present?
   d. person - what is his name?
2. Observe the patient's activity. Is he restless or somnolent?
3. Determine the patient's response to verbal stimuli. How loud must one talk to arouse him? Describe his response to the stimuli as follows:
   a. appropriate - patient follows instruction, responds to his name by opening his eyes and saying "What?" or "Yes".
   b. inappropriate
   c. none
4. If the patient does not respond to verbal stimuli, determine his response to pain. The stimuli should be applied to an uninjured area in a manner that elicits deep pain without tissue injury, such as:
   a. rubbing the sternum
   b. pinching the trapezius muscle
   c. applying pressure to the base of the fingernail with an object such as a pencil. (This may not be useful in the patient with hemiparesis or spinal cord injury).

   Describe the patient's response to painful stimuli as follows:
   a. appropriate - does he move the examiner's hand away?
   b. inappropriate - does he move aimlessly?
   c. note whether it is accompanied by decerebrate or decorticate posturing
   d. no response
5. To determine whether coma is on psychogenic origin:
   a. raise the patient's hand over his head and allow it to drop. If it falls away from the face, the patient is protecting himself and the coma is most likely of psychogenic origin. If his hand falls on his face, the coma state is probably not of psychogenic origin.
   b. gently brush the patient's eyelashes. The pateint with psychogenic coma will flinch or move his eyelids in a protective reflex.

LEVEL OF CONSCIOUSNESS	DESCRIPTION OF RESPONSE	CLINICAL PICTURE
Alert & oriented X3 A	Answers orientation questions to person, place, day appropriately	
Responds to voice V	Patient appears asleep, but will respond to simple commands	Opens eyes on command; lifts arms on command
Purposeful response to pain P	Patient appears asleep but will try to deflect or pull away from painful stimulus	Pushes hand away; tries to roll away
Non-purposeful response to pain	Patient appears asleep but painful stimulus produces random motor responses	Decorticate or decerebrate posturing
No response U	Patient appears asleep and does not respond to any type of stimulus	Remains flaccid with no response



Environmental Clues

In unwitnessed coma, the environment should be searched for any clues as to the cause of the coma. These clues may include the following:

1. Evidence at the scene suggesting trauma (a weapon, broken or displaced furniture).
2. Any medications, drugs, or drug paraphernalia found near the patient or in the household.
3. Any bottles of alcoholic beverages found with the patient.
4. If the fvictim was found in a home, a garage, or an enclosed area, any fumes notices or any evidence of improperly vented gas heaters.
5. Wallet cards that might contain medical information, physicans' prescriptions, or any materials (jewelry, tags, bracelets) identifying an existing medical condition.

Causes of Unconsciousness /Altered LOC

A useful mneumonic to help remember common causes of unconsciousness is AEIOU & TIPS:

A – Alcohol Intoxication – The effects of alcohol intoxication are so common that they require lettle elaboration. They consist of varying degrees of exhilaration and excitement, loss of inhibition, behavior aberration, slurred speech, ataxia, irritability, drowsiness, and, in advanced cases coma. High blood levels of alcohol may produce death from cardiac or respiratory arrest. A potent CNS depressant, alcohol can potentiate or produce an additive effect with CNS depressants and other drugs, notably antihistamines and mind-altering drugs, so that toxic or lethal effects may be produced with lower blood levels of alcohol. Alcoholic coma can be life-threatening when accompanied by respiratory depression and loss of corneal and pupillary reflexes. This condition requires prompt intervention.

Clues:
1. Alcohol on breath
2. Empty bottles lying around

However: merely because a pateint has alcohol on his breath, or is a known alcoholic, does not mean that his coma is caused by alcohol. Alcoholics can go into coma from many other causes.

E – Epilepsy – (Seizure Disorders) There are many types of seizures, but the type that most often produces unconsciousness is a major motor, or grand mal seizure. Seizure activity is a sign of underlying pathology that mandates investigation when it occurs for the first time, especially in the adult patient.

Clues:
1. Incontinence
2. Trauma to the tongue
3. Witnessed seizure
4. Seizure medication in environment such as Phenobarbital and Dilantin
5. Medic-alert tag

I – Insulin – (hypoglycemia/hyperglycemia) Diabetic Ketoacidosis (DKA)results from an absence or an inadequate amount of insulin, which is responsible for transport of glucose across the cell membrane. When insulin is present in inadequate amounts there is a decrease in the transport of glucose into cells and an increase in the breakdown of fat and protein. This altered metabolism ultimately results in dehydration; loss of sodium, potassium, chloride and bicarbonate; and an increase in ketone bodies, leading to the ketoacidotic state. Hypoglycemic reactions may occur when blood glucose level falls below 50mg/100ml of blood or with higher blood glucose levels when the fall has been rapid. In the diabetic, hypoglycemia may be caused by the following:

1. Too much insulin
2. Not enough food (a delayed or missed meal)
3. Unusual or vigorous physical activity or emotional stress

Hypoglycemia can occur in nondiabetic persons as well. Some causes include:

1. Chronic alcoholism
2. Pancreatic tumors
3. Anorexia nervosa
4. Pregnancy/lactation

Hypoglycemia
Bizarre behavior
Diaphoretic
Drooling

Clues:

1. Kussmaul respirations
2. Insulin injection sites
3. Medic-alert tag
4. Insulin in environment
5. Fruity odor to breath

Hyperglycemia

Look in refrigerator for insulin

O - Overdose - A wide variety of drugs and toxic substances can be responsible for producing coma, because of either their primary effects or the effects of withdrawal. Common drugs that produce coma are barbiturates, narcotics (e.g., heroin), hallucinogens, and depressants (e.g., Valium).

Clues: 1. Track marks
2. Pinpoint or dilated pupils
3. Slow respirations
4. Paraphernalia in environment

U - Uremia - (and other metabolic causes) - Uremia is a condition that results from renal failure in which the kidneys fail to excrete urea and other by products or protein metabolism. Other metabolic causes include:

1. Myxedema crisis - severe form of hypothyroidism
2. Liver failure

Metabolic causes of coma are rate.

Clues: 1. Dry scaling skin - uremic frost
2. History of kidney disease or liver disease (cirrhosis)
3. History of thyroid disease
4. Jaundice

T - Trauma/Tumors (to brain) - Any severe trauma may result in hypovolemic shock and coma.

Head trauma - may result in increased intracranial pressure and/or compression of the brain stem which is responsible for the coma state.

Clues: 1. Any evidence of trauma
2. Shock
3. Signs and symptoms of Increased Intracranial Pressure (Cheyne Stoke, Unequal Pupils, Posturing, Cushings refl.)

Tumors of the brain may cause coma as a late manifestation. They may be either primary or metastic tumors. A change in the LOC may be abrupt if bleeding into the tumor occurs.

I - Infection - Septic shock may result in coma from the effect of toxic substances on the body. The toxins cause pooling of the blood in capillaries with dilation of blood vessels.

Coma may be a late sign of meningitis. Meningitis is an inflammation of the coverings of the brain and spinal cord.

Clues: 1. Increased temperature
2. Headache prior to coma
3. Stiff neck prior to coma
4. Shock

P - Psychogenic - Coma of psychogenic origin may be seen in the emergency setting. History may reveal that the patient has been under psychiatric care or has exhibited personality alterations. The coma may have been preceded by a period of apprehension, overactivity and hyperpnea.

also Poison

Patients will also "fake" coma to gain the attention of a significant other.

Clues:
1. Patient will not allow hand to fall in his/her face.
2. Eyelids will flinch if eyelashes are brushed.
3. Hyperventilation
4. Dilated pupils
5. History of psychiatric problems or a recent emotional outburst

also Syncope

S - Stroke - (and cardiovascular problems) - Strokes occur most commonly in persons over 50 years old. They are usually caused by a cerebral thrombosis resulting in compromised cerebral blood flow and damage to brain cells.

Rupture of a cerebral artery aneurysm is most often found in persons below age 40. This condition should also be considered in young adults. Such an aneurysm is congenital and may occur anywhere in the cerebral arterial tree but is most commonly seen at the bifurcation of the internal carotid artery, located at the base of the brain. Leaking or rupture causes bleeding into the subarachnoid space resulting in coma.

Cardiovascular conditions that can produce coma are extensive. Hypertensive encephalopathy is a term used to describe a brain disorder in the pateint with chronic high blood pressure. The hypertension may be so severe as to make the patient comatose. Any disorder of the cardiovascular system which produces shock, such as hemorrhage, dehydration, and cardiac arrest, can lead to comatose states. The patient who has a fainting episode because of a sinus block or atrioventricular dysrhythmias may present with coma.

Clues:
1. Hypertension
2. Unequal pupils
3. Antihypertensive medications (Procardia, Lopressor)
4. History of previous TIA's
5. Abnormal EKG (especially bradycardia or heart blocks)

Field Treatment

See EMT-II and Paramedic protocals.

Note: Even though there are many causes of unconsciousness, diabetes and overdose cause this with such frequency that field treatment is aimed at differentiating between these conditons. As diagnostic tools, D50W and narcan are administered to elicit a response. If the patient does not respond, any further attempts at differential diagnosis should be avoided because a delay in transport may jeopardize the patient.

Don't give simultaneous

PATIENT ASSESSMENT

Scene survey (10-20 seconds)
1. Is the scene safe?
2. What is the mechanism of injury if trauma is suspected?
3. Any environmental clues such as bottles or syringe?
4. Any evidence of gas inhalation?

ABC's
1. Is the patient's airway intact?
2. Is the patient breathing effectively?
3. Does the patient have a pulse? quality? regularity?

Chief Complaint

Obtain a BRIM:

B - What is the breathing rate, rhythm, and quality?
R - What is the patient's LOC?
I - What is the pupillary response? Do the eyes open spontaneously?
M - What is the patient's muscle tone? Can he move all 4 extremities?

~~Safety~~
ABCs
C/C - Altered LOC /Unc
(R) - Oriented/Verbal/Pain
(I) - Pupils
(M) - motor (command/Pain)
Vitals - B/P
AEIOU TIPS

In addition to BRIM, if traum is suspected a complete trauma assessment should be done:

1. Chest/abdomen exam - any obvious injuries to the chest or abdomen?
2. Capillary refill - normal? delayed?
3. Blood pressure?
4. Pulse rate? rhythm? quality?

Paramedic Vital Signs

1. LOC?
2. Pupils?
3. B/P?
4. EKG?
5. Pulse?
6. Respirations? Any patterns?
7. Skin color, moisture, and temperature?
8. Lung sounds?

Physical Exam

Do a complete manipulative secondary survey with emphasis on the following:

1. Any odor on the patient's breath? ETOH? fruity smell?
2. Any signs of trauma, needle tracks, injection marks, bites?
3. Any incontinence of urine or stool?
4. Any medic alert tags or wallet cards?
5. Any bites or lacerations of the tongue?
6. Any jaundice?
7. Any fever?
8. Kussmaul breathing?

Special Questions

A. Specific to the Chief Complaint: (To be obtained from a bystander)

1. How long has the patient been unconscious?
2. What occurred immediately before the pateint lost consciousness?
3. Did the patient complain of chest pain, dizziness, or SOB or headache prior to unconsciousness?
4. Any drugs or alcohol ingested prior to unconsciousness?

B. General Special Questions:

1. Any medical history?
2. Is the pateint taking any medications?
3. Is the patient allergic to anything?

ids). Ketone bodies are strong acids, and their continuous production leads to a metabolic acidosis, which is often at least partially compensated for by a respiratory alkalosis (manifested by Kussmaul's respirations). The body's mechanism for clearing the acid load by the kidneys is overwhelmed by the continuous production of ketone bodies, and profound acidosis eventually occurs. This acidosis, along with the usually severe dehydration secondary to the osmotic diuresis, can lead to death. The treatment of this condition can be lifesaving. Diabetes mellitus is a systemic disease with a number of long-term complications:

- Blindness
  - A total of 5000 diabetics loose their sight each year.
- Kidney disease
  - A total of 10% of all diabetics develop some form of kidney disease, including end-stage kidney failure, which requires dialysis or kidney transplant.
- Peripheral neuropathy that results in nerve damage to the hands and feet and increased incidence of foot infections
- Autonomic neuropathy that causes damage to nerves that control voluntary and involuntary functions and that may affect bladder and bowel control and blood pressure
- Heart disease and stroke
  - High blood glucose and blood fat contribute to atherosclerosis.
  - Diabetics are 2 to 4 times as likely to develop heart disease as nondiabetics and are 2 to 6 times as likely to have a stroke.

Management

The treatment of diabetes mellitus consists of pharmacological therapy (insulin or oral hypoglycemic agents), diet regulation, and exercise to enable the patient's metabolism to be as nearly normal as possible.

Insulin preparations that mimic the actions of the body's natural hormone were found to be effective in 1920. In the past, they were produced from pig (porcine insulin) or ox (bovine insulin) pancreas. More recently, genetic engineering has lead to human insulin (Humulin), which seems to be associated with less antibody development. All of these forms of ***insulin*** are available in rapid-, intermediate-, and long-acting preparations. ***Insulin*** is administered by injection; it is a protein that would be digested if consumed orally.

Usually, an insulin-dependent diabetic self-administers a single dose of one of the long-acting ***insulin*** preparations each day and additional quantities of a rapid-acting ***insulin*** (lasting only a few hours) for those times of day when the serum glucose would be elevated (for example, at meal times).

Another means by which the patient self-administers ***insulin*** is via an ***insulin***-infusion pump. These devices administer a continuous dose of ***insulin*** and are adjusted so that the level of blood glucose is constantly controlled. Regular monitoring by the patient of glucose levels (blood or urine testing) is necessary to ensure adequate medication control. Medication balance is delicate. The same dosage of ***insulin*** that appears correct on one occasion may be too much or too little on another occasion depending on various factors (for example, diet, exercise, and infection).

Oral Hypoglycemic Agents

Oral hypoglycemic agents stimulate the release of insulin from the pancreas. They are effective only in patients who have functioning beta cells. Commonly prescribed oral hypoglycemic agents include chlorpropamide (Diabinese), tolazamide (Tolinase), tolbutamide (Orinase), acetohexamide (Dymelor), Glucotrol, and glyburide (Micronase).

● DIABETIC EMERGENCIES

Three life-threatening conditions may result from diabetes mellitus: hypoglycemia (insulin shock), hyperglycemia (diabetic ketoacidosis [DKA]), and **hyperosmolar hyperglycemic nonketotic (HHNK) coma.**

Hypoglycemia

Hypoglycemia is a syndrome related to blood glucose levels below 80 mg/dl. Symptoms usually occur at levels less than 60 mg/dl or at slightly higher blood glucose levels if the fall has been rapid. The condition may occur in nondiabetic patients as well. It is usually a result of excessive response to glucose absorption, physical exertion, alcohol or drug effects, pregnancy and lactation, or decreased dietary intake. In diabetics, hypoglycemic reactions are usually caused by:

- Too much ***insulin*** (or oral hypoglycemic medication)
- Decreased dietary intake (a delayed or missed meal)
- Unusual or vigorous physical activity
- Emotional stress

Less common causes and predisposing factors include:

- Chronic alcoholism (Alcohol depletes liver glycogen stores.)
- Adrenal gland dysfunction
- Liver disease
- Malnutrition
- Tumor of the pancreas
- Cancer
- Hypothermia
- Sepsis
- Administration of beta blockers (*propranolol*)
- Administration of salicylates in ill infants or children
- Intentional overdose with ***insulin***, oral hypoglycemic agents, or salicylates

Signs and Symptoms

The signs and symptoms of hypoglycemia are usually rapid in onset (often within minutes). In early stages, the patient may complain of extreme hunger and demonstrate one or more of the signs and symptoms secondary to decreased glucose availability to the brain:

- Nervousness, trembling
- Irritability
- Psychotic (combative) behavior
- Weakness and incoordination
- Confusion
- Appearance of intoxication
- Weak, rapid pulse
- Cold, clammy skin
- Drowsiness
- Seizures
- Coma (in severe cases)

NOTE:
Hypoglycemia should be suspected in any diabetic patient with behavioral changes, confusion, abnormal neurological signs, or unconsciousness. This condition is a true emergency that requires immediate administration of glucose to prevent permanent brain damage or death.

Diabetic Ketoacidosis

DKA results from an absence of or resistance to insulin. The low insulin level prevents glucose from entering the cells and causes glucose to accumulate in the blood. As a result, the cells become starved for glucose and begin to use other sources of energy (principally fat). The metabolism of fat generates fatty acids and glycerol. The glycerol provides some energy to the cells, but the fatty acids are further metabolized to form ketoacids, resulting in acidosis.

Because any acidosis increases transport of potassium from the intracellular space into the intravascular space, the subsequent diuresis results in hyperkaluria (high potassium concentration in the urine) and a total body potassium deficit (Box 19-1). In addition, the sodium concentration in the extracellular fluid usually decreases through osmotic dilution and is replaced by increased quantities of hydrogen ions, thus adding greatly to the

BOX 19-1

Common Causes of Diabetic Ketoacidosis

- Too-small ***insulin*** dose
- Failure to take ***insulin***
- Infection
- Increased stress (trauma, surgery)
- Increased dietary intake
- Decreased metabolic rate
- Other less common predisposing factors, including significant emotional stress, alcohol consumption (often associated with hypoglycemia), and pregnancy

acidosis. As blood sugar rises, the patient undergoes massive osmotic diuresis, which together with vomiting causes dehydration and shock. The associated electrolyte imbalances may cause cardiac dysrhythmias and altered neuromuscular activity (including seizures).

Signs and Symptoms

The signs and symptoms of DKA are usually related to diuresis and acidosis. They are usually slow in onset (over 12 to 48 hours) and include:

- Diuresis
  - Warm, dry skin
  - Dry mucous membranes
  - Tachycardia, thready pulse
  - Postural hypotension
  - Weight loss
  - Polyuria
  - Polydipsia
- Acidosis
  - Abdominal pain (usually generalized)
  - Anorexia, nausea, vomiting
  - Acetone breath odor (fruity odor)
  - Kussmaul's respirations in an attempt to reduce carbon dioxide levels
  - Decreased level of consciousness

NOTE:
DKA patients are seldom deeply comatose. Patients who are unresponsive should be assessed for another cause, such as head injury, stroke, and drug overdose.

Hyperosmolar Hyperglycemic Nonketotic Coma

HHNK coma is a life-threatening emergency that frequently occurs in older patients with type II diabetes or in undiagnosed diabetics. The syndrome differs from DKA in that residual insulin may be adequate to prevent ketogenesis and ketoacidosis but not enough to permit glucose use by peripheral tissues or decrease gluconeogenesis by the liver. The hyperglycemia produces a hyperosmolar state followed by an osmotic diuresis, dehydration, and electrolyte losses. Hence, these patients typically have greater hyperglycemia because they are more dehydrated and less ketone formation, since the presence of insulin in the liver directs free fatty acids into nonketogenic pathways, resulting in less acidemia than in patients with DKA (Fig. 19-4). Precipitating factors and signs and symptoms of HHNK coma include the following:

- Precipitating factors
  - Type II diabetes
  - Old age
  - Preexisting cardiac or renal disease
  - Inadequate insulin secretion or action
  - Increased insulin requirements (stress, infection, trauma, burns, myocardial infarction)
  - Medication use (thiazide, diuretics, glucocorticoids, *phenytoin*, sympathomimetics, *propranolol*, immunosuppressives)
  - Supplemental parenteral and enteral feedings

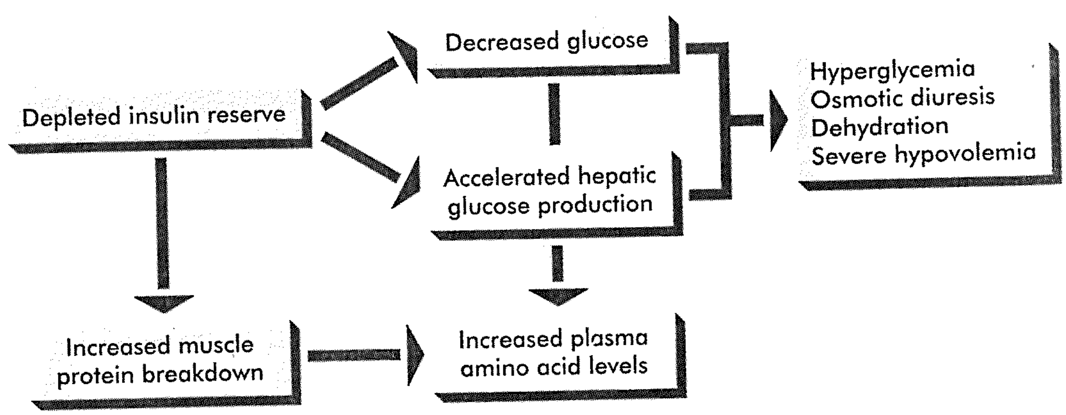


Fig. 19-4 Pathophysiology of HHNK coma.

- Signs and symptoms
  - Weakness
  - Thirst
  - Frequent urination
  - Weight loss
  - Extreme dehydration
    - Flushed, dry skin
    - Dry mucous membranes
    - Decreased skin turgor
    - Postural hypotension
    - Altered levels of consciousness
    - Tachycardia
    - Hypotension
    - Tachypnea

Assessment of the Diabetic Patient

A patient with a diabetic emergency may have a variety of signs and symptoms, many of which may mimic other more commonly encountered conditions. Therefore the paramedic must maintain a high degree of suspicion for diabetes-related illness.

In addition to the patient assessment measures appropriate for any emergency patient encounter (primary survey, secondary survey, and treatment of life-threatening illness or injury), the paramedic should be alert for medical alert information, the presence of insulin syringes, and diabetic medications (often kept in a refrigerator). Components of the patient history important in assessing diabetic patients include onset of symptoms, food intake, ***insulin*** or oral hypoglycemic use, alcohol or other drug consumption, predisposing factors (exercise, infection, illness, stress), and any associated symptoms.

Management of the Conscious Diabetic Patient

If the diabetic patient is conscious and able to converse, the paramedic should obtain a pertinent history while assessing the patient's airway, breathing, and circulation. High-concentration oxygen should be administered, and if appropriate, the patient should be given glucose.

Medical control may recommend drawing a blood sample for laboratory analysis before administering glucose. Some EMS services use field glucose testing with Dextrostix, Chemstrips, or a glucometer (Fig. 19-5). Any patient with a glucose reading of less than 80 mg/dl and signs and symptoms consistent with hypoglycemia should receive ***dextrose***. All patients who have experienced a diabetic reaction, regardless of severity, should be encouraged to be evaluated by a physician. During transport, the patient's level of consciousness, vital signs, and ECG should be continuously monitored.

Methods of glucose administration vary by protocol (Box 19-2). If the patient is alert with a gag reflex and able to swallow, sugar may be orally

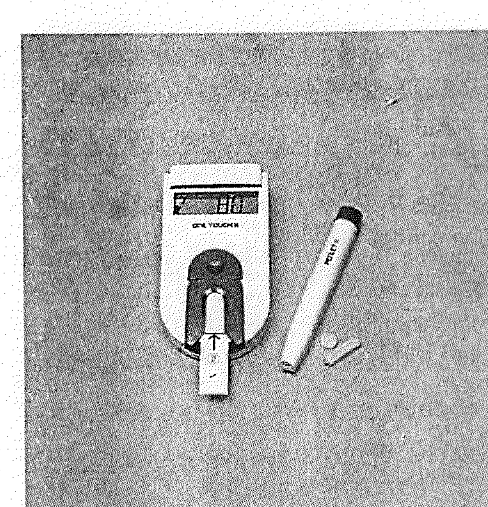


Fig. 19-5 Glucometer for measuring serum glucose levels.

BOX 19-2

Cautions for Administering Intravenous Glucose

- ***Dextrose 50%*** should not be administered to infants or young children.
- The administration of ***dextrose 50%*** may precipitate neurological complications in alcoholics and other patients with thiamine deficiency. Therefore ***thiamine*** administration before or concurrent with the administration of ***dextrose*** should be considered in patients with suspected thiamine deficiency.

administered in the form of a candy bar, a glass of orange juice mixed with sugar, or a nondiet soft drink or by sublingual or buccal administration of a glucose gel preparation. An alternate method is to slowly administer ***dextrose 50%*** through a stable peripheral vein. (This dose may be repeated by protocol.)

Management of the Unconscious Diabetic Patient

Prehospital management of any unconscious patient should be directed at airway management, high-concentration oxygen administration, and ventilatory support. Depending on protocol, an intravenous line of a lactated Ringer's solution or a saline solution should be established to replenish fluids and electrolytes (flow rate to be indicated by the patient's blood pressure and heart rate), and a blood sample should be drawn for laboratory analysis. If alcoholism or other drug abuse is suspected, medical direction may recommend the administration of ***thiamine, naloxone*** (Narcan), or both, before the administration of glucose.

TABLE 19-2 Differential Considerations in Diabetic Emergencies

Findings	Hypoglycemia	Hyperglycemia	HHNK Coma
History			
Food intake	Insufficient	Excessive	Excessive
Insulin dosage	Excessive	Insufficient	Insufficient
Onset	Rapid	Gradual	Gradual
Infection	Uncommon	Common	Common
Gastrointestinal tract			
Thirst	Absent	Intense	Intense
Hunger	Intense	Absent	Intense
Vomiting	Uncommon	Common	Uncommon
Respiratory system			
Breathing	Normal or rapid	Deep or rapid	Shallow/rapid
Breath odor	Normal	Acetone smell	Normal
Cardiovascular system			
Blood pressure	Normal	Low	Low
Pulse	Normal, rapid, or full	Rapid or weak	Rapid or weak
Skin	Pale or moist	Warm or dry	Warm or dry
Nervous system			
Headache	Present	Absent	Absent
Consciousness	Irritability	Restless	Irritable
	Seizure or coma	Coma (rare)	Seizure or coma
Urine			
Sugar level	Absent	Present	Present
Acetone level	Usually absent	Usually present	Absent
Serum glucose levels	Less than than 60 mg/dl	Greater than than 300 mg/dl	More than 600 mg/dl
Treatment response	Immediate (after glucose) (NOTE: If the hypoglycemic episode is prolonged or severe, the response may be delayed and may require more than one dose.)	Gradual (within 6-12 hours after medication and fluid replacement)	Gradual (within 6-12 hours after medication and fluid replacement)

NOTE:

If the patient's age (over 50) or clinical history suggests a transient ischemic attack or stroke, the administration of a concentrated glucose solution may exacerbate cerebral damage. (Consult with medical control.) Otherwise, *any patient in coma of unknown origin should receive dextrose,* particularly if hypoglycemia cannot be ruled out as a possibility.

If an intravenous line cannot be established, the administration of subcutaneous or intramuscular ***glucagon*** helps raise serum glucose levels by stimulating the breakdown of liver glycogen. However, ***glucagon*** is ineffective in chronic alcoholics and those with liver disease. As previously stated, all patients who experience a diabetic emergency should be transported for physician evaluation. Definitive treatment for patients with DKA requires administration of ***insulin,*** fluid replacement, and in-hospital observation.

Differential Diagnosis

Differentiating the origin of a diabetic emergency is sometimes difficult in the prehospital setting. When the paramedic is in doubt as to the cause, all diabetic patients should receive glucose. The findings in diabetic emergencies listed in Table 19-2 should assist in the differential diagnosis.

REFERENCE

1. A team approach to diabetes, *Barnes Health News* 8(4):9, 1989.

Summary

Diabetic emergencies are metabolic disorders frequently encountered in the prehospital setting. Although the illness may be life threatening, proper assessment, a thorough history, and appropriate pharmacological therapy can often reverse the immediate pathological process. Volume repletion is the initial primary therapeutic goal in DKA and HHNK coma, whereas administration of glucose is the primary goal of therapy in the hypoglycemic patient.

Imperial Valley College
Emergency Medical Services Training
(Indications/Dosage/Route Based on Imperial County Protocols)

Dextrose 50% (D50W, 50% Glucose)

Class:
- Carbohydrate

Actions:
- Provides free sugar for quick absorption into the blood stream
- Is the principle energy source utilized by the brain and other tissues

Indications:
- Suspected hypoglycemia with glucometer blood sugar level of <60
- Suspected hypoglycemia when unable to obtain glucometer reading
- Suspected hyperkalemia in hemodialysis patient (widened QRS, peaked T waves, bradycardia)

Dosage/Route:
- Hypoglycemia or suspected hypoglycemia
  - •25 gm IVP SO
  - •May repeat per BH
- Suspected hyperkalemia
  - •25 gm IVP SO

Side Effects:
- None

Contraindications:
- None

Special Information:
- Onset of action 1-2 minutes
- Determine glucometer reading prior to administration, if at all possible
- Tissue necrosis occurs with infiltration; make sure injection is IV; aspirate before and halfway through administration
- Assess effectiveness of administration with patient assessment

Pediatric Note:
- Refer to Pediatric Drug Guide
- Under 3 years of age and <15 kg
  - •Use dextrose 25% preload or dilute D50 1:1 with NS slow IVP

GLUCAGON

CLASS: Polypeptide

INDICATION: Suspected hypoglycemia when blood sugar less than 60 or unobtainable. (Refer to local protocol)

ACTION: Increases blood glucose by converting glycogen stored in the liver to glucose.

DOSAGE/ROUTE:
1 mL (1 unit) IM

SIDE EFFECTS:
Nausea
Vomiting

CONTRAINDICATIONS:
None

SPECIAL INFORMATION:

Glucagon will not work if a patient's glycogen stores in the liver are depleted.
Onset of action is 5-20 minutes.
To reconstitute, rub back and forth between palms of both hands.
After reconstitution, check that the solution is transparent and doesn't have any undissolved medication floating in it.
To assess effectiveness of Glucagon administration, reassess the patient's LOC.

OPERATIONS: ADVANCED EMT TREATMENT PROTOCOLS

SUBJECT: ALTERED NEUROLOGIC FUNCTION (NON-TRAUMATIC)

POLICY NUMBER: 2030

BLS

Ensure patent airway, give oxygen and/or ventilate prn.
Position patient as follows:
If conscious with suspected CVA, elevate head 20-30 degrees
If unconscious, place patient lateral recumbent
Immobilize spine if indicated

HYPOGLYCEMIA (suspected)
If patient is awake, has a gag reflex and can swallow:
Give oral glucose solutions to include:
fruit juices, 2-3 packets of granulated sugar dissolved in liquid,
glucopaste on tongue depressor placed between cheek and gum,
glucose tablets: 2-3 tablets, repeat as needed

SEIZURES
Protect from injury
Treat associated injuries
Febrile seizures (pediatric)
Remove clothing
Sponge with tepid water prn.
Avoid shivering

ALS

Glucometer

SO Establish IV

HYPOGLYCEMIA (altered LOC, inadequate gag reflex, insufficient response to oral glucose preparations)

SO Dextrose 50% 25 gm IVP if BS level < 60 or unobtainable; may repeat per BH

OR

SO Glucagon 1 mg IM if no IV and BS level < 60 or unobtainable

PEDIATRIC NOTE:
Refer to Pediatric Drug Guide

APPROVAL:

Bruce E. Haynes, M.D.
EMS Medical Director

IMPERIAL COUNTY EMERGENCY MEDICAL SERVICES AGENCY
EMT-II MODULAR TRIAL STUDY PROGRAM

SECTION 16
LECTURE PLAN
SHOCK, IV FLUID THERAPY & IV BOLUS MEDICATION
(Allow 16 hours)

LESSON OBJECTIVES

1. Describe the constituents of blood and their functions.
2. Distinguish among the fluid compartments of the body.
3. Describe the assessment and management of a patient with a fluid imbalance.
4. Differentiate the etiologies, signs, symptoms, and management of each classification of shock.
5. Discuss the indications, contraindications, complications, and techniques of intervention for shock.
6. Discuss fluid resuscitation for victims of traumatic injury and hyperthermia.
7. Demonstrate the proper procedure for IV setup, IV insertion into a peripheral vein, and IV bolus medication administration.
8. Discuss local protocols for shock, trauma, and hyperthermic patients.

BOX 13-1

The Fick Principle

1. An adequate amount of oxygen must be available to red blood cells through the alveolar membrane in the lungs to ensure hemoglobin saturation with oxygen. This requires adequate ventilation of the lungs through the patient's airway, a high partial pressure of oxygen in inspired air (FiO_2), and minimal obstruction to the diffusion of oxygen across the alveolar capillary membrane.
2. The red blood cells must be circulated to the tissue cells. This requires adequate cardiac function, an adequate volume of blood flow, and proper routing of blood through the vascular channels.
3. The red blood cells must be able to adequately load oxygen in the pulmonary capillaries and unload the oxygen at the site of peripheral tissue cells. This requires normal hemoglobin levels, circulation of the oxygenated red blood cells to the tissues in need, close approximation of the tissue cells to the capillaries to allow for diffusion of oxygen, and ideal conditions of pH, temperature, and other factors. Fick principle (Tissue oxygenation) = (Arterial oxygen content − Venous oxygen content) × Perfusion

temic pressure, like pulmonic pressure, has two phases: systolic and diastolic. The difference between these two pressures is the pulse pressure. Pressure is greatest at its origin (the heart) and least at its terminating point (the vena cava). This pressure gradient changes significantly at the arteriole as a result of peripheral vascular resistance.

The peripheral vascular resistance (afterload) is the total resistance against which blood must be pumped. It is essentially a measure of friction between the vessel walls and fluid and between the molecules of the fluid themselves (viscosity), both of which oppose flow. When the resistance to flow increases and the flow remains constant, blood pressure increases. Resistance to blood flow depends on fluid viscosity, vessel length, and vessel diameter.

Viscosity is the physical property of a liquid characterized by the degree of friction between its component molecules (for example, between the blood cells and between the plasma proteins). Viscosity normally plays a minor role in blood flow regulation because it remains fairly constant in healthy individuals. Vessel length in the human body also remains constant. Vessel diameter is the primary factor affecting the resistance to blood flow.

Major arteries are large and offer little resistance to flow. Arterioles have a much smaller diameter than arteries and offer the major resistance to blood flow. The smooth muscle in the arteriole walls can relax or contract, changing the diameter of the inside of the arteriole as much as fivefold. Arterial blood pressure is thus regulated primarily by the vasoconstriction or vasodilation of these vessels.

The Lungs

Adequate oxygenation of tissue cells requires that adequate oxygen be made available to the red blood cells at the capillary membrane in the lungs (the first component of the Fick principle). This is made possible by the high partial pressure of oxygen in inspired air, adequate depth and rate of ventilation, and matching of pulmonary ventilation and perfusion.

THE BODY AS A CONTAINER

The healthy body may be viewed as a smooth-flowing fluid delivery system inside a container. The container must be filled to achieve adequate preload and tissue oxygenation. Though the size of the container of any particular human body is relatively constant, the volume of the container is directly related to the diameter of the resistance vessels, which may change rapidly. Any change in the diameter of the vessels changes the volume of fluid the container holds, thereby affecting preload.

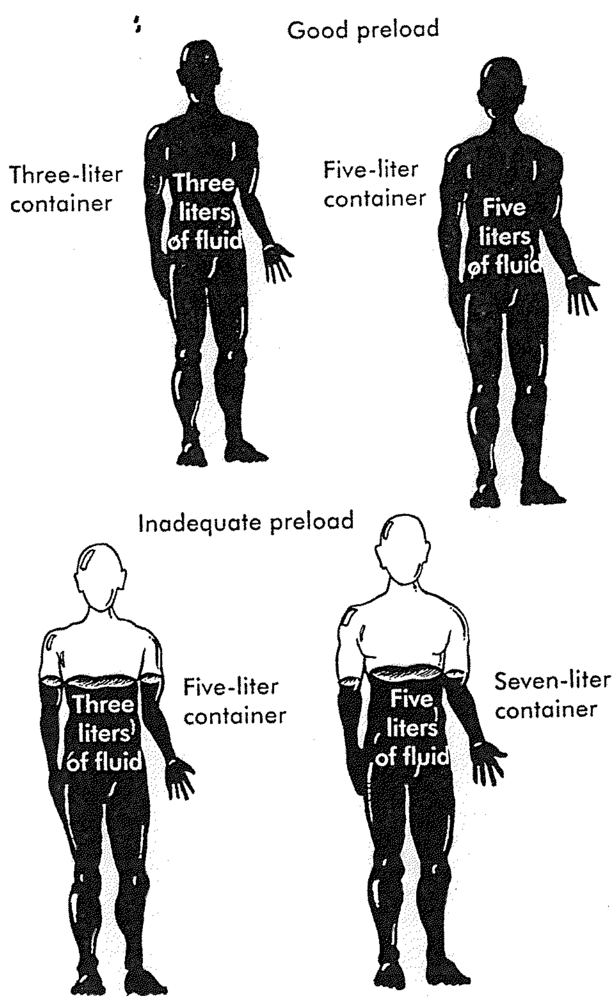


Fig. 13-1 Fluid versus container volume.

An example of this principle is a 5-L container, the normal container size for a 70-kg adult male (Fig. 13-1). If the fluid volume is 5 L, preload is adequate. With a strong myocardium, cardiac output and perfusion are also adequate. If 2 L of this fluid have been lost, either externally or internally, the remaining 3 L are inadequate to supply an effective preload. Since cardiac output depends on preload, a decrease in preload significantly decreases cardiac output.

If the patient is hypovolemic and the 5-L container has remained the same size despite the 3-L volume, the patient becomes hypotensive because of decreased cardiac output. However, if the container is reduced to 3 L by compensatory mechanisms (for example, vasoconstriction), the 3-L container can provide adequate preload to the heart with the 3 L of available fluid. (This is obviously at the expense of certain tissues that are not perfused in this constricted state.)

If fluid is adequate for a 5-L container but the container size has been enlarged to 7 L by illness or injury that results in vasodilation, the 5 L of fluid do not provide adequate preload for the container (relative hypovolemia). Other factors that are occasionally responsible for vasodilation include cardiac and blood pressure medications, allergic reaction, heat- and cold-related injuries, and alcohol and drug use.

BLOOD AND ITS COMPONENTS

The average adult male has a blood volume of 7% of total body weight, and the average adult female, 6.5% of total body weight (70 ml multiplied by kg body weight). Normal adult blood volume is 4.5 to 5 L. This amount remains fairly constant in the healthy body.

Plasma is approximately 92% water and is the blood's solvent (the liquid portion of blood). It is through plasma that salts, minerals, sugars, fats, and proteins are circulated in the body.

Plasma contains three major proteins: albumin, globulin, and fibrinogen. Albumin is the most plentiful plasma protein. It is similar in consistency to egg white and gives blood its gummy texture. This large protein helps keep the water concentration of blood low enough to allow water to diffuse readily from tissues into blood. Globulins (alpha, beta, and gamma) serve two main functions: alpha and beta globulins transport other proteins, and gamma globulins give people immunity to disease. Fibrinogen aids in blood clotting by forming a web of protein fibers that binds blood cells together.

In addition to helping the body maintain its blood supply, plasma proteins serve several purposes. Should blood acidity change, the proteins can act together as an acid or base to correct it. If the body runs short of food, plasma protein can also temporarily meet the nutritional needs of the body.

Although oxygen dissolves in plasma, it can only carry about 1% of the oxygen the body demands. Red blood cells (erythrocytes) transport

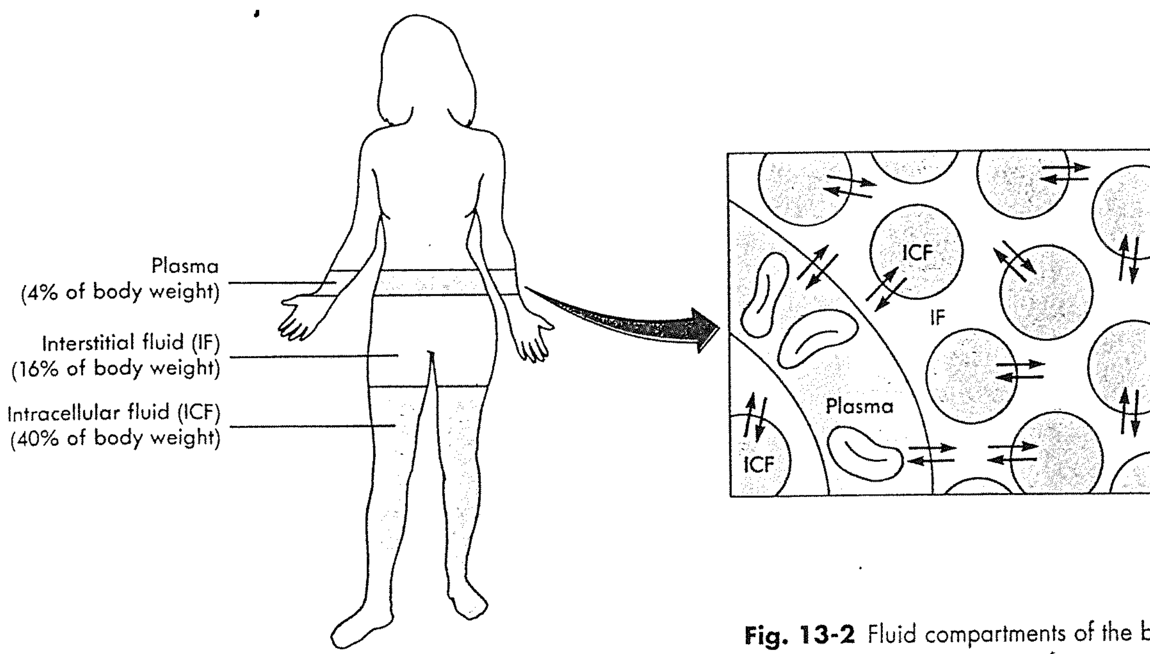


Fig. 13-2 Fluid compartments of the body.

the other 99%. Red blood cells make up approximately 45% of the blood and are the most abundant cells in the body. Red blood cells provide oxygen to tissues and remove carbon dioxide. Each red blood cell contains approximately 270 million hemoglobin molecules. These molecules allow erythrocytes to pick up oxygen in the lungs and release it to body tissues.

White blood cells (leukocytes) defend the body against various pathogens (bacteria, viruses, fungi, and parasites). The bone marrow and lymph glands constantly produce and maintain a reserve of white blood cells, but not many are present in a healthy blood stream. (White cells are outnumbered by red cells 600 to 1.) When a pathogen invades the body, the leukocyte reserves are released.

Another part of the body's defense mechanism is platelets, which help to stop escaping blood. Platelets are formed in the red bone marrow and work by swelling and adhering together to form sticky plugs, thereby initiating the clotting phenomenon.

FLUIDS AND ELECTROLYTES

Water is the main component of body mass, accounting for 50% to 60% of body weight in adults. The importance of body water is highlighted by two facts: it is the medium in which all metabolic reactions occur, and the precise regulation of the volume and composition of body fluid is essential to health. (A 20% to 25% loss of body water usually results in death.) The human body can be viewed as containing two fluid compartments: the extracellular fluid and the intracellular fluid (Fig. 13-2).

Extracellular Fluid

Extracellular fluid is the water found outside the cells and includes the intravascular and interstitial compartments. This fluid accounts for approximately 20% of total body weight, the intravascular component comprising approximately one third of this. Interstitial fluid is the extracellular fluid between the cells and outside the vascular bed. This category also includes special fluids, such as cerebrospinal fluid and intraocular fluid, and accounts for 15% to 16% of total body weight.

Intracellular Fluid

Intracellular fluid is the fluid found in all of the body cells. It accounts for 40% of total body weight.

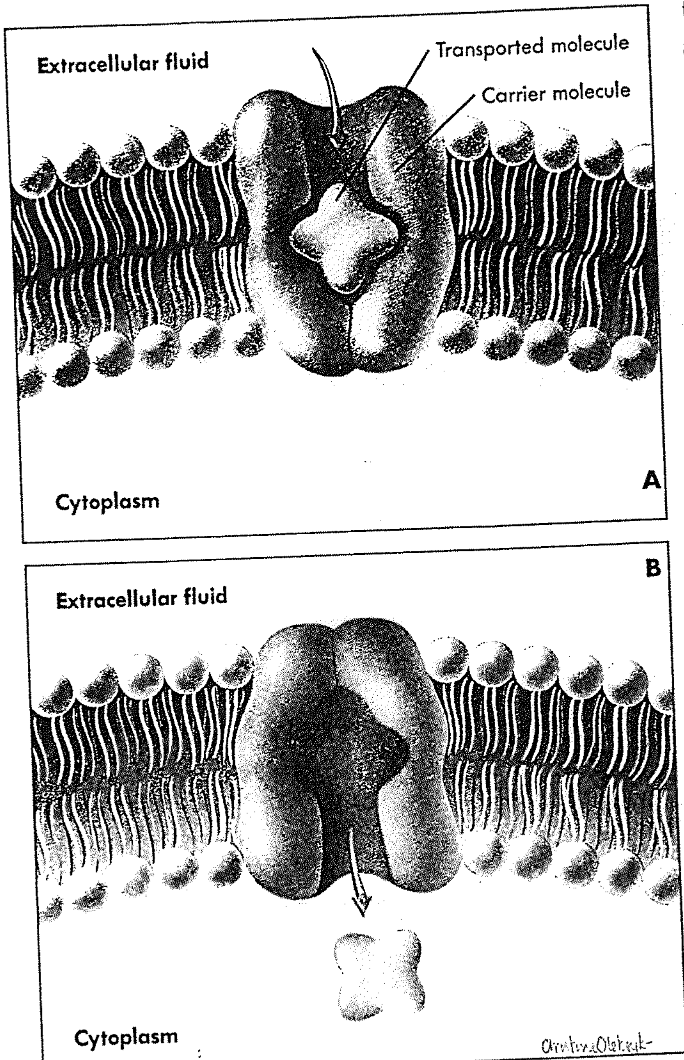


Fig. 13-6 Mediated transport by a carrier molecule. **A,** The carrier molecule binds with a molecule on one side of the plasma membrane and changes shape. **B,** The molecule is released on the other side of the plasma membrane.

BODY FLUIDS AND FLUID IMBALANCES

In the healthy body, homeostatic mechanisms maintain a constant balance between intake and excretion of water. The water gained each day approximately equals the water lost. The body gains water primarily by drinking fluids, by ingesting food containing moisture, and by forming water through the oxidation of hydrogen in food during the metabolic process. The body loses water through the kidneys as urine, through the feces, through the skin as perspiration, through exhaled air as vapor, and by the excretion of tears and saliva. Two abnormal states of body-fluid balance can occur. If the water gained exceeds the water lost, there is a water excess or overhydration. If the water lost exceeds the water gained, there is a water deficit or dehydration.

Dehydration

Dehydration may be classified as isotonic (excessive loss of sodium and water in equal amounts), hypernatremic (loss of water in excess of sodium), and hyponatremic (loss of sodium in excess of water).

Isotonic Dehydration

- Causes:
  - Usually, severe or long-term vomiting or diarrhea
  - Systemic infection
  - Intestinal obstruction
- Signs and symptoms:
  - Dry skin and mucous membranes
  - Poor skin turgor
  - Longitudinal wrinkles or furrows of the tongue
  - Oliguria (low urinary output—100 to 400 ml/24 hours)
  - Anuria (reduced urinary output—100 ml or less in 24 hours)
  - Acute weight loss
  - Depressed or sunken fontanelles in infants
- Treatment: intravenous infusion of an isotonic solution that has a solute concentration equal to that of blood

Hypernatremic Dehydration

- Possible causes:
  - Excessive use or misuse of diuretics
  - Continued administration of sodium in the absence of water intake
  - Excessive loss of water with little loss of salt
  - Profuse, watery diarrhea
  - Inhalation or ingestion of saltwater (for example, near-drowning), which may cause hypernatremia without dehydration

- Signs and symptoms:
  - Dry, sticky mucous membranes
  - Flushed skin
  - Intense thirst
  - Oliguria or anuria
  - Increased body temperature
  - Altered mental status
- Treatment: volume replacement with isotonic or occasionally hypotonic solutions (based on serum sodium levels and the clinical condition of the patient)

Hyponatremic Dehydration

- Possible causes:
  - Use of diuretics
  - Excessive perspiration (heat-related illness)
  - Salt-losing renal disorders
  - Increased water intake (for example, excessive use of water enemas)
  - Inhalation or ingestion of fresh water (for example, near-drowning) and compulsive water drinking, which may cause hyponatremia without dehydration
- Signs and symptoms:
  - Abdominal or muscle cramps
  - Seizures
  - Rapid, thready pulse
  - Diaphoresis
  - Cyanosis
- Treatment: intravenous fluid replacement with normal saline or lactated Ringer's solution; occasionally, hypertonic saline (for example, in seizures caused by hyponatremia)

Overhydration

Overhydration is an increase in body water with a decrease in solute concentration. This water excess may result from parenteral administration of excessive fluids, impaired cardiac function, impaired renal function, or some endocrine dysfunctions. Signs and symptoms of overhydration include the following:

- Shortness of breath
- Puffy eyelids
- Edema
- Polyuria (voiding a large volume of urine in a given time)
- Moist crackles
- Acute weight gain

The treatment of overhydration depends on the cause. For excessive water administration and certain endocrine problems, water restriction is the primary treatment. For patients with cardiac or renal impairment, a diuretic may be indicated. When profound hyponatremia is associated with overhydration (serum sodium level less than 120 mEq/L and associated seizures or altered consciousness), administration of saline may be indicated.

ELECTROLYTE IMBALANCES

In addition to the possibility of fluid imbalances, disturbances in the balance of electrolytes (other than sodium) may occur. These include potassium, calcium, and magnesium.

Potassium

Potassium is the major positively charged ion in intracellular fluid. The body must maintain a narrow range of normal values (serum level of 3.5 to 5 mEq/L) for normal nerve, cardiac, and skeletal muscle function. Obligate potassium losses (those that cannot be avoided) are usually minimal and are replenished through dietary intake. Excess potassium is usually readily excreted by the kidneys. Potassium plays an important role in muscle contraction, enzyme action, nerve impulses, and cell membrane function. Potassium imbalances interfere with neuromuscular function and may cause cardiac rhythm disturbances (including sudden death).

Hypokalemia (potassium deficit) can be caused by reduced dietary intake (rare), poor potassium absorption by the body, loss of gastrointestinal secretions as a result of vomiting or diarrhea, renal disease, infusion of solutions poor in potassium, and medications (most commonly diuretics, but steroids, theophylline, and others have also been implicated). Signs and symptoms of hypokalemia include the following:

- Malaise
- Skeletal muscle weakness
- Decreased reflexes

garment for more than 1 to 2 hours can lead to decreased tissue perfusion, ischemia of the underlying tissues (including the development of compartment syndrome[8]), and loss of the limb, even without underlying fracture.

Fluid Resuscitation in Shock

Almost every shock victim (with the exception of patients in cardiogenic shock) requires volume expanders as part of resuscitation. The selection of intravenous fluids for initial volume replacement varies according to medical control. In prehospital care, the most common emergency requiring fluid replacement is volume depletion secondary to hemorrhage or dehydration. The type of fluid replacement needed depends on the nature and extent of the volume loss (Box 13-5). The two main categories of fluids used in resuscitation are crystalloids and colloids.

Crystalloids

Crystalloid solutions are created by dissolving crystals such as salts and sugars in water. These solutions do not have as much osmotic pressure as colloid solutions and can be expected to equilibrate more quickly between the vascular and extravascular spaces. (Two thirds of the infused crystalloid fluid leaves the vascular space within 1 hour, so 3 ml of a crystalloid solution is needed to replace 1 ml of blood.) Examples of crystalloid solutions are lactated Ringer's solution, normal saline, and glucose solutions in water.

Hypertonic solutions have higher osmotic pressure than that of body cells and include 5% dextrose in 0.9% sodium chloride and 5% dextrose in 0.45% sodium chloride. Hypotonic solutions have a lower osmotic pressure than that of body cells (for example, distilled water and 0.45% sodium chloride [0.45% NaCl]).

Lactated Ringer's solution is generally considered the fluid of choice for resuscitating patients in shock. It is a well-balanced solution containing many of the chemicals found in human blood. Lactated Ringer's solution contains sodium chloride, small amounts of potassium and calcium, and 28 mEq of lactate, which may act as a buffer when metabolized by the liver. (Ringer's solution is also available without added lactate, which may be preferred by some physicians.) One third of the infused solution remains in the vascular space after 1 hour.

BOX 13-5

Fluid Resuscitation in Shock

Crystalloids
- Hypertonic sodium chloride
- Hypotonic sodium chloride
- Balanced salt solutions (isotonic)
  - Lactated Ringer's solution
  - Normal saline
- Glucose-containing solutions
  - D_5W

Colloids
- Blood
  - Typed and crossmatched
  - Type-specific
  - Packed red blood cells
- Plasma
- Plasma substitutes
  - Dextran
  - Hetastarch (Hespan)
  - Plasma protein fraction (Plasmanate)

Normal saline contains 154 mEq/L of sodium and has no buffering capabilities. Although preferred by some physicians, the higher chloride content of normal saline is generally considered less desirable than the more balanced lactated Ringer's solution. As in lactated Ringer's, nearly one third of the infused normal saline remains in the vascular space after 1 hour, making it an equally effective volume expander.

Glucose-containing solutions have immediate volume expansion effects, but the glucose leaves the intravascular compartment rapidly with a resultant free water increase. The volume-replacement benefits of glucose solutions may only last 5 to 10 minutes while the glucose is metabolized, so its use as a replacement fluid in volume deficits is inappropriate. Glucose solutions are most often used to maintain vascular access for administration of intravenous medications.

Colloids

Colloid solutions are those that contain molecules (usually protein) that are too large to pass through the capillary membrane. These solutions exhibit osmotic pressure and remain within the vascular compartment for a considerable time. Examples of colloid solutions are whole blood, plasma, packed red blood cells, and plasma substitutes. Whole blood, packed red cells, and plasma are generally reserved for in-hospital use.

Whole blood replacement is sometimes indicated after initial fluid resuscitation with a crystalloid solution in patients who have had a major loss of blood. Whole blood is drawn in a citrate solution to prevent clotting and is refrigerated until needed. According to blood bank regulations, the blood may be stored up to 3 weeks, but clotting factors and platelets deplete progressively. A type and crossmatch should be obtained (when possible) before a patient is given blood to determine the patient's ABO group and Rh type. The patient's blood must also be tested for red cell antibodies.

Packed red blood cells have been separated from the plasma component of blood by centrifugation. Like whole blood, packed red cells must be typed and crossmatched and may be refrigerated for up to 3 weeks. The advantage of packed red blood cells over whole blood is that the volume of hemoglobin per unit is almost twice that of whole blood. In addition, because there is no plasma, circulatory overload is less likely, transfusion reactions are less frequent, and transfusion hepatitis is less common.

Blood plasma is procured by separating the blood cells from the whole citrated blood. Blood plasma, which may be given without concern for ABO compatibility, contains fibrinogen, albumin, gamma globulins, hemoagglutinins (an agglutinin that clumps red blood corpuscles), prothrombin (a chemical that is part of the clotting cascade, the precursor of thrombin), other clotting factors, sugar, and salts. Blood plasma is sometimes used to restore effective blood volume in circulatory failure associated with burns, traumatic shock, and hemorrhage. It is also used to correct clotting deficiencies.

Although plasma substitutes do not replace red blood cells or plasma protein, they are used to restore circulating blood volume as an emergency treatment for hypovolemia caused by blood loss. Plasma substitutes such as dextran, plasma protein fraction, and hetastarch have osmotic properties similar to those of plasma. Plasma substitutes do not carry the human immunodeficiency virus (HIV) or hepatitis viruses, do not require type and crossmatching before administration, and are readily available. Accordingly, their use is appealing, particularly in mass casualty situations when blood products are scarce. They do have adverse effects, however, most notably increasing bleeding tendencies and immune suppression. Plasma substitutes may be carried on emergency vehicles, but expense and storage problems make them impractical for general use in the prehospital setting.

NOTE:
Several types of blood transfusion reactions may occur during or up to 96 hours after infusion. Symptoms may range from mild fever to life-threatening shock. If a reaction is suspected, the transfusion should be stopped, and medical direction should be contacted.

Key Principles in Managing Shock

1. Establish and maintain an open airway.
2. Administer high-concentration oxygen, and assist ventilation as needed.
3. Control external bleeding (if present).
4. By order of medical control or per protocol, initiate intravenous fluid replacement if appropriate. It is usually recommended that two large-bore intravnous lines of a volume-expanding fluid be established in cases of hypovolemia. *The administration of intravenous fluids in the prehospital setting should not delay patient transport,* since crystalloid solutions cannot restore the oxygen-carrying capacity of blood. Generally, the patient is best served

by rapid assessment, airway stabilization, immobilization, and rapid transportation to an appropriate medical facility. Many EMS authorities recommend that intravenous therapy for shock resuscitation be initiated en route to the hospital.

5. Consider use of PASG (per protocol), especially if transport time is long or a patient is deteriorating after intravenous therapy has been initiated.
6. Maintain the patient's normal body temperature. Patients in shock are often unable to conserve body heat and easily become hypothermic.
7. In the absence of spinal or head injury, position the patient in the modified Trendelenburg position (legs elevated 15 to 18 inches).
8. Monitor cardiac rhythm.
9. Frequently assess vital signs en route to the emergency department.

Management of Specific Forms of Shock

In addition to the general management appropriate to all victims of shock, there are certain management guidelines specific to each etiological classification.

Hypovolemic Shock

The treatment of hypovolemic shock is not considered complete until the circulatory deficit and its cause or causes are corrected. This may include crystalloid fluid replacement in cases of simple dehydration or volume replacement resulting from hemorrhage, definitive surgery, critical care support, and postoperative rehabilitation.

Cardiogenic Shock

The treatment of cardiogenic shock is directed toward improving the pumping action of the heart and managing cardiac rhythm irregularities. Fluid resuscitation in the adult should be initiated with a fluid challenge of 100 to 200 ml of a volume-expanding fluid. If the patient improves, fluid therapy should continue until the blood pressure stabilizes and the pulse decreases. Lung sounds should be assessed frequently. If the patient shows signs of increased lung congestion, the rate of infusion should be adjusted to keep the vein open.

Drug therapy for cardiogenic shock varies according to cause and may include vasopressors, inotropic drugs, and antidysrhythmics (see Chapters 14 and 18).

Neurogenic Shock

The treatment of neurogenic shock is similar to the treatment for hypovolemia. However, care must be taken during fluid therapy to avoid circulatory overload. Throughout the resuscitation phase, the patient's lung sounds should be closely monitored for signs of pulmonary congestion. In addition, patients in neurogenic shock may respond to the administration of vasopressors.

Anaphylactic Shock

Subcutaneous administration of epinephrine is the treatment of choice in acute anaphylactic reactions. Depending on the severity of reaction, other treatment modalities may include oral, intravenous, or intramuscular administration of antihistamines. Bronchodilators may also be indicated to treat bronchospasm, and steroids may be given to reduce the inflammatory response.

Crystalloid volume replacement is also indicated to compensate for the increased container size caused by vasodilation that results from histamine release during an anaphylactic reaction. Paramedics should anticipate the need for aggressive airway management in any allergic reaction (see Chapter 22).

Septic Shock

The treatment of septic shock in the prehospital setting may include the management of hypovolemia (if present) and the correction of metabolic acid-base imbalance. Depending on the patient's response to the infection, prehospital care may involve fluid resuscitation, respiratory support, and the administration of cardiac medications to improve cardiac output. If possible, the paramedic should obtain a thorough patient history to help identify the septic focus. There is an increased risk of septic shock in any immunocompromised group such as those with HIV infection, some cancer patients receiving chemotherapy, and patients with indwelling urinary or vascular catheters.

with hypovolemic shock. The signs used to assess compensated and decompensated hypovolemic shock are presented in Table 6-3.

An important point to remember in the assessment of patients with multiple trauma is that head injuries do not cause hypotension until the cerebellum begins to herniate through the incisura and foramen magnum. Therefore, if a patient with a head injury is found to be hypotensive, the EMT should assume that this condition is due to hypovolemia (usually blood loss) from other injuries and not from the head injury.

MANAGEMENT

OVERVIEW

At the time of publication of the fourth edition of the PHTLS course, the management of the patient in hemorrhagic shock is controversial. There are advocates of three types of treatment:

1. Maintain hypotension in the patient by restricting fluid replacement and avoiding the use of any type of medication or device that will increase the blood pressure until the patient's hemorrhage has been controlled.
2. Overhydrate the patient by replacing the lost whole blood one for one with blood and then the addition of 3:1 blood loss with Ringer's lactate. Resuscitation should be to normal blood pressure.
3. Hydrate the patient with hypertonic solutions. Resuscitation should be to normal blood pressure.

The basis of this controversy is the effect of improved blood pressure on increased bleeding versus the physiologic benefits of good tissue perfusion with oxygenated RBCs.

Table 6-3 Shock assessment in compensated and decompensated hypovolemic shock

	Compensated	Decompensated
Pulse	Increased, tachycardia	Markedly increased, marked tachycardia; can progress to bradycardia
Skin	White, cool, and moist	White, waxy, and cold
Blood pressure	Normal range	Decreased
Level of consciousness	Unaltered	Altered, ranging from disoriented to coma

A review of the physiology as it pertains to fluid resuscitation follows:

- Improved blood pressure increases the blood flow into the organs.
- The positive side of fluid resuscitation is that increased flow improves the oxygenation of the tissue by delivery of RBCs to the tissue.
- The negative side of fluid resuscitation is that as blood pressure is increased to the area of uncontrolled hemorrhage, additional whole blood is lost.
- Initial resuscitation using Ringer's lactate or saline replaces only volume and does not replace the lost RBCs.
- Fluid replacement without RBC replacement lowers the RBC mass and therefore lessens ability of the circulation fluid to carry oxygen.
- The increased bleeding produced by restoration of blood pressure brings about more RBC mass loss.
- Any device, medication, or fluid that increases the blood pressure without increasing the lost red cell mass will produce the same condition. This includes the PASG, vasopressor medications, hypertonic saline/dextran fluids, and crystalloid fluids.

There is, at the time of the publication of this chapter, no data in the literature that demonstrates, to statistical significance, that in any condition, except uncontrolled hemorrhage in the chest, that any of the just mentioned materials (except vasopressor medications) alter outcome. Vasopressor indications decrease the outcome. There are proponents of all arguments, but careful reading of the published literature is inconclusive. It is therefore important that the provider of prehospital care understands the physiology well and makes management decisions based on this knowledge.

There is early information that trauma patients do better with a lower pressure. In *Resuscitation and Anesthesia for Wounded Men*—edited by Henry K. Beecher, published in 1949, and based on World War II data—the following statements are present: "In the treatment of shock preliminary to surgery we can restore blood volume and blood pressure to normal. This is possible but is it necessary? We do not need to guess here . . . It is no . . . The patient will not suffer as long as the systolic pressure is 80 mm Hg and the skin is warm and of good color. Neither will he lose as much blood by renewed bleeding as he will if plasma is used to raise blood pressure higher than necessary during this waiting period."

The management of a patient in shock is directed toward changing the anaerobic metabolism back to aerobic metabolism. Based on the Fick principle, this reversal is produced by delivering more oxygen to the ischemic tissue cells. Improved oxygenation at the cellular level is achieved by oxygenating the RBCs and delivering them to the tissue cells. Without improving the oxygenation and delivery of RBCs, the patient will continue to deteriorate rapidly until reaching the ultimate stabilization—death.

In addition to these first two treatment objectives (oxygenation and delivery), the third objective is to reach definitive care as soon as possible for hemorrhage control and replacement of lost RBCs.

The EMT must ask four questions when deciding what treatment to provide for a patient in shock:

1. What is the cause of the patient's shock?
2. What is the definitive care for the patient's shock?
3. Where can the patient best receive this definitive care?
4. What interim steps can be taken to manage the patient's condition while he or she is being transported to definitive care?

Although the first question may be difficult to answer with a high degree of diagnostic accuracy, having some idea of what is occurring can help the EMT identify the hospital best qualified to meet the patient's needs and to decide what steps to take during transport to improve his or her condition.

AIRWAY

The airway should be evaluated initially in all patients. Patients who are not breathing or have obvious airway compromise, ventilatory rates in excess of 20 breaths per minute, or noisy sounds of respiration should receive immediate management of their airway. Specific techniques are discussed in Chapter 3: Airway Management and Ventilation and Chapter 4, the associated skills chapter.

VENTILATION

When the airway is open, patients in shock or those susceptible to shock (which includes almost all trauma patients) should receive oxygen with an FiO_2 as close to 1.0 (100% oxygen) as possible. This kind of oxygenation can only be achieved with a device that has a reservoir attached and with a high flow rate of oxygen to the device.

It is no longer correct to talk of giving the patient *x* liters of oxygen or "high flow oxygen." Instead, the EMT should discuss giving the patient an FiO_2 of some defined number. This is achieved by providing a specific flow rate to a specific device. Any device will deliver varying FiO_2s depending on the oxygen flow rate and the design of the device.

A shock patient or a potential shock patient should only be given oxygen with a device that includes a reservoir, so that an FiO_2 as close as possible to 1.0 can be achieved. Nasal prongs cannot deliver anything close to this concentration and therefore should not be used on a patient in shock (see Table 3-1, page 71). If the patient is not breathing, or is not breathing with an adequate depth and rate, ventilatory assistance using a bag-valve-mask unit should be instituted immediately.

CIRCULATION

Difficulties with circulation as identified by decreased capillary refill time, tachycardia, decreased or absent palpable pulses (radial, femoral, or carotid), and pale or cyanotic skin color are treated in several steps.

The first step is to reestablish as much circulation as possible to the brain and to increase the cardiac preload. Placing the patient in the **Trendelenburg position** (head down, with feet elevated) is the initial step for simple fainting. This position is most appropriate for the treatment of psychogenic shock only. It may be the initial step for the management of any type of shock. This position will increase the venous pressure and potentially increase edema in the injured brain. However improved blood flow and oxygenation to the brain is more important initially than is the potential damage done by increased edema.

CONSERVATION OF BODY HEAT

Maintaining the patient's body temperature within a normal range is important. Hypothermia introduces myocardial dysfunction, coagulopathy, hyperkalemia, vasoconstriction, and a host of other problems that negatively affect survival rate. It is very difficult to increase the core temperature once hypothermia has started; therefore, all steps that can be taken in the field to preserve normothermia must be initiated. These steps include the use of warm (104° F [40° C]) intravenous fluids, warm blankets, heated humidified oxygen, and a warm environment for the patient by moving him or her rapidly from the scene into the warm patient compartment of the unit. Any cold, wet clothes should be removed once inside the unit (see Chapter 12: Thermal Trauma).

Although cold does preserve tissue for a short time, the temperature drop must be very rapid and very low for preservation to occur. Such a rapid change is not the correct management for the patient in shock; therefore, body temperature must be preserved to avoid jeopardizing the patient further. Covering a patient with thick plastic sheets (such as heavy-duty garbage bags) or blankets to retain heat and keep the patient dry and placing the patient in a warm environment are important in achieving heat preservation.

Although blankets have been used traditionally, easily available plastic sheets such as 3-mil thick garbage bags are inexpensive, small, easily stored, disposable, and highly efficient devices for heat retention. Fluids, particularly intravenous fluids, given to the shock patient should be warm, not cold. The ideal temperature for such fluids is 104° F (40° C). Most ambulances do not have conventional rapid fluid warmers, but other steps can keep fluids at an adequate temperature. The fluids should not be stored in an air-conditioned compartment, nor should the patient be kept in such a compartment. A convenient storage area for fluids is in a box in the engine compartment. Fluid can also be warmed by wrapping heat packs around the bag. There are commercially available fluid warmer boxes for the patient care compartment, too. The patient compartment should be kept at 85° F (29° C) or more when transporting a severely injured trauma patient.

It is critical that the conditions should be ideal for the patient, not for the EMTs. The patient is the most important person in any emergency. Consider for a moment how you would feel swimming in a pool with a temperature at 72° F (22° C). Very rapidly you would become cold and start to shiver. The patient is in this same situation in a compartment that is air conditioned to 72° F (22° C). The patient has had all or most of his or her clothes removed; the lactated Ringer's that is being run rapidly into his or her veins, heart, and lungs are the same temperature as the compartment. The patient's rate of heat loss into the cold compartment is very high, and the cold fluid is cooling him or her faster. It is easy to cool a patient but very difficult to warm the patient up again.

Hypothermia is the third most serious condition of a trauma patient, ranking close to hypoxia and hypovolemia. It has two causes:

- Anaerobic metabolism results in a reduction of the energy production and the body cannot keep up with heat loss to the outside.
- There is a loss of heat between the body of the patient and the colder environment. This includes the introduction of cold air and fluids into the patient.

Providing warm, humidified oxygen for the patient to breathe and covering the patient with warm blankets or plastic sheets after completing the physical examination are also means for conservation of body heat.

Rapid transportation to a facility that is capable of managing the patient's condition is extremely important. Rapid transport does not mean disregarding or neglecting the treatment modalities that are important in patient care, nor neglecting proper immobilization of trauma patients. It does, however, indicate that such modalities are to be employed rapidly and that time must not be wasted with an inappropriate assessment or with unnecessarily complicated stabilization maneuvers. Many steps, such as warming the patient, starting intravenous therapy, and even performing the focused physical examination, can be accomplished in the ambulance while en route to the hospital.

PNEUMATIC ANTISHOCK GARMENT (PASG)

The PASG, as it has come to be known generically, is a controversial device because its exact role in prehospital care is still not definitively decided. Despite this controversy, it should not be neglected as a prehospital patient treatment tool. Like other medical devices, it should only be used when indicated. Specifically, it is helpful for patients requiring control of blood loss, management of pelvic instability, or long transport times. It may be detrimental to patients with penetrating thoracic trauma or short transport times. It remains contraindicated in patients with pulmonary edema, traumatic diaphragmatic herniation, or known hemorrhage above the diaphragm.

Physiology

Pressure applied by the PASG to the legs and abdomen is transmitted directly through the skin, fat, muscle, and other soft tissue to the blood vessels themselves. The vessels are compressed, and their lumen (internal opening) is reduced in size. The physiologic result is twofold. The vascular container in body areas beneath the device is made smaller, increasing the systemic vascular resistance and thereby raising the systolic and diastolic pressures. The remaining fluid can be distributed and better used in the noncompressed upper half of the body.

Hemorrhage

Compression over a bleeding site is the classic method of hemorrhage control. Effective compression can be achieved with the PASG for body areas such as the abdomen, pelvis, and the legs. The PASG is just a large pneumatic splint. It should be used to control blood loss and stabilize fractures with the same indications as a pneumatic splint.

Increased bleeding in the noncompressed portions of the body is a potential complication of inflating the PASG. The rate of hemorrhage from an open wound is proportional to the blood pressure inside the injured vessel minus the external pressure on the vessel. Less bleeding occurs with a low blood pressure than with a high blood pressure. Open vessels in the upper half of the body may well increase the rate of blood loss when the patient's blood pressure and blood flow are significantly improved, as when the PASG is applied.

Blood Pressure

Reevaluation of several studies indicates that patients with blood pressures below 50 to 60 mm Hg have a better outcome when the PASG is used than when it is not. The increased perfusion of the brain and heart that is provided by the increased vascular resistance in the lower extremities and abdomen is beneficial to such patients.

Immobilization of Fractures

Like any pneumatic splint, the PASG can also be used for fracture immobilization. The two major bones that the PASG immobilizes most effectively are the pelvis and the femur. Because hemorrhage is recognized as a major potential problem with fracture of either of these bones, compression with the PASG provides an extra benefit beyond just immobilizing the fracture. Use of the PASG solely as a splint for isolated lower extremity fractures where shock is not present or expected, however, is not recommended.

Application

The PASG is applied to the patient as quickly as possible and without delay when indications for its use in hemorrhage control or fracture management are present. The PASG is positioned under the patient in one of a number of ways. In many cases, it may be simpler to place the patient on the device (as when moving the patient onto a long board) rather than lifting the patient and inserting the garment beneath the patient.

The PASG is then securely and snugly fastened and inflated. When the Velcro® begins to crackle, between 60 and 80 mm Hg of PASG pressure (not blood pressure) has been achieved.

Deflation

Prehospital deflation of the PASG should not be done except in extreme extenuating circumstances, such as evidence of a diaphragmatic herniation, and even then only with on-line medical direction.

When deflation is necessary, it should be preceded by assessing the patient and confirming that vital signs are within normal limits. Even with vital signs within normal limits, the patient's blood volume might still be depleted: the inflated PASG has reduced the size of the patient's container, and the available blood volume may just fill the artificially reduced container. As the PASG is deflated, the patient's container size will increase. Unless a sufficient amount of fluid is present in reserve, cardiac preload will decrease, systemic vascular resistance will drop, and the patient's blood pressure and level of perfusion will deteriorate rapidly.

INTRAVENOUS FLUID REPLACEMENT

Fluids that are given to the patient fall into four basic groups: (1) water only, (2) water and electrolytes, (3) water and protein or protein substitutes such as colloids, and (4) RBCs. There are other specialty fluids that are used in the hospital, particularly fluids with large amounts of nutrients such as glucose and amino acids, or those that contain blood products such as fresh frozen plasma, platelets, or the various coagulation factors. Perhaps within the next 5 to 10 years, fluids with oxygen-carrying capability such as stroma-free hemoglobin will be available for prehospital use. At the present time, however, no prehospital fluids are capable of carrying oxygen; they are only volume expanders. Dilution of the remaining blood in the patient by the introduction of volume expanders reduces the percentage of RBCs in the vascular space and therefore the oxygen-carrying capability of the blood. In most trauma patients, a loss of RBCs has already occurred and will remain low until the site of blood loss has been secured.

The definitive care of these patients requires replacement of the RBCs. Even if intravenous access and volume replacement are accomplished in the field, it is extremely important that the patient be delivered as quickly as possible to a facility that can immediately replace the lost RBCs to improve the patient's oxygen-carrying capacity. The facility should have immediate operating room access where hemorrhage can be rapidly controlled.

Water Solutions

Solutions that contain only water and glucose (as opposed to solutions that also contain electrolytes), although isotonic when administered, can be detrimental because the oncotic pressure of the vascular space is reduced by these fluids. For example, 5% dextrose in water (D5W) goes into the body as an isotonic fluid and remains isotonic in its first passage through the vascular system. However, as the glucose is metabolized only water is left behind, reducing the oncotic pressure of the vascular space. Because rapid exchange occurs between the interstitial fluid and the vascular space, interstitial oncotic pressure is likewise reduced. To equalize the oncotic pressure between the extracellular and intracellular fluid, water flows from the extracellular space into the intracellular space. This causes the tissue cells to swell and produces several adverse effects.

Cellular edema, particularly if it occurs in an enclosed space like the skull, puts pressure on the surrounding vessels that reduces the vascular size, especially in the capillary beds. The reduction in vascular size reduces blood flow, therefore reducing the oxygen replenishment capacity. The reduced oxygen replenishment capacity, in turn, increases cerebral ischemia and causes a shift from aerobic to anaerobic metabolism, producing cellular acidosis and more swelling of the cells.

This is a vicious cycle which, although most dramatic in the enclosed skull, also occurs in other organs as well. Some organs are more constrained than others. The kidneys, for example, are each contained in a tight capsule. Swollen cells cannot stretch the capsule; therefore, renal oxygenation may be compromised. The same can occur with the muscles of the extremities that are enclosed in strong fascial compartments.

Crystalloid Solutions

Crystalloid intravenous solutions are isotonic and remain isotonic; therefore, they act as effective volume expanders for a short period of time. Both the water and the electrolytes in the solution can freely cross the semipermeable membranes of the vessel walls (but not the cell membrane), and therefore achieve equilibrium in 2 to 3 hours. The added volume in the vascular space readily translocates to the interstitial space. For a short period, the vascular space is filled by crystalloid intravenous solutions, improving preload and cardiac output. This solution has no oxygen-carrying capacity and contains no protein; therefore, blood must be used eventually to replace the volume that has been lost by the patient.

Rapid infusion of crystalloids such as lactated Ringer's should be used initially for vascular volume replenishment and followed as quickly as possible with blood. A general rule of thumb is that initial crystalloid replacement should not exceed 3 liters before whole blood is instituted. This does not mean that a patient who is severely hypovolemic from acute blood loss should not be given adequate amounts of lactated Ringer's. It does mean that blood should be administered early during the patient's resuscitation. If it is necessary to give the patient more than 3 liters of crystalloid to maintain adequate preload and cardiac output because blood is not available, the detrimental results of such replacement must be accepted.

One hour after administration of a crystalloid solution, only one-third remains in the cardiovascular system. The rest has shifted into the interstitial space. Excessive interstitial fluid shows up as edema. One of the organs most affected is the lungs. The pathologic process of increased pulmonary fluid into the interstitial space is known as pulmonary edema. The solution to this dilemma is rapid transportation to a hospital that has the capability of caring for critical trauma patients.

Colloid and Plasma Substitutes

Administration of human protein to a patient has several drawbacks. Non-A and non-B hepatitis (mostly hepatitis C) are present in 3% of these fluids. The fluids are also expensive and have short shelf lives. "Look-alike" protein molecules that stay in the bloodstream with an increased oncotic pressure are more expensive than lactated Ringer's, require some special techniques in administration, and have some complications such as increased bleeding time and anaphylactic reactions. Recent studies using a combination of hypertonic saline and a colloid (dextran) have proven effective in increasing a patient's blood pressure. However, this increased blood pressure, as explained earlier, may increase hemorrhage and can actually decrease the survival rate. These consequences may or may not have prehospital application; further studies are still needed.

A synthetic colloid solution (Hetrostarch, Hespan) is frequently used as a resuscitation fluid outside the United States.

VASCULAR ACCESS

There are two reasons for obtaining vascular access on any patient in the prehospital phase of care: volume replacement and medication administration. A third, in-hospital reason is to monitor cardiac functions. Whether vascular access is obtained through peripheral or central intravenous lines or through intraosseous infusion, there are several commonalities of use.

The size and location of intravenous line placement are based upon the indication for its use. The rate of fluid administration is directly proportional to the bore of the needle and inversely proportional to its length. When fluid replacement is required, a large bore, short catheter should be used, as it will pass a larger volume than will a smaller, longer tube.

The ideal site for access in a patient requiring fluid replacement is a large vessel in the forearm. The desirable catheter to use is a 14 or 16 gauge and just over 1 inch in length. For administering medications, the chosen vein does not need to be as large, and an 18 or 20 gauge needle will suffice. In a trauma patient, it is best to start a large bore, peripheral intravenous line (preferably two lines). Central intravenous lines are generally not needed in the field or in the emergency department for fluid replacement.

• Summary •

1. Shock has many descriptions, but the most correct is that of cellular anaerobic metabolism.
2. Survival depends on oxygen delivery to the cells. This is accomplished by oxygenation of the red blood cells in the lungs and delivery of the red blood cells to the tissue cells (Fick principle).
3. Getting oxygen into the lungs is a top priority in the management of the shock patient.
4. Besides adequate oxygenation and ventilation, the patient also requires rapid transportation to a facility where hemorrhage can be controlled and blood loss can be replaced.
5. The PASG can assist in controlling hemorrhage by compressing the abdomen and pelvis to control bleeding in those areas while maintaining perfusion through the heart, brain, and lungs. It cannot and should not be used for prolonged periods as the only management technique employed.
6. Fluid replacement is also an important component to prehospital management of shock.

7. Crystalloid solutions function as volume expanders, but they do not have oxygen-carrying capabilities and therefore are not the ideal fluid for replacement. The ideal fluid for replacement of lost blood is blood.
8. The diagnosis of shock is made by recognizing the pathophysiology it produces. Tachycardia, tachypnea, increased capillary refilling time, decreased level of consciousness, and decreased blood pressure are the prehospital findings that indicate hypoperfusion of the various organs and the body's general demand for greater oxygenation.

Scenario Solution

In the scenario at the beginning of the chapter, you encountered a patient with multisystem trauma. Based on the signs and symptoms listed next, you should have concluded that the patient is experiencing spinal shock.

Signs and symptoms include a normal level of consciousness; strong, but slow pulse; warm and dry skin; normal capillary refill; and hypotension.

The management of this patient should include oxygenation, spinal immobilization, rapid transport, and judicious use of intravenous fluids.

Review Questions

Answers provided on page 333.

1 Which of the following statements are correct? (Choose as many as are applicable.)
  A Perfusion is the process of blood passing by tissue cells.
  B Hypoxia is a condition in which cell oxygen requirements are being met.
  C Hypoperfusion is inadequate blood passing by tissue cells.
  D Hypovolemia is a condition occurring when fluid volume is too high.

2 The cellular physiologic process of shock can be divided into all of the following phases, *except:*
  A the ischemic phase
  B the stagnant phase
  C the deceleration phase
  D the washout phase

3 Which of the following is a sign and symptom of shock?
  A warm, dry skin temperature in spinal shock
  B falling blood pressure during compensated shock
  C normal pulse during septic shock
  D pink skin color in hypovolemic shock

4 All of the following statements are true regarding the pneumatic antishock garment (PASG) *except:*
  A Increased vascular resistance in the lower extremities and abdomen is beneficial to patients with systolic blood pressure between 50 to 60 mm Hg.
  B Anterior compartment syndrome is a complication associated with prolonged use of the PASG.
  C The PASG should be inflated until the patient's systolic blood pressure reaches 120 mm Hg.
  D PASG deflation in the field should only be done with on-line physician direction.

5 Which of the following is a reason for intravenous fluid replacement?
  A The PASG is applied, but not inflated.
  B The patient has pulmonary edema.
  C There is a need to replace lost red blood cells with crystalloid solution.
  D There is a need to provide a short-term volume expander.

6 Which of the following statements about the management of shock is true? (Choose as many as are applicable.)
  A Rapid transport is critical when confronted with continuing hypoperfusion.
  B Blood replacement and hemorrhage control are the definitive care for blood loss.
  C Colloid and plasma substitutes have several drawbacks including higher expense, possible contamination with hepatitis, and anaphylaxis.
  D The preferred isotonic solution for the management of a shock patient is 5% dextrose in water (D5W).

7 Which of the following statements is incorrect?
  A Severe internal hemorrhage can be adequately controlled in the prehospital setting without the need for direct surgical control.
  B The IV catheter used for a shock patient should be 18 to 20 gauge.
  C PASG is a short-term treatment modality for shock.
  D A and B

the problem is recognized early and surgically treated (draining excess fluid from within the cranial vault), the mortality rate remains between 50% to 60%.

Subacute subdural hematomas display signs and symptoms between 3 and 21 days after the insult has occurred. These hematomas also result from high-velocity type injuries but develop more slowly than acute hematomas. This slow development reflects less brain tissue involvement and damage with a better prognosis. The mortality rate for subacute hematomas is approximately 25%.

Chronic subdural hematomas may present weeks or months after a seemingly minor head injury. The smaller vessels that bridge the subdural spaces are torn and bleed more slowly. Because of the slowness in the onset of signs and symptoms, chronic subdural hematomas may go unnoticed. Although slow in onset, these types of hematomas nevertheless cause an increase in intracranial pressure, pressure on the brain tissue, and decreased cerebral perfusion. The mortality rate for chronic subdural hematomas is almost as high as that for acute subdural hematomas, approximately 50%.

The EMT should suspect a possible subdural hematoma postincident, regardless of the time lapse, if the patient exhibits any of the following signs or symptoms at the present time or any time since the injury:

- changes in level of consciousness, including unconsciousness or coma
- confusion or disorientation
- persistent or recurring headache
- blurred vision, double vision, or other affected vision
- nausea or vomiting
- personality changes, including changes in temperament or "out-of-character" behavior
- hemiparesis, hemianesthesia, or hemiparalysis
- slurring or other types of speech impediment

Prehospital management may be called for in a patient who is displaying strange behavior with no rational explanation for his or her actions. While taking the patient's past pertinent history, the EMT may find that the patient suffered a minor head injury in the past. This event may have taken place days, weeks, or even months ago. The patient may not have gone to the emergency department for treatment, or if he or she did, the physical and radiographic examinations may have been normal upon discharge.

Intracerebral Hematoma

Lacerations or tearing of blood vessels within the brain tissue itself can also produce hemorrhage and hematoma formation. This group of hematomas is referred to as **intracerebral hematomas.**

Intracerebral hematomas can occur with penetrating head trauma or when sudden decelerating head injuries drag the brain across the bony outcroppings within the cranial vault area, causing injury to the brain tissue itself.

Seizure activity is one commonly seen sign with this type of head injury. Focal signs and symptoms of intracerebral hematomas depend on the area of the brain involved.

MANAGEMENT

Prehospital management of any patient with a suspected head injury should focus on maintaining cerebral perfusion (adequate oxygen and cerebral blood flow). Cerebral perfusion can be accomplished by maintaining the patient's blood pressure and providing oxygen to the hypoperfused cells through the use of hyperoxygenation (high-flow oxygen). The patient's spine must also be protected and the patient rapidly transported to a hospital that is capable of providing definitive treatment.

As with any patient, the airway must have first priority. The head trauma patient may require the insertion of an endotracheal tube to maintain and protect the airway. Because it must be assumed that the patient also has a cervical spine injury, in-line stabilization must be maintained while establishing and maintaining the airway. Intravenous lidocaine (1 mg/kg) has been administered immediately prior to intubation based on animal research showing that lidocaine can blunt the increase in ICP that occurs during the act of intubation.

The head injury patient is also likely to vomit. Suction equipment, including large bore catheters such as the tonsil-tip, should be readily available. The patient must be well secured to a long backboard in case it becomes necessary to quickly turn the patient onto his or her side to prevent aspiration.

Once the head and neck are manually stabilized, the airway controlled, and adequate ventilation established, bleeding can then be controlled and circulation reestablished. Bleeding scalp vessels are easily compressed by gentle, continuous direct pressure. If there is obvious deformity or palpable bony defect or instability, the bleeding can be controlled by compressing the area around the wound while taking care to press against a stable area of the skull. Bleeding from the patient's nose or ears presents still another challenge. While the blood loss needs to be controlled, complete tamponade of the blood flow may result in increased intracranial pressure and cause further brain tissue damage. These areas should be covered with a clean dressing but allowed to "leak" slightly. Hypovolemic shock may occur as a result of gross bleeding since the face and scalp are highly vascular in nature.

If the adult head trauma patient has not experienced excessive bleeding from the face, scalp, or other injuries, shock is rarely due to brain injury unless it is the terminal event. In children, because of their normal lower blood volume, hypotension can be more threatening than in the adult.

The multiply injured patient with head trauma who is in shock should be managed like any other trauma patient in shock. Management should include control of any major hemorrhage, use of the pneumatic antishock garment (PASG) as indicated, and body heat retention. The patient may need fluid resuscitation, which is another reason for rapid transportation to the closest appropriate hospital or for interception with an advanced life support (ALS) unit capable of providing it. The injured brain must be perfused with oxygenated blood under adequate pressure to survive. However, the patient with an isolated head injury needs only maintenance fluids to help minimize cerebral edema. An intravenous line using lactated Ringer's or normal saline (local protocol will dictate which to use) should be initiated with the head trauma patient. If the vital signs are adequate, the intravenous line should be maintained at a maintenance rate of no more than 125 cubic centimeters (cc) per hour and monitored closely to prevent overhydration. If signs of hypovolemic shock appear, the intravenous flow can be adjusted to help maintain pressure.

Surgery to decrease intracranial pressure cannot be performed in the field. The method that can be used to help decrease intracranial pressure and brain stem herniation is hyperventilation. It is well documented that hypoxia and hypercarbia will aggravate or increase brain tissue swelling. The use of 100% oxygen will cause cerebral vasoconstriction and subsequently decrease the intracranial pressure, which will improve cerebral oxygenation. In the deteriorating head injury patient with signs of herniation, a $PaCO_2$ of 30 to 35 mm Hg or lower is desirable. This can be achieved by increasing the patient's oxygen concentration and rate of ventilation from the normal 12 to 16 breaths per minute to a rate of 20 to 24 breaths per minute. It is also essential for the EMT to allow adequate time between each assisted inhalation for exhalation to occur. Failure to allow for adequate exhalation will cause the patient to retain CO_2, thus causing increased intracranial pressure.

A variety of medications such as diuretics (mannitol, Lasix®, etc.) can be used to draw fluid from the interstitial and intracellular spaces in the head injured patient who is showing signs of increased intracranial pressure. Reduction in fluid can decrease cerebral edema and thus decrease the intracranial pressure. These diuretic-type drugs do work well, but like hyperventilation, they are only temporary agents. While causing a decrease in cerebral edema, circulating blood volume, and intracranial pressure, they may also allow for a more rapid expansion of an intracranial hematoma. Because of this potential danger, the use of diuretics and a wide range of other medications should be limited to in-hospital use with the head trauma patient; they should not be administered in the prehospital setting.

Treatment of head injured patients is focused on maintaining cerebral perfusion and oxygenation. Treatment efforts are directed at maintaining the patient's blood pressure, providing supplemental oxygen, and initiating care for increased intracranial pressure when clinical signs are present.

TRANSPORTATION

Not only is it important in prehospital management to transport the suspected head injury patient to the appropriate facility, the EMT must also notify and alert the facility about the patient's type of injury. An example of a radio transmission to alert the receiving facility of a head injury patient might go like this:

> Base, this is unit 224, we are transporting to your facility a 26-year-old male patient who was the driver of a motor vehicle involved in a high-speed, sudden deceleration incident. The patient was unrestrained during the incident and was unconscious at the scene upon our arrival. The patient's vital signs are as follows: B/P 124/80 initially, last pressure 138/80; pulse 62 and regular; respirations 38, irregular and shallow; pupils unequal with the right being larger than the left and slow to react to light. The patient's Glasgow Coma Scale is 5 with a score of eyes—1, motor—3, and verbal—1. The patient also responds with flexion on his right side and does not move his left side. The patient is intubated and ventilations are being assisted at a rate of 30 times per minute with a bag-valve device and 100% oxygen. The patient is also secured to a long backboard with head and neck immobilization. We have an ETA to your location of 7 minutes.

This radio traffic should alert the receiving facility that a potential head injury patient is en route, and preparations should be initiated for his arrival and subsequent treatment.

It must be stressed how important it is for the EMT to maintain an accurate record of the suspected head injury patient's vital signs and examinations performed, both at the scene and during transport. Information such as which pupil was "blown" first or any changes in the patient's condition can be key information and helpful in determination of the course of treatment necessary.

The suspected head injury patient should be transported with the head elevated whenever possible to help reduce brain tissue swelling. Elevating the head can be done while still maintaining adequate spinal immobilization by simply elevating the head end of the long backboard. Time is of the essence for the head trauma patient, but so is transporting the patient safely and rapidly to a facility that is capable of providing adequate neurologic care.

fractures—known or as yet unknown—that may be present.

- Once immobilized, the patient needs to be rapidly transported to the hospital. Unless the patient is trapped, there should be no reason for field management of an unstable trauma patient to require more than 10 minutes.
- During transport the patient should be further assessed from head to foot, and additional necessary care should be administered.

The patient must also be continually **reassessed,** since conditions that did not initially appear life threatening can become so in minutes. The flowchart in Figure 15-1 shows key considerations and decisions the EMT faces with the trauma patient.

WHY DO TRAUMA PATIENTS DIE?

In the immediate posttrauma period, trauma patients may die from hypoxia as a result of airway/ventilatory failure, circulatory failure (cardiac or fluid), or brain injury. Subsequently, they may die from the biochemical and pathophysiologic effects of prolonged hypoxia and hypoperfusion, leading to immune system failure (sepsis) or other organ failure (renal, hepatic, pulmonary). If such prolonged anaerobic metabolism can be prevented or reduced, the likelihood of the progression of multiple organ failure (MOF) will be diminished.

Of course, even with the best planned and executed resuscitation, not all trauma victims can be saved. However, with the EMT's attention focused on the reasons for early traumatic death, a much larger percentage of patients may survive, and there may be less residual morbidity than without the benefit of the EMT's correct and expedient field management.

The EMT's efforts at resuscitation must be focused on rapid treatment for hypoxia, hypoperfusion, and hemorrhage control. The initial interventions are aimed only at the restoration of these vital functions and rapid transportation to the appropriate facility to continue the care started in the field and to provide homeostasis. All other treatment of the critical trauma patient is fruitless unless these primary goals are met. Sometimes the most dramatic visible injuries are ignored, and attention is paid only to those conditions that will result in death if they are not managed appropriately and immediately. The management of multisystem trauma patients must be based on resolving the problems that cause death.

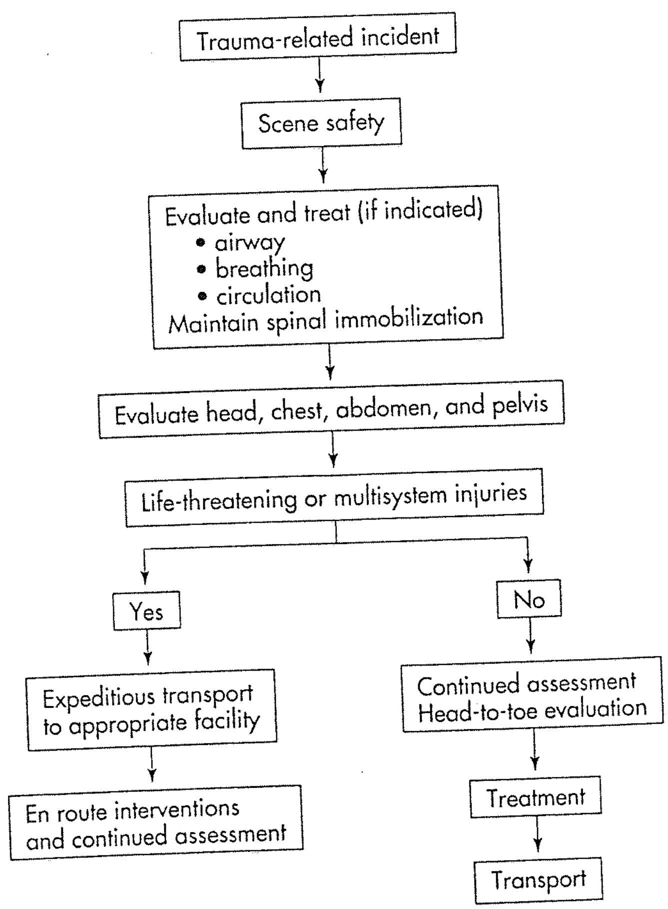


Figure 15-1 Key considerations in treating the trauma patient.

STRATEGIES FOR MULTISYSTEM TRAUMA PATIENT CARE

The key to the prehospital management of the multisystem trauma patient is organizing the knowledge and skills involved into a sound strategy—a plan of attack that can serve as a framework and method for dealing with such patients in a systematic, rapid, and thorough way. Throughout this text such a plan has been presented. It can be outlined briefly as follows:

1. Evaluate the three **S**'s: safety, scene, and situation.
2. Rapidly assess the patient's systemic condition. Evaluate the airway, breathing, and circulation with attention for possible spine injury. Focus on ventilation, shock, hemorrhage control, and spinal immobilization.
3. Provide intervention for these problems as they are found.
4. Reassess vital functions to evaluate the effectiveness of the interventions.
5. Reassess the head, chest, and abdomen to locate potentially life-threatening conditions. Rapidly provide needed interventions for any conditions that are found.
6. Immobilize the patient and expedite transport to the closest appropriate facility.
7. Perform a rapid secondary survey, and provide additional management while en route to the hospital.

Essential to this strategy is the rapid differentiation between patients with multisystem trauma and those with only simple trauma. The needs and priorities of each category are significantly different. All multisystem trauma patients, regardless of apparent stability, must be considered unstable until definitive diagnosis and treatment can be provided in the hospital.

A variety of independent studies published in the past few years have shown that the initiation of transport to a proper facility without delay is one of the key EMT treatments affecting survival in patients with multisystem trauma. The concepts of the golden hour from injury to management and the golden 10 minutes in the field, along with the increasing potential for permanent injury and mortality that results from each delay in definitive care, are universally accepted axioms of current trauma care.

Multisystem trauma patients should be held in the field only for urgent actions that will alter the outcome positively or that are mandated by the scene/situation (e.g., time needed for extrication).

Those steps that the EMT might legitimately take the time to do before beginning transport with such patients include the following:

- establishing scene safety
- rapid assessment (limited to life-threatening conditions)
- emergent interventions
- extrication of the patient
- proper packaging of the patient (longboard, hemorrhage control, spinal immobilization, etc.)

Any trauma patient who currently has, or has demonstrated by history or physical examination, any of the following should be classified and managed as a critical trauma patient:

- lowered level of consciousness
- any period of unconsciousness
- dyspnea
- significant bleeding
- shock (compensated or decompensated)
- incontinence
- significant injury of the head, face, neck, thorax, abdomen, or pelvis
- history of major medical problem
- mechanism of injury that commonly produces significant internal injuries

Only patients without injuries or conditions that are commonly associated with any potential life-threatening impact should be considered and managed as simple trauma patients (i.e., those with an isolated extremity fracture). If any doubt exists, the patient must be considered to have multisystem trauma.

Patients meeting the American College of Surgeons' criteria for physiologic, anatomic, or mechanism of injury for potentially severe injuries should be transported to a trauma center if one is present in the community. It is appropriate to bypass nontrauma centers to reach a trauma center.

In children, pregnant patients, and the elderly, injuries must be considered to be more serious than their outward appearance, have a more profound systemic impact, and have a greater potential for producing rapid devastating decompensation. In pregnant patients, there are at least two patients to care for, the mother and one or more fetus(es), both (or all) of whom may have sustained injury. In children and the elderly, the physical examination can be falsely reassuring and vital signs misleading. Compensatory mechanisms differ from younger adults and may not reveal abnormalities until the patient is profoundly compromised.

Occasionally, problems at the scene can impede the EMT's ability to rapidly initiate transport of the trauma patient. Multiple patient situations, the inability to reach the patient because of external hazards, or the inability of the EMT to extricate the patient from the situation clearly call for alterations in the standard approach to resuscitation. Many lifesaving procedures must be accomplished prior to patient removal. However, once the scene has become safe for the patient and rescuer, or the patient has become available for extrication, all efforts should again be directed at the rapid provision of care for life-threatening injuries and expedient transportation of the patient to a definitive care facility. The needs of the patient must always be the major determinant of the care provided.

Managing multisystem trauma patients is based on ensuring scene safety, rapidly identifying and treating life-threatening problems, and providing expeditious transport to an appropriate receiving hospital (trauma center).

STRATEGY FOR SIMPLE TRAUMA PATIENT CARE

When the EMT's initial assessment indicates that a patient has solely simple trauma, the continued care focuses on a more detailed head-to-toe examination and on the management and stabilization of each injury. Since urgency has been ruled out in such cases, it is prudent to ensure that the injuries have been identified and properly managed prior to moving and transporting the patient to protect the patient from any additional harm. Rapid transportation (lights and siren) should not be used with patients who only have simple trauma since it presents an unwarranted risk of traffic collision and an unnecessary danger of further injury. Although the EMT should take the time needed to identify and treat each injury, this should not be misinterpreted as an invitation to linger. Although no extreme urgency exists, the patient may suffer from undue delay and may, as a result of the simple trauma or of unrecognized injuries, deteriorate if field time is needlessly extended. (See Box 15-1 for key factors in the management of prehospital trauma.)

Box 15-1 The Key Dozen Factors in the Management of Prehospital Trauma

1. Ensure safety of responders and patient.
2. Use rapid assessment to identify systemic deficits and patients with multisystem trauma.
3. Provide high FiO_2 to maintain $SpO_2 > 90\%$ to 92%
4. Provide airway management and ventilation as indicated.
5. Protect normal body temperature (use warm intravenous fluids and inhaled gases, monitor patient compartment temperature, keep patient covered).
6. Stop any significant external bleeding.
7. Use the pneumatic antishock garment to control pelvic bleeding or use as indicated in individual EMS system.
8. Provide basic shock treatment.
9. Provide rapid warm fluid replacement en route to the hospital.
10. Protect spine and other musculoskeletal injuries by immobilization to a longboard.
11. Include significant medical problems or history in assessment and care.
12. Rapidly initiate transport to an appropriate facility.

• Summary •

1. Maintaining a focus on priorities is the key to successful management of the multisystem trauma patient, whether there is one patient or many. The information contained in this text serves as a framework for a general approach to the patient.
2. Generally, no more than 10 minutes is necessary or recommended in the field when dealing with the critical trauma patient. If a review of your own recent run reports shows that you are taking more time, try to determine the reason. Can such patients really afford the wasted time at the scene?
3. Providing for the basics of airway, ventilation, and circulation management and adequate immobilization can be done in various ways. The EMT must select the methods that best meet the patient's needs in each circumstance. Is the time in the field being spent efficiently—to rapidly provide needed intervention—or is it wasted through inefficient techniques or by caring for low-priority injuries? We all may occasionally find ourselves being meticulous and tidy when the patient's needs dictate a broader and more sweeping approach.
4. Trauma care:
   - is dependent on the ability to perform a meaningful rapid assessment
   - is dependent on the ability to quickly locate and recognize life-threatening and potentially life-threatening conditions
   - must follow a given set of priorities that establish an efficient and effective plan of action, based on available time frames and any dangers present at the scene, if the patient is to survive
   - must provide appropriate intervention and stabilization
   - must be integrated and coordinated between the field, the emergency department, and the operating room. Each and every provider, at every level of care and at every stage of treatment, must be in harmony with the rest of the team.
   - must have as its goal the provision of definitive surgical care of the critical trauma patient within the golden hour

These basic concepts, rather than new specific skills, are the cornerstone to reducing morbidity and mortality in the critical trauma patient.

above 75%, evaporation decreases; at levels approaching 90%, evaporation essentially ceases.

Maintenance of Thermoregulation

The balance between heat loss and heat production is constantly changing because of changes in metabolic rate or changes in the external environment. The extent to which the hypothalamus initiates and integrates physiological activity to maintain body temperature is not fully understood. However, several hyperthermic and hypothermic compensatory responses are thought to be the basis for maintaining thermoregulation (Box 25-1).

HYPERTHERMIA

Heat illness results from one of two basic causes: (1) The normal thermoregulatory mechanisms are overwhelmed by environmental conditions such as heat stress (exogenous heat load) or, more commonly, by excessive exercise in moderate-to-extreme environmental conditions (endogenous heat load) and (2) failure of the thermoregulatory mechanisms, as may be encountered in older adults or ill or debilitated patients. Either cause may result in heat illness such as heat cramps, heat exhaustion, or heat stroke.

Heat Cramps

Heat cramps are brief, intermittent, and often severe muscular cramps that frequently occur in muscles fatigued by heavy work or exercise. They are believed to be caused primarily by a rapid change in extracellular fluid osmolarity resulting from sodium and water loss.

Heat cramps are suffered by persons who sweat profusely and subsequently drink water without adequate salt. During environmental heat stress, 1 to 3 L of water per hour may be lost through sweating. Each liter contains between 30 and 50 mEq of sodium chloride. The water and sodium deficiency combine to cause muscle cramping, which normally occurs in the most heavily exercised muscles, including the calves and arms (although any muscle may be involved). These patients are usually alert with hot, sweaty skin; tachycardia; and normotension; they have a normal core temperature. Heat cramps are easily treated by removing the patient from the hot environment and replacing sodium and water. In severe cases, medical control may recommend intravenous infusion of a saline solution.

Heat Exhaustion

Heat exhaustion is a more severe form of heat illness characterized by minor aberrations in mental status (irritability, poor judgment), dizziness, nausea, headache, and mild-to-moderate core temperature elevation (less than 103° F [39° C]). In severe cases, orthostatic dizziness and syncope may occur.

Like heat cramps, heat exhaustion is more commonly associated with hot ambient temperature; it results in profuse sweating. With water and salt deficiency, electrolyte imbalance and vasomotor regulatory disturbances contribute to inadequate peripheral and cerebral perfusion, the signs of which are characteristic of this illness. Rapid recovery generally follows fluid administration. Patients with significant fluid abnormalities or orthostatic hypotension may require intravenous administration of a saline solution. Left untreated, heat exhaustion may progress to heat stroke.

Heat Stroke

Heat stroke is a syndrome that occurs when the thermoregulatory mechanisms normally in place to meet the demands of heat stress break down entirely. This failure results in body temperature elevated to extreme levels (usually greater than 105.8° F [41° C]), producing multisystem tissue damage and physiological collapse. Heat stroke is a true medical emergency. The syndrome may be classified into two types: classic heat stroke and exertional heat stroke.

Classic heat stroke occurs during periods of sustained high ambient temperatures and humidity. The illness commonly affects the young, older adults, and those who live in poorly ventilated homes without air conditioning. Examples include young children left in an enclosed automobile on a hot afternoon and older persons confined to a hot room during a heat wave. Classic heat stroke victims also frequently suffer from chronic diseases such as diabetes, heart disease, alcoholism, or schizophrenia, which predispose them to the syndrome. Many of these patients take prescribed medications such as diuretics, antihypertensives, tranquilizers, and anticholinergics, which further impair their ability to tolerate heat stress. In these patients, the illness develops from poor dissipation of environmental heat.

In contrast to patients with classic heat stroke, patients with **exertional heat stroke** are usually young and healthy. Commonly afflicted groups include athletes and military recruits who exercise in hot and humid conditions. In these situations, heat accumulates more rapidly in the body than it can be dissipated into the environment.

Clinical Manifestations

Body temperature is controlled in the hypothalamic thermoregulatory centers. These centers receive their information largely from the temperature of circulating blood and from peripheral thermoreceptors in the skin. In response to hypothalamic stimulation, the respiratory rate quickens to increase heat loss via exhaled air, cardiac output expands to provide increased blood flow through skin and muscle to enhance heat radiation, and sweat gland activity increases to enhance evaporative heat loss. These compensatory mechanisms require a normally functioning central nervous system to integrate thermal inputs and initiate appropriate thermoregulatory responses and an intact cardiovascular system to transport excess heat from the core to the periphery. Dysfunction in either or both of these systems leads to rapidly increasing core temperatures.

Central Nervous System Manifestations

The central nervous system manifestations of heat stroke vary. Some patients may be in frank coma; others exhibit confusion and irrational behavior before collapse. Convulsions are common and can occur early or late in the course of the illness. Since the brain stores little energy, it depends on a constant supply of oxygen and glucose. Decreased cerebral perfusion pressure results in cerebral ischemia and acidosis, and increased temperatures markedly increase the metabolic demands of the brain. The extent of cerebral damage depends on the severity and duration of the hyperthermic episode.

Cardiovascular Manifestations

A rise in skin temperature reduces the thermal gradient between the core and the skin and evokes an increase in skin blood flow (peripheral vasodilation) that results in cutaneous flushing. Although in the classic form of heat stroke sweating is usually absent (because of dehydration, drug use that impairs sweating, direct thermal injury to sweat glands, or sweat gland fatigue), 50% of exertional heat stroke cases have persistent sweating that results from increased catecholamine release. Therefore the presence of sweating does not preclude the diagnosis, and cessation of sweating is not the cause of heat stroke.

As the illness progresses, peripheral vasodilation results in decreased vascular resistance and shunting. High-output cardiac failure is common, manifested by extreme tachycardia and hypotension. Cardiac output may initially be 4 to 5 times that of normal, although as temperatures continue to rise, myocardial contractility begins to decrease and patients can demonstrate an elevated central venous pressure. In any age group, the presence of hypotension and decreased cardiac output indicates a poor prognosis.

Other Systemic Manifestations

Other systemic manifestations that may be associated with heat stroke include pulmonary edema (plus concomitant systemic acidosis, tachypnea, hypoxemia, and hypercapnia), myocardial dysfunction, gastrointestinal bleeding, aberrations in renal function (secondary to hypovolemia and hypoperfusion), hepatic injury, clotting disorders, and electrolyte abnormalities.

Management

If untreated, heat stroke almost invariably culminates in death. The factors most important to a successful outcome are initiation of basic and advanced life-support measures, rapid recognition of the heat illness, and rapid cooling of the patient. After ensuring adequate airway, ventilatory, and circulatory support, the patient with heat stroke should be managed as follows:

1. Move the patient to a cool environment and remove all clothing. If available, use hyperthermic thermometers such as rectal probes or tympanic membrane devices to monitor core temperature. Take and record the temperature at least every 5 minutes during the cooling process to ensure adequate rates of cooling and to avoid rebound hypothermia. Rebound hypothermia can best be avoided by stopping the cooling measures when the patient's core temperature reaches approximately 102° F (39° C).
2. Begin cooling by fanning the patient while keeping the skin wet and massaging the patient with bags of ice or commercial cold packs. Lowering the body temperature by this method should be continued en route to the receiving hospital. If transport is to be delayed, medical control may recommend complete immersion or spraying of tepid water (60° F [16° C]) over the body surface. Ice-water submersion or cold-water cooling should be avoided because these methods may precipitate shivering, frank shaking, peripheral vasoconstriction, and convulsions as the body temperature is being lowered.
3. If hypovolemia is present, give the patient an initial fluid challenge of 500 ml over 15 minutes. In the majority of patients, the blood pressure rises to a normal range during the cooling process as large volumes of blood in the cutaneous vessels shift back to the central circulation (rapid cooling improves cardiac output directly). Therefore be extremely cautious with fluid replacement and closely monitor the patient for signs of fluid overload. Vigorous fluid administration may precipitate pulmonary edema, especially in the older adult.
4. Administer pharmacological agents as prescribed by medical control. Depending on the patient's status and response to cooling methods, these drugs may include ***diazepam*** for sedation and seizure control, ***mannitol*** to promote renal blood flow and diuresis, and glucose to treat hypoglycemia.

ACCIDENTAL HYPOTHERMIA

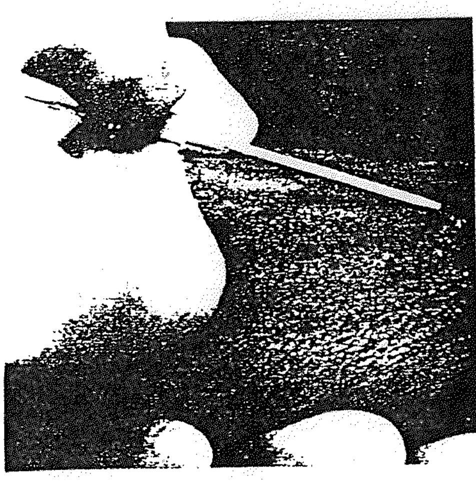
Hypothermia (core temperature less than 95° F [35° C]) may result from a decrease in heat production, an increase in heat loss, or a combination of the two factors. Although hypothermia may result from metabolic, neurological, traumatic, toxic, and infectious causes, it is most commonly seen in cold climates and during periods of exposure to extreme environmental conditions. Failure to recognize and properly treat hypothermia can lead to significant morbidity and mortality.

Pathophysiology

Cold exposure produces a cascade of physiological events to conserve core heat. Initially there is immediate vasoconstriction in the peripheral vessels and a simultaneous increase in sympathetic nervous discharge, catecholamine release, and basal metabolism. In addition, the heart and respiratory rates increase dramatically, as does the blood pressure. As cold exposure continues, preshivering muscle tone increases, and the body generates heat in the form of shivering. Shivering continues until the core temperature reaches approximately 86° F (30° C), glucose or glycogen is depleted, or insulin is no longer available for glucose transfer. When shivering stops, cooling is rapid, and there is a general decline in all physiological responses.

With continued cooling, respirations decline slowly, pulse and blood pressure decrease, and there are significant decreases in blood pH and commonly electrolyte imbalances. Hypovolemia can develop from a shift of fluid out of the vascular space, with increased loss of fluid through urination (cold diuresis). After early tachycardia, progressive bradycardia develops that is often refractory to ***atropine***. Significant electrocardiographic (ECG) changes occur, including prolonged PR, QRS, and QT intervals; obscure or absent P waves; and ST-segment and T-wave abnormalities. In addition, the J wave (Osborn wave) may be present

appendix 2



Intravenous Fluid Therapy

* OBJECTIVES

- Explain what IV therapy is and why it is used in prehospital medicine.
- Describe the equipment and supplies needed to provide IV therapy.
- Learn the steps of and demonstrate proficiency in starting an IV infusion.
- Discuss the importance of proper maintenance and monitoring of the IV patient.
- Demonstrate how to transport the IV patient properly.
- Describe the possible complications of IV therapy.

It is recommended that EMTs wear protective gloves whenever there is a possibility of coming in contact with a patient's blood, body fluids, mucous membranes, traumatic wounds, or sores. See Chapter 31.

In many states, EMTs are being taught the basics of **venipuncture** and **intravenous (IV) therapy** for use in the field to expand the level of life support care given. IV therapy should only be administered by IV-certified EMTs; follow local protocol.

☐ WHAT IS INTRAVENOUS INFUSION?

Intravenous therapy, commonly called IV, refers to the administration of fluids, drugs, or blood directly into the circulatory system by way of a vein. When blood is administered, the technique is called **transfusion.** When sterile fluids other than blood or blood products are administered through a line injected into the venous system, the technique is called **infusion.**

An IV is a lifeline through which fluids and medications are administered to a patient. The fluid container can empty its reserve in minutes. A dropcock, or drip chamber, placed below the container, regulates the flow of the fluid.

☐ BODY FLUID COMPOSITION

Body fluids bathe each cell and are involved in all bodily chemical reactions. Without the proper amounts of body fluids, cells dehydrate and die. Body fluid consists of water (60 percent of the volume of adult bodies and 75 percent of infant bodies) and electrolytes (sodium and potassium). These fluids are found both inside and outside the cell. Extracellular fluid includes the interstitial fluid between the cells and the capillary walls, and blood plasma within the vascular system.

☐ WHY INFUSION?

IVs are started in the field for four major reasons:

- To add fluid volume to the circulatory system when there is an imbalance or depletion of normal body fluids, as in hemorrhage, burns, and dehydration.
- To establish and maintain a life support or access line for fluid or medication in a patient whose condition is questionable. It is difficult to get into a vein and start an IV after hypovolemia or circulatory collapse.
- To provide access for the administration of medications in a myocardial infarction or cardiac arrest, diabetic emergencies, drug overdose, etc.
- To maintain electrolyte, fluid, and nutrient balances for those unable to eat or with problems of severe nausea, vomiting, and/or diarrhea.

Be sure to get specific instructions from the physician and document the orders.

☐ ADDING FLUID VOLUME

A significant decrease in fluid volume must be countered rapidly, or shock may result. The body may also go into chemical imbalance and negatively affect the functioning of vital organs. The types of solutions used for field IVs include **crystalloids** and **colloids.**

Colloids and crystalloids are volume expanders given to patients whose condition results in compromised circulation of blood to body tissues. They do not carry oxygen or replace blood but can provide electrolytes, protein, and volume expansion to help maintain blood pressure.

Crystalloid solutions quickly expand plasma, are rich in electrolytes, and take effect more quickly than colloids. However, they last only a short time. Colloids take effect more slowly than crystalloids but last longer in the plasma. They are particularly helpful for patients with hypovolemic or cardiogenic shock. Examples of colloids are **dextran** and **hetastarch** (**serum albumin** is a natural colloid). Examples of crystalloids are: (Table A2-1)

- **N.S.,** or **normal saline,** which is 0.9 percent sodium chloride in sterile water.
- **Lactated Ringer's,** an isotonic, buffered solution of electrolytes (sodium, chloride, potassium, calcium, and lactate) that closely approximates normal blood electrolyte contents (Figure A2-1).
- **D_5W,** which is 5 percent dextrose and sterile water. It is used in cases where an IV is established as a lifeline or a medication route.

☐ SETTING UP AN IV

The equipment used by EMTs is usually disposable. Some medical facilities provide reusable, sterile infusion sets. In any case, the equipment will basically be the same, consisting of:

- The fluid to be infused.
- The IV set (Figure A2-2 shows micro drip and macro drip sets), consisting of the connector (to the fluid bottle or bag), drip chamber, screw clamp or flow adjustment valve, Y injection site (for medications), needle adapter, and needle and catheter.

TABLE A2-1
Common Intravenous Fluids

SOLUTION	ABBREVIATION	COMPONENT ELECTROLYTES
Lactated Ringer's	LR	NaCl, potassium chloride (KCL), calcium chloride (CaCl), sodium lactate
Quarter-normal saline	1/4 NS	0.2 NaCl
Half-normal saline	1/2 NS	0.45% NaCl
Normal saline	NS	0.9% sodium chloride (NaCl)
5% dextrose	D5W	5% dextrose
10% dextrose	D10W	10% dextrose

Note: D5 or D10 as a prefix indicates the solution is made containing dextrose. For example, Lactated Ringer's in 5% dextrose would be abbreviated D5LR.

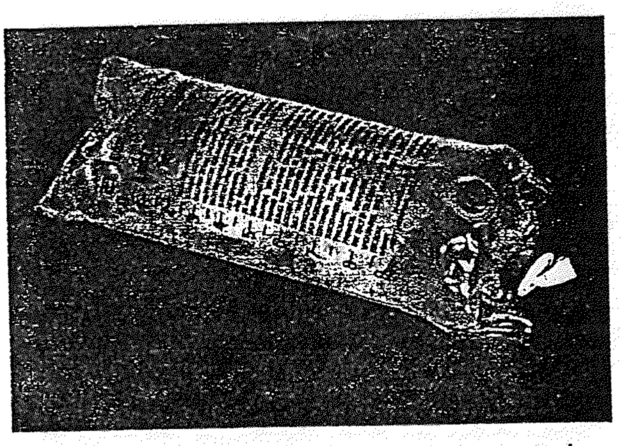


FIGURE A2-1 Lactated Ringer's, a solution commonly used in IV administration.

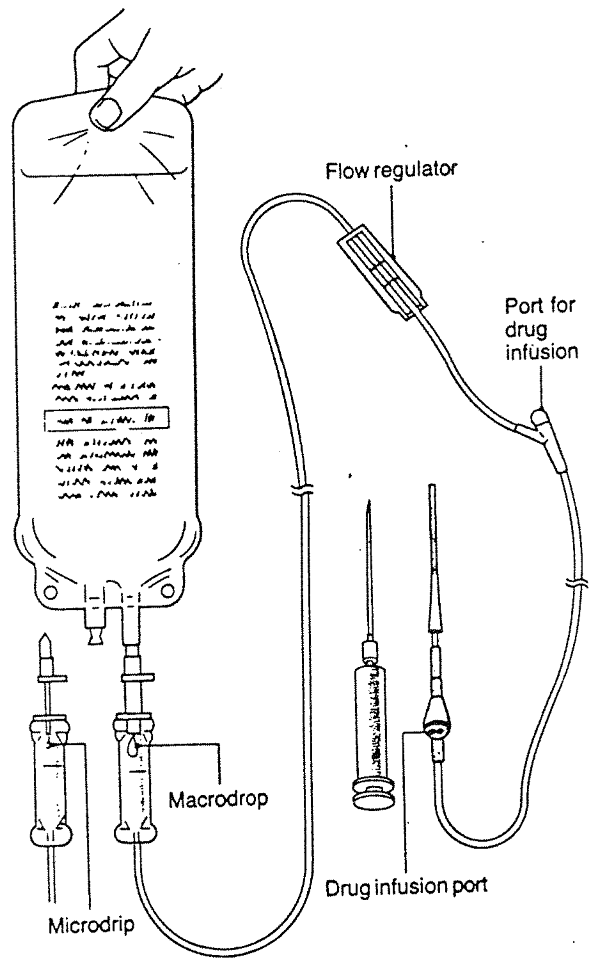


FIGURE A2-2 Comparison of macrodrip and microdrip IV administration sets.

- Auxiliary equipment (Figure A2-3), such as an arm board, antiseptic solution, tape to secure the IV tubing to the patient's arm and the arm to the board, a tourniquet to aid in selection of the insertion site, gauze pads or a sterile dressing such as Opsite to cover the insertion site, materials to log or write down any necessary records concerning the procedure, and IV extension tubing to give added length to the IV while transporting.
- Several gloves and possibly a face mask and eye protector to be worn by the EMT.

It is important that all equipment be sterile. If the equipment is contaminated, germs may be introduced into the body and cause infection. If you do not *know* that the equipment is sterile, consider it *contaminated.* A sterile object remains sterile only if touched by another sterile object. It is very important that you be honest and make it known if a piece of equipment becomes contaminated — it needs to be replaced.

Choosing the IV Set, Needles, and Catheters

Two types of IV sets are commonly used — **macro drip** and **micro drip.** The macro drip sets are used for rapid fluid replacement by large drops of fluid through a large-bore tube. This macro drip, or standard, infusion set is typically used for adults to give large amounts of

FIGURE A2-3 Auxiliary equipment.

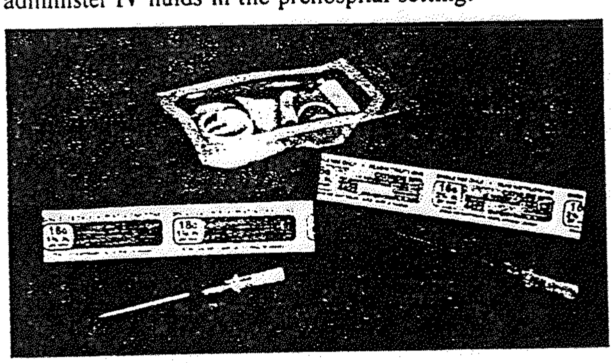
fluid. The micro drip set has a small-bore tube, allowing a smaller drop, and is used for children, for maintaining a lifeline, or for other situations where control of the IV rate is critical.

The primary type of needle used to enter the vein in the field is an over-the-needle catheter (a plastic catheter inserted over a hollow needle). Other types of needles are a butterfly or winged hollow needle, or a plastic catheter inserted through a hollow needle. (Figure A2-4). In general, a short, large-bore needle is best for IV therapy. One- or-two-inch-length catheters are the most commonly used in the field, with needle sizes of 14, 16, and 18 gauge (the lower the gauge, the larger the bore of the needle) for fluid replacement. An 18 gauge is generally the smallest used in any adult and most children, but a 20 gauge may be used for small children or older adults with fragile veins that will not accommodate a lot of fluid.

The other variable that should be considered when selecting an intravenous cannula is its length. The longer the cannula, the less the flow rate will be. The flow rate through a 14 gauge, 5 cm catheter (approximately 125 mL/minute), is twice the flow rate through a longer, 16 gauge, 20 cm catheter. For cannulation of a peripheral vein, a needle and catheter length of 5 cm is adequate while the cannulation of a central line requires a needle length of 6–7 cm and catheter length of at least 15–20 cm.

Other needed equipment includes alcohol swabs, povidone-iodine solution, tape, and sterile dressings.

FIGURE A2-4 The IV needles most commonly used to administer IV fluids in the prehospital setting.



Assembling the Equipment

Following these procedures, and using only the type of fluid ordered by the physician, perform the following steps.

1. Check the container to make sure that the expiration date has not passed.
2. Plastic bag infusion sets are preferable to glass in the field. If a glass bottle is used, inspect it for cracks.
3. Remove the sterile seal from the end of the tubing closest to the drip chamber and insert the tubing into the container. The tubing also has a sterile seal on it. You may have to loosen this seal to allow the liquid to flow, but you should not remove it.
4. With either container, check for seal leakage, cloudiness, discoloration, or contamination. Do not use any fluid that is colored or cloudy or that contains floating particles. Save the bag and report the problem to your equipment manager so that he or she can inspect other supplies in the same lot.
5. As you open the packages to assemble the infusion set, keep all necessary items sterile by *not* touching areas that will come in contact with the fluid. Do *not* use your teeth to rip open the coverings on the bags and tubes. It is a good idea to have extra alcohol wipes and a spare catheter near. Tear the tape to the right size for securing the catheter and tubing.
6. Connect the infusion set to the fluid container by holding the drip chamber, removing any protective coverings (do not touch the spike tip), then inserting the piercing pin into the fluid container with a twisting motion (Figure A2-5).
7. Attach the extension tubing, then squeeze and release the drip chamber or reservoir on the infusion set until it is about half full.
8. Remove the protective cover from the needle adapter. Inspect the needle and cannula for irregularities. If the needle is not sharp and without burrs and if the cannula is not smooth, discard them.
9. Open wide the flow adjustment valve, and flush any air from the tubing. No air should be left in the line, or it may enter the patient's vein, causing an air embolus or blockage. Some EMTs save time and eliminate this step by prehanging IV fluids. If you use this procedure, label the bag with the time, date, and your initials. Fluids and tubing should be discarded after a maximum of twelve hours.

FIGURE A2-5 Hold the drip chamber and insert the piercing pin into the fluid container with a twisting motion. (Apply protective gloves before initiation of an intravenous line.)

10. Adjust the flow valve until the flow stops, then replace the protective cover over the needle adapter and protect it from contamination.
11. Select the needle or IV catheter best suited to the patient (18 gauge is normally used). The needle should be large enough that it will enter the vein easily but without tearing it.
12. Select an appropriate infusion set. Is it for fluid replacement? Micro or macro?
    - Be familiar with the type of IV fluid — always use the same type of fluid if hanging a new container.
    - Be aware of any additives in hanging a new bottle or in the original container of the IV field.
    - Keep the container three feet above the insertion site at all times.
    - Time-label the IV solution container. Tape the side of the container with date, time hung, and rate of solution per hour.

These points are discussed with ideal conditions in mind, but often in the field, they are not, and time is at a premium. Documentation is often left until you reach the emergency department, where the flow rate and possibly the fluid may be changed. It is very helpful to hospital personnel if, on a piece of tape over the insertion site, you write the gauge of the needle, the date, the time the IV was started, and the initials of the EMT who started the IV.

☐ THE IV PROCEDURE

Follow these steps in administering an IV:

1. Explain the procedure to your patient and why you are doing it. Be professional and calm, allowing the patient to have confidence in you. Ask the patient about any possible allergies to tape, fluids, iodine, etc.
2. Prepare yourself properly to prevent any possibility of the patient's blood coming into contact with you. Wear surgical gloves and possibly a face mask and eye protection (see Chapter 31).
3. Select a proper site.
    - Unless the patient's arms have been severely traumatized, use arms rather than legs for placing IVs. The arms have a lower risk of phlebitis than legs.
    - Have the patient hang his or her arm for a couple of minutes. Apply the tourniquet three or four inches (adult) above the antecubital fossa (Figure A2-6). The tourniquet should occlude the venous pressure but not the arterial. If a blood pressure cuff is used as a tourniquet (sometimes good for better control), inflate it to 15 to 20 mmHg below the systolic blood pressure. The distal pulse should still be present.

FIGURE A2-6 Place a constricting band above the site for the venipuncture.

- If it is not, loosen the tourniquet until the arterial pulse returns.
- Look on the forearm or back of the hand for a fairly straight vein that lies on a flat surface. The vein should feel springy when you palpate it. Usually the forearm is the first choice. Creation of a **pulse wave** helps in locating a good vein (Figure A2-7). (The American Heart Association's Advanced Cardiac Life Support Text recommends using the antecubital vein in cases of cardiac arrest. This vein can also be used in cases of severe circulatory collapse.)
- Choose the top side of the arm above the wrist or the back of the hand.
- It is a good idea to start the IV as low as possible on the limb. If a problem arises, the next IV will need to be inserted above the heart in relation to the first site. The **basilic, cephalic,** or **median veins** are common sites for IVs.
- Avoid sites where veins are near injured areas, or where arterial pulsations are found close to the vein being considered.
- Stay away from joints.
- Because the needle must enter the vein lengthwise, know the direction of the vein. Track the direction for one to one and one-half inches (or at least the length of the catheter used).

4. Prepare the IV site. You should scrub and disinfect the site in two separate steps. Use an alcohol scrub to remove dirt, dead skin, blood, mucous, and other contaminants from the surface.
   - Cleanse the selected site with an iodine or alcohol swab. Sponge the antiseptic directly over the selected vein, then rub in a circle until an area one to three inches is covered. Rub in a circular motion, starting at the puncture site and going out. Never go back over the area just cleaned with the same wipe.
   - If a povidone-iodine solution is used, follow with an alcohol wipe in a circular motion, starting at the venipuncture site. This reduces the risk of a reaction. If you have scrubbed and disinfected with alcohol in both steps, be sure to prep for at least sixty seconds using at least two or three wipes. Do not rush this step. It takes time for alcohol to act on the skin microorganisms.
5. If the patient is responsive, briefly explain the purpose of the IV and the procedure for initiating it.
6. Have the patient clench and unclench his or her fist several times. This will improve venous distention. Now select a distended vein that appears straight and that lies on a flat surface. Do not palpate the vein with your bare fingers or soiled

FIGURE A2-7 Hold the patient's hand and press downward with your thumb. This will create a pulse wave that will help your select a good vein.

gloves after disinfection. Put on a pair of fresh, clean gloves immediately before starting the IV.

7. Stabilize the vein by gently applying pressure on it an inch below the point where the needle will enter. (If you feel a pulse, *do not* use this site. It is an artery! Select another site.)
8. Press the vein downward, toward the wrist, so that the vein does not roll.
9. With the bevel (the slanted end of the needle) up, align the needle so that it will enter the skin at a twenty- to forty-degree angle and in the direction of the venous flow. Remember — the needle must enter the vein lengthwise (Figure A2-8). Some services use a bevel-down technique in cases of difficult or rolling veins.
10. Pierce the skin and insert the needle into the vein (Figure A2-9). Smooth movement of one-fourth to one-half inch hurts the patient much less than small, apparently insignificant movement as the IV is started. You should feel some resistance, then a "pop" when the vein is punctured. A confirmation that the needle is in the vein is when the blood appears in the **flash chamber** at the end of the needle (Figure A2-10).
11. A difficult IV start may be enhanced by using a syringe. A syringe may mean the difference between success or failure. To perform this procedure:
    - Insert the needle about 5 mm, but no more.
    - Slide the catheter into the vein by pushing the hub until the catheter is fully in the vein (Figure A2-11). *Do not* advance the needle and catheter

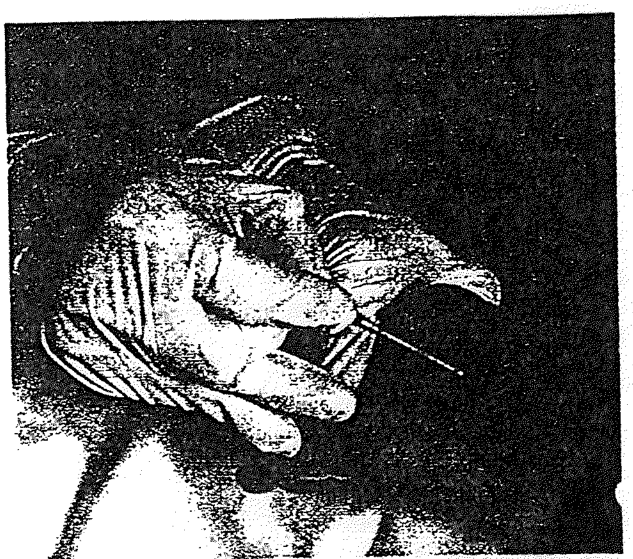


FIGURE A2-8 Hold the needle at a 20- to 40-degree angle in the direction of the venous flow, bevel up!

FIGURE A2-9 Pierce the skin and insert the needle into the vein.

FIGURE A2-10 Blood appearing in the flash chamber is confirmation that the needle is in the vein.

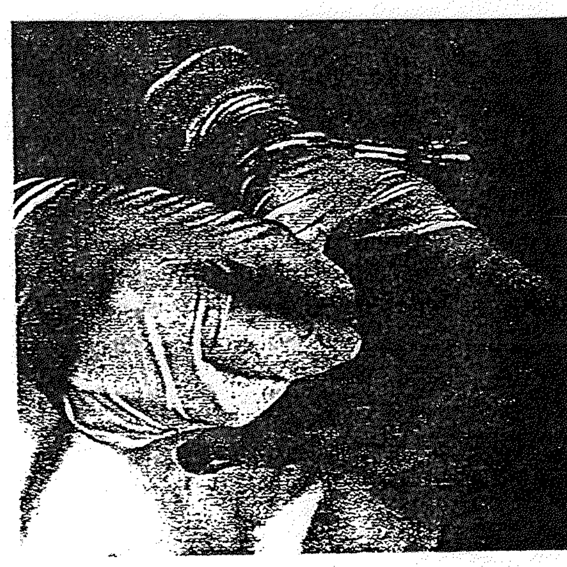


FIGURE A2-11 Now slide the catheter (and IV tubing) into the vein.

together, and *do not push the catheter back over the needle or push the needle back into the catheter*. This action may cause the catheter to be sheared off by the needle.

- While holding the catheter hub in place, carefully withdraw the needle.

12. Maintain firm pressure on the vein above the catheter and make a quick, visual check to see that all is ready.
13. Remove the protective cap from the end of the infusion set, then attach the needle adapter by twisting it securely into the hub. The area around the infusion site should be clean and dry.
    - Remove the tourniquet (Figure A2-12).
    - Blood loss through the catheter can be stopped by compressing the vein near the tip of the catheter with a finger or thumb.
14. Open the flow adjustment valve.
15. The fluid should drip steadily into the drip chamber. If it does not, gently pull the catheter out 2 to 3 mm only. The drip should now flow steadily.
16. Apply povidone-iodine solution and cover the infusion site with a small gauze pad (follow local protocol; some use a clear cover for the IV site, such as **opsite**). Be aware of **radine** allergy (swelling and redness).

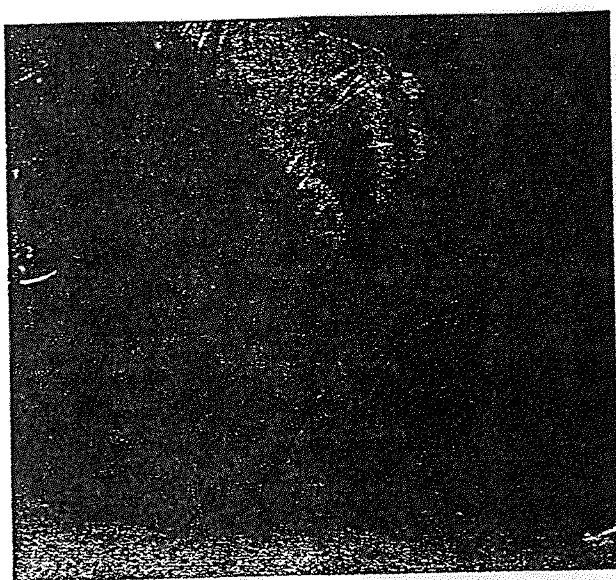


FIGURE A2-12 After holding the catheter hub in place and withdrawing the needle, remove the tourniquet.

FIGURE A2-13 Tape the catheter securely in place and tape the looped IV tubing to the arm.

17. Tape the catheter securely in place (Figure A2-13). Taping is critical in maintaining the IV. However, do not apply tape completely around the extremity. This could cause a tourniquet effect, decreasing circulation to the distal portion of the extremity.
18. Loop the IV tubing and tape it to the arm with generous, secure taping (Figure A2-14). Attachments such as a T-tube and IV loop can reduce the problem of pinching off a large loop of IV. *Do not* tape the point of connection between the catheter and the infusion set, however. Apply an arm board if it is necessary to minimize arm motion. (See also Figure A2-15).
19. Write with ink on the tape the type of cannula used, the needle gauge, the catheter length, the time and date, and the initials or signature of the EMT who performed the procedure (Figure A2-16).

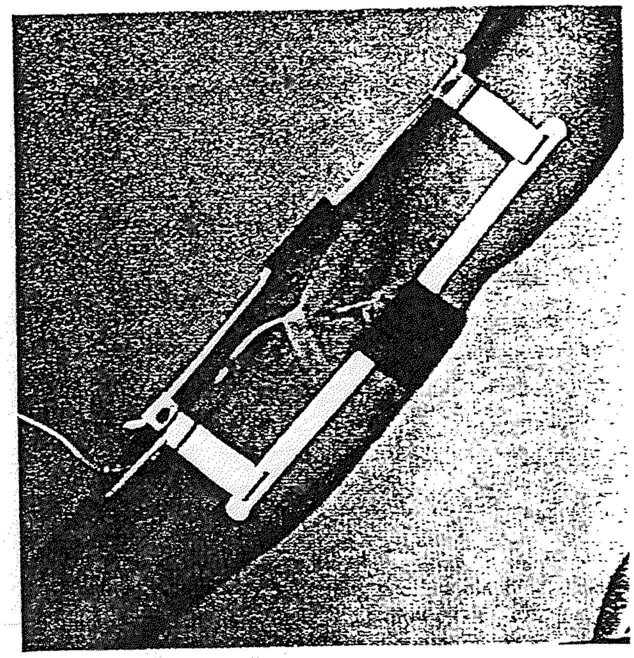


FIGURE A2-14 Securing an IV.

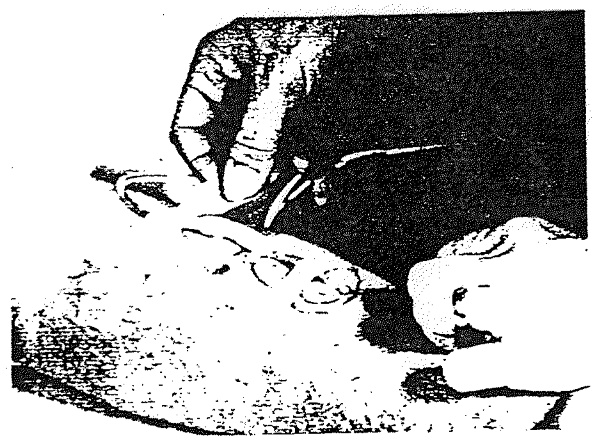


FIGURE A2-15 A butterfly catheter may also be used in the field. Loop the IV tubing and securely tape it to the arm.

20. Adjust the infusion to the flow rate (ml/hour) ordered by the physician. It is essential that the proper flow rate be monitored and maintained (Figure A2-17). Too much IV fluid can be dangerous to the patient, especially to children. To adjust the infusion to the ordered flow rate, you must know the volume to be infused and the amount of time that the volume is to be infused. The following formula will allow you to calculate the proper flow rate:

$$\text{flow rate in drops per minute} = \frac{\text{volume to be infused} \times \text{drops per ml that the set delivers}}{\text{Infusion time in minutes}}$$

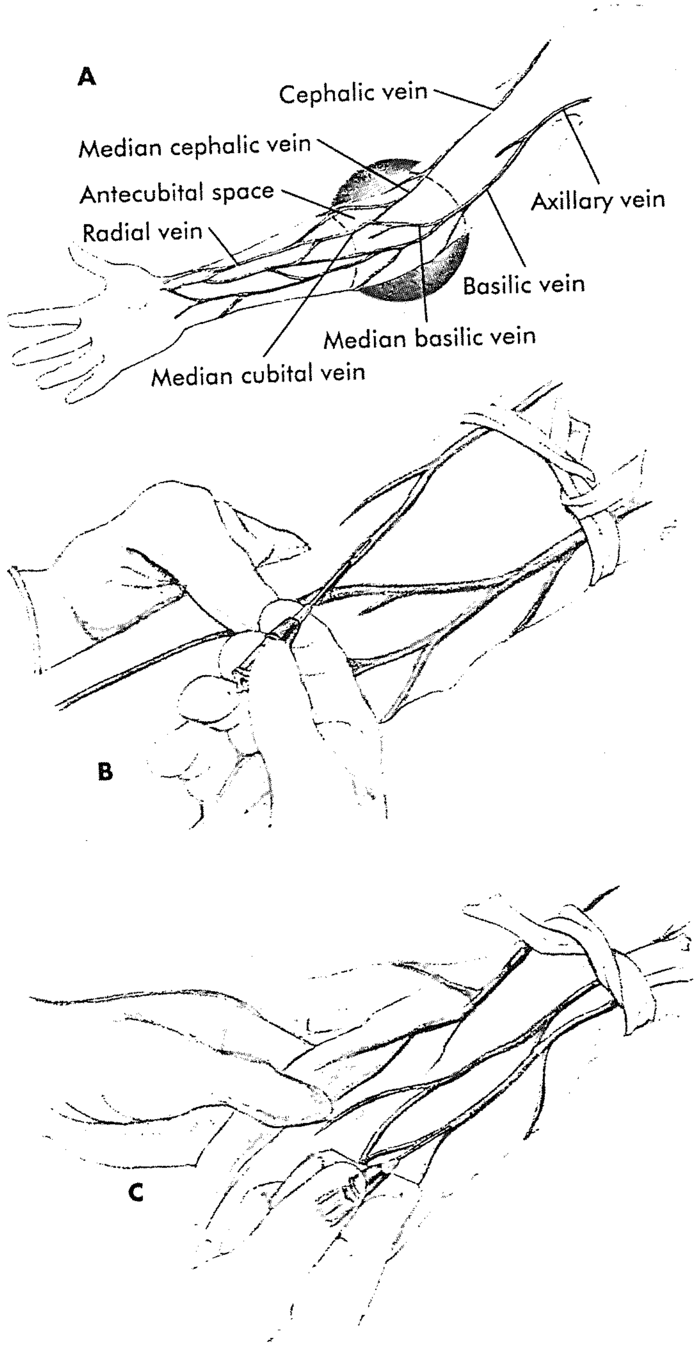


Fig. 13-26 A, Veins of the upper extremity. **B,** Antecubital venipuncture. **C,** Dorsal hand venipuncture.

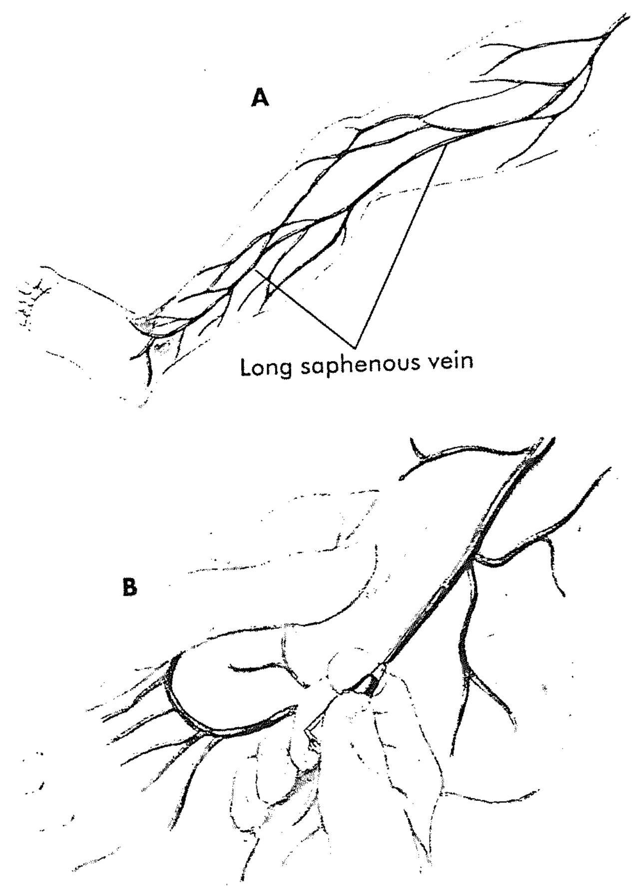


Fig. 13-27 A, Long saphenous vein. **B,** Venipuncture of the long saphenous vein.

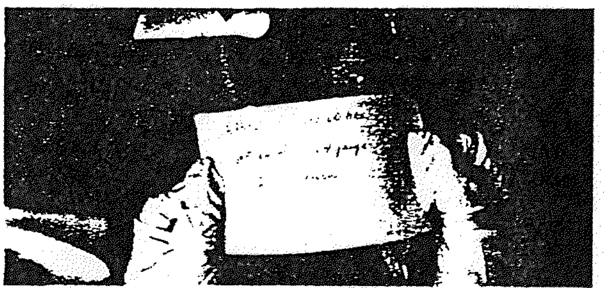


FIGURE A2-16 Label the bag.

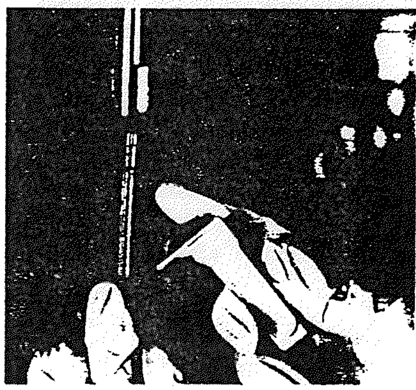


FIGURE A2-17 Turn on the IV and check the flow.

If the physician orders an infusion of 1 liter (1,000 ml) of normal saline in four hours, and the infusion set is capable of providing 10 drops per minute is calculated thus:

$$\frac{1000 \text{ ml} \times 10 \text{ (drops per ml)}}{240 \text{ minutes}} = \frac{42 \text{ drops per}}{\text{minute}}$$

The greater the pressure, the greater the flow. However, pressures greater than 250 mmHG to 300 mmHG may cause rupturing.

☐ MAINTAINING THE IV

The IV is fragile and must be handled with care. Carefully monitor the flow rate and make sure that the flow adjustment valve is working properly. Occasionally reposition the arm and inspect the tubing for kinks. Check fluid levels to make sure that you do not run out. Palpate the area around the IV to confirm that the IV is infiltrating the vein and not the tissues surrounding the vein.

If the IV stops dripping:

1. Check the tourniquet to make sure that you have released it.
2. Check the level of fluid in the bottle or container, and increase its height. The IV bottle should always be at least three feet above the insertion site.
3. Reposition the arm.
4. Check the tubing.
5. Check the catheter by pinching off the tubing a few inches ahead, then pinch and release the tubing between the kink and the catheter. You should see a reddish tinge of blood enter the line. If the catheter is plugged, radio the hospital and follow the physician's instructions. He will probably have you begin an IV at another location.
6. Check to see that the flow adjustment valve has not been accidentally closed.

☐ SIGNS TO LOOK FOR AT THE SITE

1. Check that the tape is holding the catheter secure and is not wet.
2. Ask the patient if there is any pain or burning at the IV site.
3. Check the skin to see if it is cool to touch around the site. If it is warm to the touch, there is probably or infiltration.
4. Make sure that the connection between the catheter and tubing is secure.

An infected IV site could cause complications. Signs of infection include:

1. A red line coming from the site (a hard red vein, indicating **phlebitis**) or *any* redness.
2. Any discharge at the site.
3. Any swelling around or above the site. This probably means that the IV catheter is out of the vein and that the fluid is escaping into the tissues. You must discontinue the IV immediately.

☐ TRANSPORTING THE IV PATIENT

When the stabilized patient is ready to be moved, elevate the fluid container well above the level of the heart via an IV pole or a well-instructed helper. If the fluid is in a bag rather than a bottle, the bag may be placed under the patient's head or shoulder until it can be hung up. The helper carrying the IV needs to stay at the infusion site as the patient is moved. Watch the IV continuously for complications.

Moving down a staircase or over rough terrain can dislodge an IV, so take steps to guard against accidental dislodging. You can safely stop the IV drip for two to three minutes if necessary and strap the fluid container to the patient to move over rough terrain. Do not exceed this limit, however, as the blood will clot and the IV will need to be restarted.

When the ambulance is reached, the IV carrier enters the compartment *before* the patient. Place the container on the hanger when possible. A quick check of the IV equipment and the patient should take place before the ambulance proceeds. Continually monitor vital signs during transport. When you arrive at the hospital, the IV helper takes the IV off of the holder. *Then* the patient can be unloaded, with the helper again at the infusion site.

It is necessary to transport a patient with an IV, make sure that all information about the patient and the fluid has been given to the ambulance team and that an IV-trained EMT is on the ambulance. Know what the physician wants you to do if the fluid runs out before the ambulance reaches its destination.

□ PATIENT ASSESSMENT PRIOR TO TRANSPORTATION[1]

Before transporting an IV patient to another facility, obtain a report about the patient from the nurse in charge. Also do a quick evaluation with the nurse present. It is of prime importance that you have as much information as possible regarding that patient, since any problem that may occur from then on is your responsibility. Be aware of and plan for the problems that can occur if the patient is anxious or confused. A confused patient can try to pull out the IV.

Figure A2-18 is the recommended documentation form that you need to fill out along with the nurse. This is another precaution. If, during the examination, you find something wrong, draw it to the attention of the nurse and tactfully ask the nurse to correct the problem. The documentation form is necessary and helpful for the following reasons:

- It provides a complete checklist for IV and other body tubings.
- You are able to use this checklist as a quick patient assessment.
- It validates any abnormalities that may be present.
- It provides continuity for health-care workers.
- The form filled out accurately is important for insurance claims and for verification of the health-care facility.
- It provides patient protection for optimum care.

[1] These sections were prepared with the help of Dave Dodds.

□ IV MEDICATION

Before transporting a patient with an IV infusing, make sure that you have the following information:

- Patient's name.
- Physician's name.
- The diagnosis.
- Allergies.
- List of medications previously administered.
- The name, dosage, drip rate, and amount of time over which the medication should be infused.
- The reason why the patient is receiving this medication (certain drugs can be administered for different reasons).
- The kind of solution and number of milliliters the container holds. The label on the solution is usually bright-colored. The label should contain the name of the patient, the name of the medication, the dosage, the date, and the time administration was started.
- Do not forget to check at the IV site and make a quick assessment of the body area for any rash that could be the beginning of an allergic reaction to the medication, along with a change in vital signs.

It is extremely important to know the following information about medications:

- The generic and chemical name.
- The classification.
- Indications for use.
- Adverse reactions.
- Normal dosage.
- Signs and symptoms of a reaction.
- What to do if a reaction occurs.

Important tip: At the time you obtain the medication information, write the facts on index cards and file them in an accessible place in your ambulance. You can then refer to them in the future (they are also handy for studying purposes). The dispatcher should have a current Physician's Drug Reference.

□ IV COMPLICATIONS

Three major complications that can result from infusion are infection, **pyrogenic reactions,** and phlebitis. These risks can be minimized with proper attention to technique.

Infection usually results from poor aseptic techniques. Being careful to *prevent contamination* is the key. A patient who has an IV in his or her vein has an open entry into his or her circulatory system.

To prevent contamination when working with an IV:

- Keep all possible equipment sterile.
- Use sterile or unsterile but clean gloves. Use of a surgical mask is also suggested.
- Examine equipment, solutions, and tubing for flaws.
- Always use aseptic or sterile technique.
- Remove rings and watches from the patient. The watch may act as a tourniquet.
- Always maintain sterility when opening packages or any IV equipment.
- Examine all packages and equipment for flaws.
- Inform the patient of the reasons for your precautions.

Pyrogens (foreign proteins) enter the body by way of contaminated fluid. If fluid shows leakage or cloudiness, *do not use it*. Pyrogenic reactions usually begin one-half hour after the IV is begun and present with the following:

- Abrupt fever.
- Severe chills.
- Backache, headache.
- Nausea, vomiting.
- Malaise.
- Shock, with a possibility of vascular collapse.

If these reactions occur, *stop the infusion immediately!* Begin a new IV with new equipment in the other arm. Treat for shock, and advise the physician by radio.

A misplaced needle (misses the vein or tears through it) will cause fluid to leak into the surrounding tissues. Visible and palpable swelling will occur, and the patient will experience a painful, burning sensation. Stop the IV and begin a new one in the other arm. Inform the physician of your actions.

Phlebitis

Phlebitis is the localized inflammation of a vein that leads to the formation of a small clot. As the clot grows, inflammation increases, partially or completely blocking the vessel, or detaching from the vessel and lodging elsewhere in the body. Phlebitis greatly increases the patient's risk of sepsis, as bacteria tends to accumulate at the site. Trauma, diabetes mellitus, age, or immunodeficiency can foster such an accumulation, rapidly leading to septic shock and death.

Patients in emergency departments have almost twice the risk of complications from IV therapy as patients whose IVs were initiated in other parts of the hospital. And patients whose IVs were begun in the field have over four and one-half times as much phlebitis than patients whose IVs were begun in the emergency room. Twenty-two percent also developed fevers — five and one-half times the percentage of those whose IVs were started in the emergency room. Even more alarming, symptoms of phlebitis can continue to develop even after the catheter has been removed and may not appear for days.[2]

The causes for these higher rates of complication seem to be incomplete decontamination, catheters that are too large, and rough insertions. In a field setting involving trauma, there must necessarily be trade-offs between ideal circumstances and speed. With trauma patients, using smaller catheters is not an option because of the need for rapid fluid infusion. But experts recommend that there be no attempt to speed up decontamination and that all IVs started in the field be removed and replaced in the hospital.

□ OTHER IV THERAPY COMPLICATIONS

Other complications may arise from an IV that is not started or tended to properly. Always check to see if the IV is positioned properly, and if the tourniquet is still on.

Plastic Embolus

A plastic embolus may be caused by withdrawing the needle from the catheter, then reinserting the needle, causing the sharp, beveled tip of the needle to cut off a small piece of the plastic catheter. Radio-opaque catheters are better than radiolucent for finding catheters that have been sheared off. However, the opaque catheters are more difficult to "slide" into the skin.

Air Embolus

An air embolus may result from a malfunction of the infusion line, or from allowing the fluid to run out completely, thus drawing air into the line via the air vent. The victim of an air embolus will rapidly develop shock and cyanosis and may possibly become unconscious. If an air embolus is suspected, use a hemostat to clamp the tubing close to the body. Place your patient on his left side, with legs elevated and head down. Inform the base physician. Lower the head of the stretcher or bed. Give oxygen and transport to the nearest emergency room.

---

[2] David Lawrence, "Prehospital IV Therapy," *JEMS*, January 1990, pp. 51–52.

Circulatory Overload

Circulatory overload, or too much fluid in the circulatory system, can be caused by a "runaway" IV, or by an IV that provides too much fluid. This may force fluid into the lungs, causing pulmonary edema. Signs of circulatory overload are:

- Venous distention.
- Raise in blood pressure.
- Shortness of breath.
- Coughing.
- Increased respiratory rate.
- Dyspnea.
- Frothy sputum resulting from fluid builuup in the lungs.
- Cyanosis.

If these signs are present:

1. Use a microdrip.
2. Elevate the patient's head.
3. Turn the IV to **TKO** (to keep open). Leave the IV inserted, as the patient will probably need it for IV medications such as **Lasix,** which is used to rid the body of fluid.
4. Notify the physician immediately. Monitor the patient closely, be prepared to give emergency care, and document the entire procedure.

Allergic Reactions

If your patient has an IV medication infusing or has an additive to his or her IV, be alert to a possible allergic reaction. Watch for the following signs:

- Itching.
- Rash.
- Shortness of breath.
- Anaphylactic shock can develop.

If there is a medication infusing:

1. Clamp off.
2. *Do not discontinue the IV,* but slow down to TKO.
3. Monitor the patient closely.
4. Be prepared to give emergency care.
5. Transport to the nearest emergency room.
6. Document the entire procedure.
7. A medical doctor may order fluid or a medication change.

Infiltration

Infiltration means the escape of IV fluids into the surrounding tissues, which can cause tissue damage and **necrosis.** If the IV solution contains a drug toxic to subcutaneous tissue, it can be disastrous; it could require reconstructive surgery.

It is of utmost importance to monitor the IV site for edema, pain, and temperature. The area above the IV site may feel cooler or warmer. Look for leakage of fluid around the site. Another sign could be a sluggish flow rate.

Stabilize the extremity with the IV. It is important for the extremity with the IV to be still. Use of the catheter over the needle rather than the butterfly will reduce the occurrence of damage with movement. If infiltration occurs, stop the IV and begin a new one in the other arm. Inform your base physician of your actions.

Blood Back-up In Tubing

During your observation, you may notice blood beginning to back up in the tubing and/or possibly a clot at the end of the catheter. Look for the obvious first, remembering that a blood back-up or clotting usually occurs due to a slow or absent flow rate or improper placement of the IV solution container. Also, if you forget to flush the IV, the blood will run up the tubing. If this happens, the tube must be unhooked from the IV, flushed, then reconnected.

Start at the top. Check to see that the IV container is not empty. Is it elevated enough? How is the flow rate? If the purpose of the IV is TKO (to keep open), that can be a factor. Do not forget that a TKO drip rate needs to be wide open for one to two seconds, every one-half to one hour. Is the drip chamber half full? Check the flow clamp for position. Observe if the tubing in kinked, or if the tubing is dangling and preventing the solution from reaching the patient.

Next, check the IV site. Are any signs present that might explain the problem, such as the catheter being lodged against the vein wall? Gently move the catheter slightly. You may have to attempt to aspirate the clot out of the catheter with a sterile syringe. Never irrigate IV if you cannot aspirate a clot, for this could cause an embolus. Remove the IV and start a new one.

Cold

IV solutions can freeze in the tubing or container very rapidly. You may want to start an IV in the ambulance or in a heated building, if possible. Protect the tubing and container from cold during transport. If a patient in hypovolemic shock is receiving large volume of fluid, warm them to body temperature or you may cause the patient's core temperature to drop, triggering hypothermia.

☐ BLOOD TRANSFUSIONS

As an EMT, you do not normally transport blood transfusion patients. If you feel uncomfortable or if the medication maintenance does not fall within the realm of your duty, ask the facility to send a nurse or physician to perform those duties/skills.

☐ IV DISCONTINUATION

It is important to evaluate the circumstances before you decide to discontinue the IV. Use the following guidelines:

- Discontinue an IV immediately if the fluid is going into the tissues, not the vein.
- With a clotted-off IV, it is possible (if your arrival to the other facility is within five minutes) to wait for another opinion before you discontinue.
- If your patient has thrombophlebitis (signs include sluggish flow rate, edema around the IV site, and a vein that looks like a red line; the vein will be hard, warm, and sore), you *must* discontinue the IV.

To discontinue an IV:

1. Explain to the patient why his or her IV needs to be discontinued. Also, explain that he or she will probably need to have another one inserted upon arrival at the other facility.
2. Gather all equipment: two 2 × 2s or 4 × 4s and tape.
3. Whenever blood or body fluids are being handled, wear protective clothing.
4. Open your packages and prepare two pieces of tape about three inches long.
5. Clamp off the IV.
6. Loosen all the tape on the IV site.
7. Stabilize the extremity and hub.
8. Gently pull out the catheter and apply pressure immediately upon removal.
9. Place a 2 × 2 on the IV site and hold pressure for two minutes to prevent a hematoma from forming. (Be aware of patients with clotting problems — you may have to apply a pressure dressing.)
10. Apply a 2 × 2, and tape.
11. If infiltration is present, elevate the extremity on a pillow.
12. Apply a warm, moist pack when possible.
13. Document and record:
    - Amount of fluid left in the bag.
    - Amount of fluid the patient received.
    - Time of discontinuation of the IV.
    - Any other problems.
14. Dispose of used needles in a Sharps container (Figure A2-19).

FIGURE A2-19 Dispose of used needles in a Sharps container.

☐ COMMUNICATION WITH THE EMERGENCY DEPARTMENT

During IV therapy, it is essential that you communicate effectively with personnel in the emergency department. Repeat all orders verbally to the emergency department so that everyone understands what has been ordered.

contact with a semipermeable membrane across which water-soluble substances diffuse into a dialyzing fluid (dialysate). After an interval, equilibration of the patient's blood with the dialysate normalizes the electrolyte composition, and waste products are eliminated.

The amount of substance that transfers during dialysis depends on the difference between the concentration on the two sides of the semipermeable membrane, the molecular size of the substance, and the length of time the blood and the dialysate remain in contact with the membrane. In patient's with end-stage renal disease, hemodialysis is usually performed 3 times a week for 4 to 5 hours each time.

Hemodialysis

In hemodialysis, the patient's heparinized blood is pumped through a surgically constructed arteriovenous fistula, which is an internal anastomosis between an artery and a vein, or an arteriovenous graft, which is a synthetic material grafted to the patient's artery and vein (Fig. 21-5). These "internal shunts" are usually located in the inner aspect of the patient's forearm or much less commonly in the medial aspect of the lower extremity. Other patients may have a small, button-shaped device **(hemasite)** usually located in the upper arm or proximal, anterior thigh. A hemasite is similar to an arteriovenous graft but has an external rubber septum sutured to the skin through which a dialysis catheter is inserted for treatment.

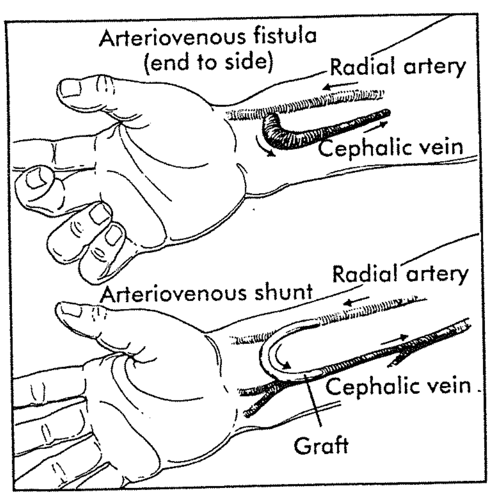


Fig. 21-5 Arteriovenous fistula.

Peritoneal Dialysis

In peritoneal dialysis, the dialysis membrane is the patient's own peritoneum. The dialysate is infused into the peritoneal cavity by a temporary percutaneous or permanently implanted catheter. Fluid and solutes diffuse from the blood in the peritoneal capillaries into the dialysate. After 1 to 2 hours, when equilibration has occurred, the dialysate is drained and fresh fluid is infused. Peritoneal dialysis works considerably more slowly than hemodialysis, but over time it is just as effective and does not require chronic blood access. A major complication of peritoneal dialysis is peritonitis, which usually results when the procedure is not performed with proper aseptic technique.

Dialysis Emergencies

Emergencies the paramedic may encounter when caring for a patient with acute or chronic renal failure may result from the disease process itself or from complications of dialytic therapy. For example, these patients may experience problems associated with vascular access, hemorrhage, hypotension, chest pain, severe hyperkalemia, disequilibrium syndrome, and the development of an air embolism. In addition, the paramedic should be aware of problems that may result from concurrent medical illness and its treatment. These include decreased ability to tolerate the stress of significant illness or trauma, inadvertent overadministration of intravenous fluid, and altered metabolism and unpredictable action of drugs.

Vascular Access Problems

Problems associated with vascular access are bleeding at the site of puncture for dialysis, thrombosis, and infection. Bleeding from the fistula or graft is usually minimal and can generally be controlled by direct pressure at the site. (However, excessive pressure can cause thrombosis in the graft or fistula.) A potential complication of an internal shunt is development of a pseudoaneurysm,

Students are directed not to access AV Graft/Fistula.

an
kin
for

e is
in-
ary
ter.
the
to
lia-
eri-
wly
ef-
ess.
eri-
ure
te.

iter
re-
s it-
For
ems
hy-
dis-
f an
ould
con-
e in-
sig-
nin-
abo-

are
om-
a or
con-
, ex-
graft
rnal
ysm,

which can rupture, causing a large hematoma and possible hypovolemia. If this occurs, direct pressure should be applied to the hematoma, and the patient should be assessed and treated for significant blood loss. This situation requires rapid transport for physician evaluation.

Fistulas and grafts that become occluded as a result of thrombus formation usually require surgical intervention or the administration of a thrombolytic agent to restore flow. Patients with a surgical anastomosis are instructed to periodically check for the presence of a bruit or "thrill" to verify unobstructed circulation. Attempts to clear the graft by irrigation or aspiration are not generally recommended. If thrombosis occurs while the patient is undergoing dialysis, the dialysis should be stopped and intravenous fluids initiated in an alternative site. Decreased blood flow is a common precipitating cause of thrombosis and is a main reason that blood pressure is not obtained in the arm with a vascular access.

Infection at the site of vascular access is usually the result of the puncture made during dialysis. Therefore meticulous sterile technique is mandatory when caring for these patients, and routine vascular access using this route should be discouraged. Vascular access infection should be considered when a dialysis patient has unexplained fever, malaise, or other signs of systemic infection.

Hemorrhage

Patients receiving dialysis have an increased risk of hemorrhage because of their regular exposure to anticoagulants and the decrease in their platelet function. Therefore a patient who experiences hemorrhage from trauma or a medical condition (for example, gastrointestinal bleeding) should be closely monitored for signs of hypovolemia. Most patients on dialysis have a baseline anemia that lowers their reserves when they have acute hemorrhage. Any significant blood loss (whether external or internal) may manifest as dyspnea or angina. If hemorrhage from trauma occurs in an extremity with a fistula or graft, bleeding should be controlled and the extremity immobilized in the normal fashion, using special care to try to avoid obstructing circulation in the anastomosis.

Hypotension

Hypotension is not infrequently associated with hemodialysis. This may result from the rapid reduction in intravascular volume, abrupt changes in electrolyte concentrations, or vascular instability that may occur during the procedure. In addition, the patient's compensatory mechanisms to cope with these physiological alterations may be impaired, resulting in an inability to maintain normal blood pressure. Patients with hypotension caused by dialysis must be cautiously managed with the administration of volume-expanding fluids. The paramedic should be careful not to produce a fluid overload, which may manifest as hypertension and the classic signs of congestive heart failure (pulmonary edema, shortness of breath, crackles, engorged neck veins, liver congestion and engorgement, and pitting edema). Most patients respond to a relatively small (200- to 300-ml) fluid challenge. If they do not, other potentially serious etiologies should be considered.

Chest Pain

The episodes of hypotension and mild hypoxemia that commonly occur during dialysis may result in myocardial ischemia and chest pain. The patient may also complain of other symptoms associated with decreased oxygen delivery, such as headache and dizziness. Although these complaints may indicate an evolving myocardial infarction, they are often relieved with the administration of *oxygen,* fluid replacement, and antianginal medications. Regardless, all patients with chest pain should be treated as though a myocardial infarction has occurred.

Dysrhythmias resulting from myocardial ischemia may also be associated with dialysis. The most common ischemic rhythm disturbances are premature ventricular contractions, which generally respond well to the administration of supplemental *oxygen* and *lidocaine.* If dialysis is in progress, the procedure should be discontinued, and the paramedic should consult with medical control.

Imperial Valley College
Emergency Medical Services Training
(Indications/Dosage/Route Based on Imperial County Protocols)

0.9% Sodium Chloride Solution (Normal Saline)

Class:
- Crystalloid Solution

Actions:
- Increases circulating volume by remaining in the circulatory system
- Electrolyte solution which is osmotically equivalent to blood

Indications:
- Dilution of IV drip medications
- Life-line for administration of medications
- Fluid replacement
- Only IV solution listed in Imperial County Protocols

Dosage/Route:
- IV drip piggyback/TKO/Wide Open/fluid challenge/calculated drip rate

Side Effects:
- None with normal use

Contraindications:
- None

Special Information:
- Contains: 154 meq sodium/liter and 154 meq chloride/liter
- Use conservatively in patients with suspected head injury to decrease the development of cerebral edema
- Monitor patient to determine if fluid overload is developing
- Standing order for the adult patient whenever IV is indicated

Pediatric Note:
- Refer to Pediatric Drug Guide
- 20 ml/kg initial bolus via volutrol rapid IV drip SO for:
  - Cardiac arrest (non-traumatic)
  - PEA
  - Shock/hypotension (non-traumatic)
  - Trauma
- 20 ml/kg initial bolus via volutrol SO for:
  - Environmental emergencies
- May repeat per BH

DATE: 11/01/01

OPERATIONS: ADVANCED EMT TREATMENT PROTOCOLS

SUBJECT: CHEST PAIN (Suspected Cardiac Origin)

POLICY NUMBER: 2070

BLS	ALS
Ensure patent airway, give oxygen and/or ventilate prn. Do not allow patient to walk or exert self	**SO** Nitroglycerin 0.4 mg (1/150 gr) SL q 5 min. X 3 if BP > 90 systolic additional NTG per BH **SO** Aspirin 320 mg PO **SO** Establish IV TKO **<u>HYPOTENSION</u>** **<u>(suspected Cardiogenic Shock or NTG reaction)</u>** **SO** Fluid challenge 100-250 mL with clear lungs; may repeat per BH

NOTE: For suspected thoracic aortic aneurysm, transport immediately.

APPROVAL:

Bruce E. Haynes, M.D.
EMS Medical Director

DATE: 11/01/01

OPERATIONS: ADVANCED EMT TREATMENT PROTOCOLS

SUBJECT: ENVIRONMENTAL EMERGENCIES

POLICY NUMBER: 2090

BLS

Remove patient from hostile environment
Ensure patent airway
Give oxygen and/or ventilate prn.

COLD EXPOSURE:
Remove wet clothing
Handle patient gently and avoid unnecessary movement
Institute gentle warming with blankets or warm packs
Do not apply heat directly to the skin or rub the injured areas
Apply dressings to blistered or necrotic areas
Prolonged CPR may be indicated
If alert, give warm fluids; if altered LOC - NPO

HEAT EXHAUSTION:
Loosen or remove clothing
Cool gradually (fanning, massaging, sponging tepid water); avoid shivering
If conscious and no nausea, give small amounts of cool liquids (preferably water)

HEAT STROKE:
Rapid cooling: loosen/remove clothing
Ice packs to axillae, groin,
Flush with cold water, fan patient
Avoid shivering

ALS

SO Establish IV prn.

HEAT EXHAUSTION/STROKE:
SO Consider fluid challenge of 500 mL NS (if clear lungs)
May repeat per BH, limit 2 liters

APPROVAL:

Bruce E. Haynes, M.D.
EMS Medical Director

IMPERIAL COUNTY EMERGENCY MEDICAL SERVICES AGENCY
POLICY/PROCEDURE/PROTOCOL

DATE: 11/01/01

OPERATIONS: ADVANCED EMT TREATMENT PROTOCOLS

POLICY NUMBER: 2160

SUBJECT: SHOCK/HYPOTENSION (non-traumatic)

BLS

Ensure patent airway, give oxygen and/or ventilate prn.
Keep warm
Nothing by mouth
Remove dermal NTG prn.
If not contraindicated, place patient supine with legs elevated

NOTE: Do not use Trendelenberg position

ALS

SO Establish IV TKO
SO For mild hypotension, give fluid bolus 250-500 mL NS; may repeat per BH order
SO For profound hypotension, establish 2 IVs (preferably enroute) run wide open to max 2 liters
Run IVs to maintain systolic BP 90 mm Hg; additional fluids per BH order

See Chest Pain Protocol for cardiogenic shock.

APPROVAL:

Bruce E. Haynes, M.D.
EMS Medical Director

DATE: 11/01/01

OPERATIONS: ADVANCED EMT TREATMENT PROTOCOLS

POLICY NUMBER: 2170

SUBJECT: TRAUMA

BLS

Ensure patent airway, give oxygen and/or ventilate prn.
Maintain spinal immobilization prn.
Control external bleeding
Immediate transport if patient critical

ABDOMINAL TRAUMA

Cover eviscerated bowel with saline soaked pads
Flex hips and knees if not contraindicated

CHEST TRAUMA

Cover open chest wound with three-sided occlusive dressing; release dressing if suspected tension pneumothorax develops

EXTREMITY TRAUMA

Splint fractures as they lie if no neurovascular impairment
Splint dislocations in position found
Immobolize joints above and below injury, if possible
Fractures with neurovascular impairment may be aligned with gentle, unidirectional traction before splinting
If circulation is not restored after two attempts at straigthening, splint as it lies and transport immediately

ALS

SO Establish IV (preferably enroute); bilateral IV's wide open for hypovolemic shock to max 2 liters
Adjust rate per vital signs; target systolic BP 80-90 mm Hg (except head trauma)
Additional fluids per BH

HEAD TRAUMA

SO If GCS less than or equal to 8, maintain BP at or above 100 systolic with IV fluids

BLS

TRAUMATIC ARREST
Consider Determination of Death Protocol
If in doubt, initiate CPR
Assist ventilations with cervical in-line stabilization (if applicable)
See Policy #2060 for use of AED

IMPALED OBJECTS
Immobilize (exceptions: may remove object if in face or neck and ventilation is compromised; if object interferes with CPR; or if object interferes with transport)

AMPUTATED PARTS
Place in plastic bag and keep cool during transport
Do not place in water or directly on ice

OPEN NECK WOUNDS
Cover with occlusive dressing

ALS

TRAUMATIC ARREST

BHP	Consider discontinuing CPR in blunt trauma
SO	Insert Combitube with in-line stabilization if indicated
SO	Establish 2 IVs while enroute, run wide open to max 2 liters; additional fluids per BH order

BLS	ALS
HELMETS to include full face motorcycle helmets and football helmets: **<u>Indications for removing helmets in the field:</u>** Inability to assess and/or reassess airway and breathing Inability to adequately manage airway and breathing Improperly fitted helmet allowing for excessive movement of head Proper spinal immobilization cannot be performed due to helmet Cardiac arrest NOTE: When removing football helmet, may be necessary to remove shoulder pads as well to properly immobilize spine	

APPROVAL:

Bruce E. Haynes, M.D.
EMS Medical Director

EMT-II MODULAR TRIAL STUDY

CLINICAL EVALUATION FROM

IMPERIAL COUNTY EMS AGENCY
EMT-II Modular Trial Study

CLINICAL SKILL EVALUATION FORM

IV CANNULATION & FLUID THERAPY

1. Gathers appropriate equipment.	( ) YES	( ) NO
2. Takes appropriate body substance isolation precautions.	( ) YES	( ) NO
3. Properly prepares IV bag & tubing.	( ) YES	( ) NO
4. Properly applies constricting band to extremity.	( ) YES	( ) NO
5. Selects appropriate size catheter.	( ) YES	( ) NO
6. Properly cannulates vein using aseptic technique.	( ) YES	( ) NO
7. Connects IV tubing firmly to cannula.	( ) YES	( ) NO
8. Releases constricting band.	( ) YES	( ) NO
9. Sets proper IV flow rate.	( ) YES	( ) NO
10. Properly disposes of contaminated sharp.	( ) YES	( ) NO

Total number of IV attempts this shift: ____________

Total number of IVs established successfully this shift: ____________

COMMENTS: ____

____

____

____

____

____

Student Name: ____________________ Date: ____________

Evaluator's Signature: ____________________ Title: ____________

IMPERIAL COUNTY EMS AGENCY
EMT-II Modular Trial Study

CLINICAL SKILL EVALUATION FORM

IV PUSH MEDICATION

1. Asks patient for known allergies. ( ) YES ( ) NO
2. Selects correct medication. ( ) YES ( ) NO
3. Assures correct concentration of drug. ( ) YES ( ) NO
4. Draws up medication or assembles pre-filled syringe correctly. ( ) YES ( ) NO
5. Cleanses injection site (Y-port or hub). ( ) YES ( ) NO
6. Inserts needle/connects needeless port. ( ) YES ( ) NO
7. Pinches tubing (aspirates for blood return - Dextrose only). ( ) YES ( ) NO
8. Administers correct dose at proper push rate. ( ) YES ( ) NO
9. Flushes tubing (runs wide open for a brief period). ( ) YES ( ) NO
10. Adjusts drip rate to TKO. ( ) YES ( ) NO
11. Properly disposes of syringe and needle. ( ) YES ( ) NO
12. Observes patient for desired effect/adverse side effects. ( ) YES ( ) NO

COMMENTS: ______________________________

____

____

____

____

____

Student Name: ______________________ Date: ____________

Evaluator's Signature: ______________________ Title: ____________

EMT-II MODULAR TRIAL STUDY

FINAL SKILL EVALUATION

National Registry of Emergency Medical Technicians
Advanced Level Practical Examination
INTRAVENOUS THERAPY

Candidate:______________________ Examiner:______________________

Date:______________________ Signature:______________________

Time Start:____________ Time End:____________

	Possible Points	Points Awarded
Checks selected IV fluid for: - Proper fluid (1 point) - Clarity (1 point)	2	
Selects appropriate catheter	1	
Selects proper administration set	1	
Connects IV tubing to the IV bag	1	
Prepares administration set [fills drip chamber and flushes tubing]	1	
Cuts or tears tape [at any time before venipuncture]	1	
Takes/verbalizes infection control precautions [prior to venipuncture]	1	
Applies tourniquet	1	
Palpates suitable vein	1	
Cleanses site appropriately	1	
Performs venipuncture - Inserts stylette (1 point) - Notes or verbalizes flashback (1 point) - Occludes vein proximal to catheter (1 point) - Removes stylette (1 point) - Connects IV tubing to catheter (1 point)	5	
Releases tourniquet	1	
Runs IV for a brief period to assure patent line	1	
Secures catheter [tapes securely or verbalizes]	1	
Adjusts flow rate as appropriate	1	
Disposes/verbalizes disposal of needle in proper container	1	
TOTAL	21	

CRITICAL CRITERIA

___ Exceeded the 6 minute time limit in establishing a patent and properly adjusted IV
___ Failure to take or verbalize infection control precautions prior to performing venipuncture
___ Contaminates equipment or site without appropriately correcting situation
___ Any improper technique resulting in the potential for catheter shear or air embolism
___ Failure to successfully establish IV within 3 attempts during 6 minute time limit
___ Failure to dispose/verbalize disposal of needle in proper container

You must factually document your rationale for checking any of the above critical items on the reverse side of this form.

National Registry of Emergency Medical Technicians
Paramedic Practical Examination
INTRAVENOUS BOLUS MEDICATIONS

Candidate:______________________________

Date:______________________________

Examiner:______________________________

Signature:______________________________

Time Start:____________ Time End:____________

NOTE: Check here (____) if candidate did not establish a patent IV and do not evaluate these skills.

	Possible Points	Points Awarded
Asks patient for known allergies	1	
Selects correct medication	1	
Assures correct concentration of drug	1	
Assembles prefilled syringe correctly and dispels air	1	
Continues infection control precautions	1	
Cleanses injection site (Y-port or hub)	1	
Reaffirms medication	1	
Stops IV flow (pinches tubing)	1	
Administers correct dose at proper push rate	1	
Flushes tubing (runs wide open for a brief period)	1	
Adjusts drip rate to TKO (KVO)	1	
Voices proper disposal of syringe and needle	1	
Verbalizes need to observe patient for desired effect/adverse side effects	1	

CRITICAL CRITERIA

IV BOLUS SUB-TOTAL 13 [ ]

___Failure to begin administration of medication within 3 minute time limit

___Contaminates equipment or site without appropriately correcting situation

___Failure to adequately dispel air resulting in potential for air embolism

___Injects improper drug or dosage (wrong drug, incorrect amount, or pushes at inappropriate rate)

___Failure to flush IV tubing after injecting medication

___Recaps needle or failure to dispose/verbalize disposal of syringe and needle in proper container

INTRAVENOUS PIGGYBACK MEDICATIONS

	Possible Points	Points Awarded
Has confirmed allergies by now (award point if previously confirmed)	1	
Checks selected IV fluid for: - Proper fluid (1 point) - Clarity (1 point)	2	
Checks selected medication for: - Clarity (1 point) - Concentration of medication (1 point)	2	
Injects correct amount of medication into IV solution given scenario	1	
Connects appropriate administration set to medication solution	1	
Prepares administration set (fills drip chamber and flushes tubing)	1	
Attaches appropriate needle to administration set	1	
Continues infection control precautions	1	
Cleanses port of primary line	1	
Inserts needle into port without contamination	1	
Adjusts flow rate of secondary line as required	1	
Stops flow of primary line	1	
Securely tapes needle	1	
Verbalizes need to observe patient for desired effect/adverse side effects	1	
Labels medication/fluid bag	1	

CRITICAL CRITERIA

IV PIGGYBACK SUB-TOTAL 17 [ ]

___ Failure to begin administration of medication within 5 minute time limit

___ Contaminates equipment or site without appropriately correcting situation

___ Administers improper drug or dosage (wrong drug, incorrect amount, or infuses at inappropriate rate)

___ Failure to flush IV tubing of secondary line resulting in potential for air embolism

___ Failure to shut-off flow of primary line

You must factually document your rationale for checking any of the above critical items on the reverse side of this form.

EMT-II MODULAR TRIAL STUDY

QUALITY IMPROVEMENT

IMPERIAL COUNTY EMT-II MODULAR TRIAL STUDY FORM

RUN REVIEW *(to be completed by EMT-II)*

Date: ___/___/___ **BH Run #** ______________

Patient Age: ________ **Sex** ☐ **M** ☐ **F**

Chief Complaint: ____

EMT-II Agency: ______________________________ ☐ First Responder ☐ Ambulance

EMT-II Name: ______________________________ **Certification #** ______________

Time EMT-II Arrived on Scene: ____________

Time patient turned over to ALS provider: ____________

Location of ALS meet: ☐ On Scene ☐ Enroute rendezvous ☐ At hospital

EMT-II Ambulance Provider Only: Total Time on Scene: ____________

If scene time greater than 20 minutes, explain extenuating circumstances: ______________

____

Receiving Facility: ☐ PMH ☐ JFK ☐ ECRMC ☐ Other: ______________

SKILLS & MEDICATIONS *(to be completed by EMT-II)*

1. **Determination of Blood Glucose*:*** Value: __________
2. **Intravenous Access (Normal Saline):** # of Attempts:__________

   Successful: ☐ Yes ☐ No Amount Infused: __________
3. **Dextrose 50%** Amount given: ______________

   Patient Response: ☐ Improved ☐ No Change ☐ Worse

EVALUATION (*To be completed by receiving EMT-P/RN/MD*)

Was IV patent upon arrival? ☐ **Yes** ☐ **No**

Was IV properly assembled and secured upon arrival? ☐ Yes ☐ No

Comments: ____

Imperial County EMS Use Only

Date Received: ___/___/___ **File Number:**____________

How did procedure(s) effect the overall patient condition?

☐ Improved ☐ No Change ☐ Worse

EMS Medical Director (signature): ______________________________