

# **Allergies and Poisoning**

**EMS Continuing Education  
Technician through Technician-Advanced Paramedic**

**Consistent with the  
National Occupational Competency Profiles  
as developed by  
Paramedic Association of Canada  
and  
“An Alternate Route to Maintenance of Licensure”  
as developed by Manitoba Health**

**Evaluated for content by:  
Dr. W. VanDyk**

**Developed by:  
Educational Subcommittee – Paramedic Association of  
Manitoba**

**Revised August 2011**

## **Disclaimer**

These documents were developed for improved accessibility to “An Alternative Route to Maintenance of Licensure” for all paramedics in Manitoba. Regional implementation of Alternate Route is at the discretion of the local EMS Director.

This is a supportive document to the National Occupational Competency Profiles and “An Alternative Route to Maintenance of Licensure.” It is not the intent that this package be used as a stand-alone teaching tool. It is understood that the user has prior learning in this subject area, and that this document is strictly for supplemental continuing medical education. To this end, the Paramedic Association of Manitoba assumes no responsibility for the completeness of information contained within this package.

It is neither the intent of this package to supercede local or provincial protocols, nor to assume responsibility for patient care issues pertaining to the information found herein. Always follow local or provincial guidelines in the care and treatment of any patient.

This package is to be used in conjunction with accepted models for education delivery and assessment, as outlined in “An Alternative Route to Maintenance of Licensure”.

This document was designed to encompass all licensed training levels in the province Technician, Technician-Paramedic, Technician-Advanced Paramedic. Paramedics are encouraged to read beyond their training levels. However, the written test will only be administered at the paramedic’s current level of practice.

All packages have been reviewed by the Paramedic Association of Manitoba’s Educational Subcommittee and physician(s) for medical content.

As the industry of EMS is as dynamic as individual patient care, the profession is constantly evolving to deliver enhanced patient care through education and standards. The Paramedic Association of Manitoba would like to thank those practitioners instrumental in the creation, distribution, and maintenance of these packages. Through your efforts, our patient care improves.

This document will be amended in as timely a manner as possible to reflect changes to the National Occupational Competency Profiles, provincial protocols/Emergency Treatment Guidelines, or the Cognitive Elements outlined in the Alternate Route document.

Any comments, suggestions, errors, omissions, or questions regarding this document may be referred to [info@paramedicsofmanitoba.ca](mailto:info@paramedicsofmanitoba.ca) , attention Director of Education and Standards.

# **Allergies and Poisoning**

## **Conventions Used in this Manual**

Black lettering without a border is used to denote information appropriate to the Technician Level and above.

|| Text with the single striped border on the left is information appropriate to Technician-Paramedic and above.

||| Text with the double striped border on the left is information appropriate to Technician-Advanced Paramedic and above.

## **Table of Contents**

Routes of Exposure: .....	4
Ingested (gastrointestinal):.....	4
Inhaled (respiratory):.....	4
Injection (vascular):.....	4
Absorption (topical): .....	5
Poison Information Centers .....	5
General Principles of Toxicologic Management .....	6
Scene Survey .....	6
Primary and Secondary Survey .....	6
Ingested Poisons .....	6
General Principles of Assessment.....	6
General Principles of Management.....	8
Inhaled Poisons.....	8
Presentation of Toxic Inhalations.....	8
General Management of Toxic Inhalations .....	9
Specific Inhaled Toxins .....	10
Surface Absorbed Poisons .....	11
General Principles of Management.....	11
Treatment.....	12
Drug Overdose and Substance Abuse .....	13
Alcohol Abuse .....	13
Allergies and Anaphylaxis .....	16
The Immune System .....	16
Allergies .....	17
Pharmacological agents: .....	21
Epinephrine.....	21
Atropine Sulfate.....	24
Naloxone .....	26
Flumazenil .....	27
References.....	29

## **Routes of Exposure:**

In order to have a destructive effect, poisons must gain entrance into the body. The 4 portals of entry are ingestion, inhalation, injection and absorption. It is important to note that regardless of the portal of entry, toxic substances may have both immediate and delayed effects.

### **Ingested (gastrointestinal):**

This is the most common route of poisoning. Frequently ingested poisons include: household products, petroleum based agents (gasoline, paint), cleaning agents, cosmetics, medications (prescription, non-prescription and illicit), plants and foods. Toxic ingestions can involve burns to the lips, tongue, throat and esophagus. Delayed effects can result from absorption of the poison in the gastrointestinal tract, immediate and delayed depending on the substance and the amount ingested.

### **Inhaled (respiratory):**

Inhaling a poison results in fast absorption in the alveolar-capillary membrane. The inhalation of toxins can cause pulmonary edema or destroy tissues. The absorption of toxins at the alveolar level can affect other body systems.

Commonly inhaled poisons include:

- Toxic gases
- Carbon monoxide
- Ammonia
- Chlorine
- Freon
- Toxic vapors, fumes, or aerosols
- Carbon tetrachloride
- Methylene chloride
- Tear gas (pepper and non-pepper lacrimators)
- Mustard gas
- Nitrous oxide

### **Injection (vascular):**

Injection of a toxic agent under the skin, into muscle, or into the circulatory system also may result in both immediate and delayed effects. The immediate local reaction is at the site of the injection. Later, as the toxin is distributed throughout the body by the circulatory system, delayed systemic reactions can occur. In addition, an allergic or anaphylactic reaction may appear.

Other than intentional injection of illicit drugs, most poisonings by injection result from the bites and stings of insects and animals. Most insects that sting and bite belong to the class hymenoptera, which includes bees, hornets, yellow jackets, wasps, and ants. Only the females in this group can sting. In addition, spiders, ticks, and other arachnids, such as scorpions, notoriously cause poisonings by injection. Higher life forms that bite and sting include snakes and marine animals. Marine animals with venomous stings include jellyfish (especially the Portuguese man-of-war), stingrays, anemones, coral, hydras, and certain spiny fish.

### **Absorption (topical):**

Surface absorption is the entry of a toxic substance through the skin. This most frequently occurs from contact with poisonous plants, such as poison ivy, poison sumac, and poison oak. Many toxic chemicals may also be absorbed through the skin. The organophosphates, often used as insecticides, are easily absorbed through dermal contact.

### **Poison Information Centers**

Poison information centers have been set up across the United States and Canada to assist in the treatment of poison victims and to provide information on new products and new treatment recommendations. They are usually based in major medical centers and teaching hospitals that serve a large population. Most poison control centers now have computer systems that access information rapidly.

Poison information centers are usually staffed by physicians, pharmacists, nurses, or poison control specialists trained in toxicology. These experts provide information to callers 24 hours a day. They update information routinely and offer the most current treatment protocols.

Memorize the number of the nearest poison information center and routinely access it. There are several advantages to this practice. First, the poison control center can help you immediately determine potential toxicity based on the type of agent, amount and time of exposure, and physical condition of the patient. Second, in about four out of five cases, the most current, definitive treatment can be started in the field. Finally, the poison information center can notify the receiving hospital of current treatment even before arrival of the patient.

Poison Control

- emergency- 787-2591

- inquiries- 787-2444

## **General Principles of Toxicologic Management**

Although specific protocols may vary for managing toxicological emergencies, certain basic principles apply to most situations.

### **Scene Survey**

Begin assessment with a thorough evaluation of the scene. Be alert for potential hazards to the rescuer. In toxicological emergencies, put rescuer safety first. Despite a strong desire to assist and treat patients, you will be useless if incapacitated. To ensure rescuer safety, keep the following tips in mind.

Suicidal patients have the potential for violence. Therefore, look for signs of overdose such as empty pill bottles.

In chemical emergencies or spills involving hazardous material, make sure the proper clothing and equipment are available. Assign these materials to rescuers who have been trained in their use.

By taking such precautions, you will avoid turning rescuers into patients.

### **Primary and Secondary Survey**

Once you have evaluated the scene, begin the standard primary survey. Pay particular attention to the airway and respiratory status. Following the primary survey, complete the secondary survey and develop a patient history. Relay information to the local poison information center or as your local policies indicate. Using the most current treatment, limit absorption and enhance elimination of the poison. If indicated, and qualified/certified, administer an antidote.

### **Ingested Poisons**

Poisoning by ingestion is the most common route encountered in prehospital care. Prompt and accurate assessment, followed by appropriate prehospital treatment, is essential.

### **General Principles of Assessment**

In cases of ingestion, it takes time for the poison to make its way from the gastrointestinal system into the circulatory system. Therefore, you need to find out not only what was ingested, but when it was ingested and how much. A thorough patient history is essential to patient management.

## Taking the History

Begin your history by trying to find out the type of poison ingested, route of intoxication, the quantity of the poison, the time elapsed since ingestion, and whether the patient took any alcohol or other potentiating substance. Ask the victim about drug habituation or abuse. Inquire about underlying medical illnesses and allergies. In cases of poisoning, remember that inaccuracies creep into nearly half the histories because of drug-induced confusion, patient misinformation, or deliberate patient attempts at deception. Therefore, suspect information given by patients in cases of drug overdoses.

Before leaving a suicidal patient who claims to have been “just kidding,” consider the legal ramifications. You may be charged later with patient abandonment. At the same time, be aware of protective custody laws in your state. If in doubt on whether or not to place the patient in protective custody, contact your local law enforcement agency.

The following questions will help you to develop a relevant history.

- What did you ingest? (Obtain pill containers and any remaining contents, samples of the ingested substance, or samples of vomitus. Bring with patient to ED.)
- When did you ingest the substance? (Time affects your decision to use emesis, gastric lavage, activated charcoal, or an antidote.)
- How much did you take (or eat)?
- Did you drink any alcohol?
- Have you attempted to induce vomiting?
- Have you done anything to treat yourself?
- Have you been under psychiatric care? If so, why? (Answers may indicate a potential for suicide.)
- What do you weigh?
- Prescribed medications and compliance.

**Physical Examination** Since the history can be unreliable, the physical examination assumes an important role. The physical examination of the poisoned patient has two purposes:

(1) to provide physical evidence of intoxication

(2) to find any underlying illnesses that may account for the patient's symptoms or that may affect the outcome of the poisoning. As you complete the primary and secondary assessments, pay attention to the following patient features.

**Skin:** Is there evidence of cyanosis, pallor, wasting, or needle marks? Flushing of the skin may indicate poisoning with an anticholinergic substance. Staining of the skin may occur from chronic exposure to mercuric chloride, bromine, or similar chemicals.

**Eyes:** Pupillary constriction (with Opioids or cholenergics) or dilation (with Anticholinergics and Sympathomimetics) occurs with various types of poisons (marijuana, methamphetamines, narcotics). Inquire about impaired vision, blurring of vision, or coloration of vision.

**Mouth:** Inspect the mouth for signs of caustic ingestion, presence of gag reflex, amount of salivation, any breath odor, or presence of vomitus.

**Breath:** Breath sounds may reveal evidence of aspiration, atelectasis, or excessive

pulmonary secretions.

**Circulation:** Cardiac examination may give clues about the type of poison ingested. For example, the presence of tachydysrhythmias (methamphetamine) or bradydysrhythmias (organophosphates) may suggest specific toxins such as those shown in parentheses

**Abdomen:** Abdominal pain may result from poisoning by salicylates, methyl alcohol, lead or caustics.

It is not unusual for a rescuer to be confronted with a patient that has ingested a combination of drugs, either in a suicide attempt or in an effort to get "high." These multiple ingestions present a therapeutic dilemma. Each chemical not only acts by itself, it may also act synergically with the others. A common example of this is the "speedball" (heroin mixed with cocaine). If the narcotic overdose is treated, the rescuer is often presented with a patient that is now in a cocaine-induced catecholamine crisis (tachycardia, hypertension, seizures).

It is important to learn the regional names for street drugs. But this is no guarantee of accuracy. Street terminology changes from area to area. A "hit" of cocaine ("blow," "coke," "snort," or a dozen other names) may mean whatever amount the body can handle—from 2-200 mg.

### ***General Principles of Management***

As mentioned previously, priorities in the treatment of the poisoned patient are maintenance of airway, breathing, and circulation. Prevention of aspiration is one of the major objectives of prehospital care. Aspiration pneumonia is a serious concern in the definitive care of the toxic patient.

Assess the adequacy of the patient's airway by observing ventilatory effort, estimating the force and volume of expiration, and detecting any sounds of obstructed breathing. In cases that require more definitive airway management, endotracheal intubation (orally or nasally) may be necessary. Generally, nasal intubation is preferred for patients who have a gag reflex. This may be checked by brushing the eyelashes. If there is a blink or lash reflex there is probably an intact gag reflex. High-flow oxygen supplementation should be routinely performed.

### ***Inhaled Poisons***

Toxic inhalations can result from a poisonous environment or from self-induced inhalations. The following material provides guidelines for assessing and managing common toxic inhalations.

### ***Presentation of Toxic Inhalations***

The principal effects of inhaled toxic substances include respiratory signs and symptoms such as tachypnea, coughing, hoarseness, stridor, dyspnea, chest pain, chest tightness, chest retraction, and wheezing, rales, or rhonchi. A particularly dangerous form of inhalation results in patients who seek "highs" through the use of the chemical

propellants found in many aerosol products. They spray these chemicals into a paper or plastic bag and inhale them. This produces a relative hypoxia causing the patient to lose consciousness as the chemicals displace oxygen in the lungs. Signs and symptoms of aerosol inhalation include:

- CNS:
  - Dizziness,
  - headache,
  - confusion, seizures
  - hallucinations,
  - coma
- Respiratory:
  - Tachypnea,
  - coughing,
  - hoarseness, stridor,
  - dyspnea,
  - retractions,
  - chest pain,
  - chest tightness,
  - wheezing, rales, or rhonchi
- Cardiac:
  - Dysrhythmias

### ***General Management of Toxic Inhalations***

The first priority in treating patients who have inhaled toxic gases or powders is safe removal from the source; the following general principles of management apply to most patients exposed to toxic inhalations.

Safely remove the patient from the poisonous environment, in doing so, take the following essential precautions.

- Wear protective clothing prior to entry.
- Use appropriate respiratory apparatus.
- Remove patient's contaminated clothing.

Perform the necessary primary and secondary assessments.

Initiate the following treatment procedures.

- Establish and maintain airway.
- Administer high-flow oxygen.
- Intubate and assist ventilations, if appropriate.
- Administer CPR, if necessary.
- Use a bag-valve-mask or demand valve.
- Establish venous access.
- Contact poison control.

## ***Specific Inhaled Toxins***

Aside from aerosol inhalations, most emergencies that involve inhaled poisons can create a potentially toxic environment for the rescuer. Whenever you come across the following commonly inhaled poisons, survey the scene for hazards to your own safety.

### **Carbon Monoxide**

Carbon Monoxide (CO) is an odorless, tasteless gas that is often the byproduct of incomplete combustion. Because of its chemical structure, it has more than 209 times the affinity of oxygen to bind with the red blood cell's hemoglobin (producing carboxyhemoglobin). Once this molecule has bound with hemoglobin, it is very resistant to removal and causes an effective hypoxia. Because of the variability of the signs and symptoms, people ignore CO poisoning until very toxic levels occur. Common settings for CO poisoning include improperly vented heating systems or the use of a small barbecue to heat a house or camper. **Note:** A pulse oximeter reads the level of CO attached to hemoglobin as if it were O<sub>2</sub>, thereby giving a false high reading in cases of CO poisoning.

Signs and symptoms of CO poisoning include:

- Headache
- Nausea, vomiting
- Confusion or other altered mental status
- Tachypnea

### **Treatment**

Because of the difficulty in removing CO from hemoglobin definitive treatment is often performed in a hyperbaric chamber. In this specially designed environment, oxygen under several atmospheres of pressure surrounds the body. In field settings, take the following supportive steps:

- Safely remove the patient from the contaminated area.
- Begin immediate ventilation of the area.
- Initiate the following treatment procedures:
  - Establish and maintain the airway.
  - Administer high-flow oxygen.
  - Intubate and assist ventilations, if Appropriate.
  - Administer CPR, if necessary.
  - Use a bag-valve-mask or demand valve.
  - Establish venous access.

## **Surface Absorbed Poisons**

Many poisons can be absorbed through the skin. These include the organophosphates, cyanide, and other toxins.

### ***General Principles of Management***

Whenever you suspect absorption of a poison (especially such agents as cyanide or strychnine) take the following steps:

- Safely remove the patient from the poisonous environment. Be sure to adhere to the below procedures:
  - Wear protective clothing
  - Use appropriate respiratory apparatus.
  - Remove patient's contaminated clothing.
  - Perform primary and secondary assessment.
- Initiate the following treatment procedures.
  - Establish and maintain the airway.
  - Administer oxygen.
  - Intubate and assist ventilations, if appropriate.
  - Administer CPR, if necessary.
  - Use a bag-valve-mask or demand valve.
  - Establish venous access
  - Contact poison control.

### **Organophosphates**

Organophosphate compounds are used as insecticides in residential as well as commercial agriculture. Poisoning by the organophosphates is common in agricultural areas where crop dusters are used. Exposure has also been employed as a suicide method. Because of their chemical structure organophosphates are capable of producing a variety of signs and symptoms affecting both the parasympathetic (muscarinic effects) and the sympathetic (nicotinic effects) nervous systems.

### **Presentation**

Organophosphates inactivate the enzyme cholinesterase at the synaptic junction, causing elevated levels of the neurotransmitter acetylcholine. This produces excessive salivation, lacrimation, and gastrointestinal problems, including abdominal pain, nausea, vomiting and incontinence (urine and diarrhea). This cholinergic pattern may be remembered with the mnemonic SLUDGE (excessive Salivation, Lacrimation, Urination, Diarrhea, Gastrointestinal distress, Emesis). The patient may have constricted pupils (miosis). While bradycardia is often associated with organophosphate poisoning, stimulation of nicotinic receptors will produce a tachycardia instead. The prehospital management of organophosphate poisoning is aimed at reversal of the deleterious excessive cholinergic effects (cholinergic crisis).

Decontamination of a patient with organophosphate poisoning is essential. All clothing should be removed. Repeated washing with copious amounts of water and soap are recommended. All clothing, including all leather items, should be discarded according to Environmental Canada guidelines for hazardous materials.

### ***Treatment***

In suspected cases of exposure to organophosphates, take the following steps.

- DO NOT Induce Vomiting; gastric lavage is the preferred method of decontamination.
- Establish and maintain airway. This may require vigorous suctioning due to the excessive secretions caused by the organophosphate.
- Administer oxygen at a high concentration.
- Intubate and assist ventilations, if appropriate.
- Administer CPR if necessary.
- Use a bag-valve-mask or demand valve.
- Establish venous access.
- Atropine (as per local protocols indicate)
- Seizures may be managed with diazepam (Adult 5-10 mg initially, with repeats per medical protocol; Child 0.25-0.4 mg/kg).
- Contact poison control.

## **Drug Overdose and Substance Abuse**

In general terms, drug overdose refers to poisoning from a pharmacological substance, either legal or illegal. This can occur by accident, miscalculation, changes in the strength of a drug, suicide, poly drug use, or recreational drug usage. Many overdose emergencies seen in the field occur in the habitual drug abuser. It is difficult to obtain a good history in these cases. However if you know regional street-drug slang, a more accurate history may be obtained. It is imperative that you maintain a nonjudgmental attitude in these cases. Addiction is both a physiological and a psychological problem.

The presentation of the drug overdose will vary based on the substance used. Management should be the same as for any ingested, inhaled, or injected poison. Poison control should be contacted for additional direction.

As a final word of warning, always survey the scene -in cases of drug overdose. Be alert for possible rescuer danger. Do not go running into a known "crack house" or "shooting gallery" without backup.

### **Alcohol Abuse**

Alcohol (ethyl alcohol or ethanol) depresses the central nervous system, potentially to the point of stupor, coma, and death. In patients with severe liver disease, metabolism of alcohol may become impaired, which increases the course and severity of intoxication. At low doses, alcohol has excitatory and stimulating effects, thus depressing inhibitions. At higher doses, alcohol's depressive effect is more obvious. Abuse of and dependence upon alcohol is called alcoholism. It is a major problem in our society and contributes to many accidents, including automobile collisions, drowning, burns, trauma, and drug overdoses.

Alcohol is completely absorbed from the stomach and intestinal tract in approximately 30-120 minutes after ingestion. Once absorbed, alcohol is distributed to all body tissues and fluids. Concentrations of alcohol in the brain rapidly approach the level of alcohol in the blood.

### **Physiological Effects**

Alcohol should always be considered a CNS depressant, despite the initial increased activity as seen with lower doses. In addition, alcohol causes a peripheral vasodilator effect on the cardiovascular system, resulting in flushing and a feeling of warmth. Therefore, in extreme cold conditions, alcohol may cause the blood vessels to dilate, resulting in an increased loss of body heat. The diuretic effect seen when large amounts of alcohol are ingested results from inhibition of vasopressin (antidiuretic hormone). In the absence of vasopressin, urine production increases. The "dry mouth" syndrome experienced after alcohol consumption is thought to be the result of alcohol-induced cellular dehydration.

## **General Alcoholic Profile**

Although the symptoms of alcoholism vary from periodic "binge drinker" to daily drinker, the following are warning signs of the disease:

- Drinks early in the day
- Prone to drink alone and secretly
- Periodic binges (may last for several days)
- Partial or total loss of memory (blackouts) during period of drinking
- Unexplained history of gastrointestinal problems (especially bleeding)
- "Green Tongue Syndrome" (using chlorophyll-containing substances to disguise the odor of alcohol on the breath)
- Cigarette burns on clothing
- Chronically flushed face and palms
- Tremulousness
- Odor of alcohol on breath under inappropriate condition.,

## **Consequences of Chronic Alcoholism**

Alcohol abuse adversely affects the body in many ways. Some of the most common effects include:

- Poor nutrition
- Alcohol hepatitis
- Liver cirrhosis with subsequent esophageal varices
- Loss of sensation in hands and feet
- Loss of cerebellar function (Balance and coordination)
- Pancreatitis
- Upper gastrointestinal hemorrhage (often fatal)
- Hypoglycemia
- Subdural hematoma (due to falls)
- Rib and extremity fractures (due to falls)

The odor of an alcoholic beverage on the patient's breath should be considered a poor indicator of alcohol intoxication. Insulin shock, subdural hematomas, and other life-threatening disease processes may mimic the signs and symptoms of alcohol intoxication.

## **Withdrawal Syndrome**

The alcoholic may suffer a withdrawal reaction from either abrupt discontinuation of ingestion after prolonged use or from a rapid fall in the blood alcohol level after acute intoxication. Withdrawal symptoms can occur several hours after sudden abstinence and can last up to 5-7 days. Seizures (alcohol withdrawal epilepsy sometimes called "rum fits" may occur within the first 24-36 hours of abstinence. Signs and symptoms of a withdrawal syndrome include:

- Coarse tremor of hands, tongue, and eyelids

- Nausea and vomiting
- General weakness
- Increased sympathetic tone
- Tachycardia
- Sweating
- Hypertension
- Orthostatic hypotension
- Anxiety
- Irritability or a depressed mood
- Hallucinations
- Poor sleep

Delirium tremens (DTs) may occur on the second or third day of the withdrawal. Delirium tremens is characterized by a decreased level of consciousness as the patient hallucinates and misinterprets nearby events. Seizures and delirium tremens are ominous signs. There is a significant mortality from delirium tremens. Medical control may order diazepam (Valium) in severe cases.

### **In-Field Treatment of Alcohol Abuse**

Some alcoholics will drink methanol (wood alcohol) or ethylene glycol (a component of antifreeze) if ethanol is unavailable. Ingestion of these chemicals can cause blindness or death. Alcohol intoxication, whether acute or chronic, should not be underestimated as a toxic emergency problem. In cases of suspected alcohol abuse, take the following steps.

- Maintain airway.
- Determine if other drugs are involved.
- Start IV using D5W, LR, or 0.9 percent NS. (as local protocols indicate)
- Chemstrip and administer 25 mg of D50W if hypoglycemic.
- Maintain an empathetic attitude, and reassure the patient of help.

## **Allergies and Anaphylaxis**

An allergic reaction is an exaggerated immune response to any substance, that is introduced by a variety of means. It is a reaction by the body's immune system which has released chemicals to fight the stimulus. It may be mild and local, with hives or itching, or it can be systemic and severe causing shock and respiratory failure.

Anaphylaxis is an acute, generalized, and violent antigen-antibody reaction - the most severe form of an allergic reaction – that may be rapidly fatal even with prompt and appropriate emergency medical care. Anaphylaxis develops in seconds to minutes after the ingestion, injection, inhalation, or absorption of an antigenic substance. Anaphylaxis is an emergency in which appropriate prehospital intervention by EMS personnel is life-saving. The two most common signs of this are wheezing (a high-pitched, whistling breath sound usually because of a blockage of the airway and heard on expiration) and urticaria. Urticaria is when there are areas of generalized itching or burning that have small, multiple raised areas on the skin.

## **The Immune System**

The immune system plays a central role in anaphylaxis and allergies. The immune system is a complicated system responsible for combating infection. Components of the immune system can be found in the blood, the bone marrow, the connective tissues, and in the lymphatic system. The immune response is a complex cascade of events. The goal of the immune response is the destruction or inactivation of pathogens, abnormal cells or foreign molecules such as toxins. The body can accomplish this through two mechanisms: cellular immunity and humoral immunity. Cellular immunity is the T Lymphocyte production in the thymus gland which is responsible for fighting infections of biological agents living in the body (viruses and fungal infections). Humoral immunity derives from B lymphocytes resulting in the formation of special proteins called antibodies. This begins with the exposure of the body to an antigen, which is a protein capable of inducing an immune response. If the body has never been exposed to a particular antigen, the response of the immune system is different than if it has been previously exposed to the particular antigen. The initial response to an antigen is called the primary response. Following exposure to a new antigen, several days are required before both the cellular and humoral components of the immune system respond. Generalized antibodies (IgG and IgM) are initially released to help fight the antigen. At the same time, other components of the immune system begin to develop antibodies specific for the antigen and a memory of the particular antigen. If the body is exposed to the same antigen again, the immune system responds much faster. This is called the secondary response. Antibodies specific for the offending antigen are released as part of the secondary response. These antigen-specific antibodies are much more effective in facilitating removal of the offending antigen than the generalized antibodies released during the primary response.

## Allergies

The initial exposure of an individual to an antigen is referred to as sensitization. Sensitization results in an immune response. Subsequent exposure induces a much stronger secondary response. Generally, a person is not made uncomfortable by and does not even notice, this kind of normal immune response.

Some individuals can become hypersensitive (overly sensitive) to a particular antigen. Hypersensitivity is an unexpected and exaggerated reaction to a particular antigen, which usually results in some kind of discomfort for the individual. Hypersensitivity is often used synonymously with the term allergy.

Signs and Symptoms of an Allergic Reaction and Anaphylaxis	
<p><b>Respiratory System</b></p> <ul style="list-style-type: none"><li>➤ Sneezing or an itchy, runny nose (initially)</li><li>➤ Tightness in the chest or throat</li><li>➤ Irritating, persistent dry cough</li><li>➤ Hoarseness</li><li>➤ Respirations that become rapid, labored, or noisy</li><li>➤ Wheezing and / or stridor</li></ul> <p><b>Circulatory System</b></p> <ul style="list-style-type: none"><li>➤ Decrease in blood pressure as the blood vessels dilate</li><li>➤ Increase in pulse rate (initially)</li><li>➤ Pale skin and dizziness, as the vascular system fails</li><li>➤ Loss of consciousness and coma</li><li>➤ ECG Sinus Tachycardia with ectopic beats</li></ul>	<p><b>Skin</b></p> <ul style="list-style-type: none"><li>➤ Flushing, itching, or burning skin; especially common over the face and upper chest</li><li>➤ Urticaria over large areas of the body, both internally and externally</li><li>➤ Swelling, especially of the face, neck, hands, feet, and/or tongue</li><li>➤ Swelling, cyanosis or pallor of the lips</li><li>➤ Warm, tingling feeling in the face, mouth, chest, feet, and hands</li></ul> <p>➤</p> <p><b>Other findings:</b></p> <ul style="list-style-type: none"><li>➤ Anxiety, a sense of impending doom</li><li>➤ Abdominal cramps</li><li>➤ Headache</li><li>➤ Itchy, watery eyes</li><li>➤ Decreasing mental status</li></ul>

Table 1.1 Common Signs and Symptoms of Allergic Reactions/Anaphylaxis  
Adopted and Modified from: *Emergency: Care and Transportation of the Sick and Injured*, 7<sup>th</sup> Ed. 1999.

There are two types of hypersensitivity reactions: delayed and immediate.

### Delayed Hypersensitivity

Delayed hypersensitivity is a result of cellular immunity and does not involve antibodies. Delayed hypersensitivity usually occurs in hours and days following exposure and is the sort of allergy that occurs in normal people. Delayed hypersensitivity most commonly results in a skin rash and is often due to exposure to certain drugs and chemicals. The rash associated with poison ivy is an example of delayed hypersensitivity.

## **Immediate Hypersensitivity**

Examples of immediate hypersensitivity reactions are hay fever, drug allergies, food allergies, eczema, and asthma. An antigen that causes release of the IgE antibodies is referred to as an allergen. Following exposure to a particular allergen, large quantities of IgE are released. This becomes attached to the membranes of basophiles and mast cells. These are specialized cells of the immune system which contain chemicals that assist in the immune response. These cells release histamine, heparin, and other substances into the surrounding tissues when the allergen binds to them.

The principal chemical mediator of an allergic reaction is histamine.

Histamine is a potent substance which causes bronchospasm, increased intestinal motility, vasodilation, and increased vascular permeability. Increased vascular permeability causes the leakage of fluid from the circulatory system into the surrounding tissues. Histamine acts on specialized histamine receptors present throughout the body. There are two classes of histamine receptors. H1 receptors, when stimulated, cause bronchoconstriction and contraction of the intestines. H2 receptors cause peripheral vasodilation and secretion of gastric acids. The goal of histamine release is to minimize the body's exposure to the antigen. Bronchoconstriction decreases the possibility of the antigen entering through the respiratory tract. Increased intestinal motility serves to move the antigen quickly through the gastrointestinal system with minimal absorption of the antigen into the body. Vasodilation and capillary permeability help remove the allergen from the circulation where it has the potential to do the most harm.

The most severe type of allergic reaction is termed anaphylaxis. This usually occurs when a specific allergen is injected directly into the circulatory system. When the allergen enters the circulation, it is distributed widely throughout the body. The allergen interacts with both basophiles and mast cells, resulting in the massive dumping of histamine and other substances associated with anaphylaxis. The principal body systems affected by anaphylaxis are the skin, the respiratory system, the cardiovascular system, the gastrointestinal system, and the nervous system. Histamine causes widespread peripheral vasodilation, as well as increased permeability of the capillaries. Increased capillary permeability results in "leaking" and a marked loss of plasma from the circulation. People sustaining anaphylaxis can actually die from circulatory shock. Another substance called slow-reacting substance of anaphylaxis (SRA) is also released from the basophiles and mast cells. This causes spasm of the bronchiole smooth-muscle causing an asthma-like attack and occasionally asphyxia. SRA potentiates the effects of histamine, especially on the respiratory system.

The signs and symptoms of anaphylaxis begin within 30 - 60 seconds following exposure to the offending allergen. In a small percentage of patients, the onset of signs and symptoms may be delayed over an hour. The severity of the reaction is often related to the speed of onset. Reactions that develop very quickly tend to be much more severe.

The signs and symptoms of anaphylaxis can vary significantly. Patients suffering an anaphylactic reaction often have a sense of impending doom, this is followed by flushing, generalized itching (pruritis) and an increase in heart rate. Often there is the immediate development of hives (urticaria). Itching and hives are the most common manifestations.

Virtually all body systems may be affected in anaphylaxis or other allergic reactions.

## **ASSESSMENT**

As with any emergency, the initial step in the management of suspected anaphylaxis is to complete the primary assessment, particularly evaluation of the patient's airway, breathing, and circulation to detect and correct any immediate threats to the patient's life. The primary assessment should be followed by a more detailed secondary assessment.

### **Primary Assessment**

#### **Respiratory System**

The first step in the primary assessment is to evaluate the airway. Laryngeal edema is a frequent complication of anaphylaxis and can threaten the airway. Initially, laryngeal edema will cause a hoarse voice. As the edema worsens, the patient may develop stridor. Finally, complete airway obstruction may develop, resulting from either massive laryngeal edema or laryngospasm or both.

Any problems with the airway must be immediately corrected before moving to the next step of the primary assessment. If an airway problem is detected, first apply basic airway maneuvers such as head positioning or the modified jaw thrust maneuver. Use oropharyngeal and nasopharyngeal airways with caution as they can cause laryngospasm. If the patient is having severe airway problems, consider early endotracheal intubation to prevent complete occlusion of the airway. It is important to remember that the glottic opening may be smaller than expected due to laryngeal edema. Because of this, a smaller-than-average endotracheal tube may be needed. Also, it is important to remember that the larynx will be very irritable and any manipulation of the airway may lead to laryngospasm. Ideally, the most experienced member of the crew should perform endotracheal intubation, as one attempt may be all that will be possible. Equipment for placement of a surgical airway, such as a needle cricothyrotomy, should be available in case it is needed.

After assessing and managing the airway, evaluate breathing. As detailed earlier in the chapter, anaphylaxis adversely affects the respiratory system. Bronchospasm is a common finding in anaphylaxis. First, evaluate the rate and depth of breathing. Often the patient will have tachypnea, but will have shallow respirations. Quickly auscultate both sides of the chest. Wheezing indicates bronchospasm. Beware of the silent chest as this can indicate severe bronchospasm with minimal air movement. The first step is to administer high-flow oxygen with a nonrebreather mask. If respirations are shallow, consider assisting them with positive-pressure ventilation. Patients with significant dyspnea may require endotracheal intubation and mechanical ventilation.

Next evaluate circulation. Anaphylaxis causes peripheral vasodilation and increased capillary permeability. Both can cause hypotension. The pulse rate may be either fast or slow depending on the stage of the illness. Be prepared to provide CPR if the pulse is absent.

During the primary assessment, be sure to obtain a baseline assessment of the patient's mental status, using the AVPU scale ( Alert, Verbal, Painful, Unresponsive response to

stimuli). One of the earliest signs and symptoms of shock is a change in the patient's mental status. The patient's mental status may deteriorate as the condition worsens. Thus it is important to monitor the patient's mental status over time using objective criteria. Always consider the possibility of trauma in anaphylaxis. If there is any suspicion of coincidental trauma, stabilize the cervical spine. It is not uncommon for people to injure themselves as they try to escape from wasps and bees. They may fall or otherwise cause an injury. The signs and symptoms of trauma may be masked by the signs and symptoms of anaphylaxis. Always err on the side of safety and immobilize if there is any possibility of cervical spine injury.

### **Secondary Assessment**

When performing the secondary assessment on a patient with a severe allergic reaction or anaphylaxis, concentrate on the skin, the respiratory system, the cardiovascular system, and the gastrointestinal system - the body areas most affected by the mediators of anaphylaxis.

Begin at the head and pay particular attention to the airway. A hoarse voice, stridor, or a croupy cough can indicate laryngeal edema or spasm. Quickly evaluate the chest by inspection, palpation, and auscultation. Listen closely for wheezing and reduced airflow. Next turn your attention to the gastrointestinal system. Note any nausea, vomiting, or diarrhea, which are all associated with histamine release. Check peripheral pulses. Also note the presence of edema. Many patients will have hives - raised red blotches across the body, primarily due to histamine release which causes plasma leakage resulting in the classic wheals.

The vital signs are an important part of the secondary assessment. Accurately determine the pulse rate, respiratory rate, and blood pressure. The vital signs should be repeated frequently. Use pulse oximetry (if available) and continuous ECG monitoring.

### **MANAGEMENT**

Complete the primary and secondary assessment as soon as possible. Apply airway management techniques as needed and administer oxygen via a non rebreathe mask. Place the patient in a supine position with legs slightly elevated and apply a blanket or other covering to maintain body temperature.

### **Fluid and Pharmacological Therapy**

Intravenous fluid replacement is critical in preventing hypovolemia and hypotension. As soon as possible, establish an IV with a crystalloid solution of normal saline and run it TKO. If the patient is hypotensive (BP < 90 systolic) fluids should be administered wide open-reassess after 500cc, maximum volume of 2 litres. If time allows, place a second IV line TKO.

The principal treatment of anaphylaxis is pharmacological. Emergency medications used in the treatment of anaphylaxis include epinephrine, antihistamines, and corticosteroids. Occasionally, inhaled beta agonists, such as albuterol, may be required and administered by hospital staff.

## **Pharmacological agents:**

### **Epinephrine**

Generic Name:

Epinephrine, Adrenaline

Trade Names:

Adrenaline Chloride, Epifrin, Epinephrine ECK, EpiPen, Vaponefrin

Class:

Bronchodilator/Sympathomimetic; antiglaucoma agent

Available:

Inhalation solutions, injection (1:1,000 and 1:10,000), eye drops

Action:

Bronchodilation, peripheral vasoconstrictor, stops leakage from blood vessels, effects occur within seconds of administration and can last up to 20 minutes

Indication: History of exposure to a known precipitant (allergic reaction), anaphylaxis, (not local protocol -bronchial asthma, exacerbation of COPD cardiac arrest)

Contraindication:

None per Manitoba Health Protocol (Used cautiously in patients with serious underlying cardiovascular disease, hypertension, pregnancy)

Adverse Reactions:

Elevated heart rate, palpitations, pale skin, chest pain, arrhythmias, nausea/vomiting, anxiousness, dizziness/headache

Drug Interactions:

Beta blockers antagonize the therapeutic effect of epinephrine, digitalis potentiates the proarrhythmic effects, tricyclic antidepressants and MAO inhibitors potentiate the cardiovascular effects, phenothiazines cause a paradoxical decrease in BP.

Dose:

Epinephrine 1:1000 (1 mg epinephrine in 1 ml solvent), mild-moderate allergic reaction, Bronchial asthma, Exacerbation of COPD, 0.3 mg SC/IM, 6yrs-adult, 0.15 mg SC/IM child (1-5 years) Repeat for either patient once in 15 minutes if required.

Duration of action:

Onset 1-4 minutes, duration 1-4 hours for injection.

Special Considerations:

Epinephrine should be protected from the light, Blood pressure, pulse, and ECG must be

constantly monitored

#### Administering Epinephrine:

Assessment – Clinical Features of Anaphylaxis must include one of the following: swelling of the face, mouth or throat, shortness of breath or respiratory distress, complete or partial upper airway obstruction, hypotension or shock (anaphylactic shock). Other symptoms your patient may be exhibiting could include hives or itching, coughing or wheezing, abdominal pain, nausea and vomiting, anxiety, stupor or coma.

Administer 0.15 mg of epinephrine 1:1000 solution for children ages 1-5 SC/IM, administer 0.3 mg of epinephrine 1:1000 solution for people age 6 and over SC/IM.

#### Documentation:

Date and time of administration. Indication for initiating protocol ie. Signs & symptoms.

Dosage, route and site of administration. Signature.

Disposal of equipment: Dispose of vial and syringes into appropriate sharps container.

## **DIPHENHYDRAMINE (BENADRYL)**

#### Class:

- antihistamine, anticholinergic,

#### Mechanism of Action

- blocks cellular histamine receptors
- decreases vasodilation
- decreases motion sickness
- reverses extrapyramidal reactions

#### Indications

- symptomatic relief of allergies, allergic reactions, anaphylaxis, acute dystonic reactions due to phenothiazines, motion sickness

#### Contraindications

- glaucoma, hypertension, narrow angle glaucoma, infants
- patients taking monoamine oxidase inhibitors

#### Adverse Reactions

- sedation, hypotension, seizures, visual disturbances, vomiting, urinary retention, palpitations, dysrhythmias, dry mouth and throat
- paradoxical CNS excitation in children

#### Drug Interactions

- potentiates effects of alcohol and other anticholinergics
- MAOIs prolong anticholinergic effects of diphenhydramine

#### How Supplied

- tablet: 25, 50 mg
- capsules: 25, 50 mg
- 50 or 100 mg prefilled syringes, vials (IV or IM)
- elixir 12.5 mg / 5 ml

#### Dosage and Administration

- adult: 25-50 mg IM or IV or PO
- pediatric: 1-2 mg/kg IV, IO slowly or IM
- if given PO: 5 mg / kg / 24 hours

#### Duration of Action

- onset: 15-30 minutes
- peak effect: 1 hour
- duration: 3-12 hours

#### Special Considerations

- if used in anaphylaxis, often used in conjunction with epinephrine and steroids

## Atropine Sulfate

### Class:

Anticholinergic agent

### Mechanism of Action:

Parasympatholytic: inhibits action of acetylcholine at postganglionic parasympathetic Neuroeffector sites

Increases heart rate in life-threatening bradydysrhythmias

### Indications:

Hemodynamically significant bradycardia

Asystole

Organophosphate poisoning

Bronchospastic pulmonary disorders

### Contraindications:

Tachycardia

Hypersensitivity

Unstable cardiovascular status in acute hemorrhage and myocardial ischemia

Narrow-angle glaucoma

### Adverse Reactions:

Headache, dizziness, palpitations, nausea and vomiting

Tachycardia, dysrhythmias, anticholinergic effects (blurred vision, dry mouth, and urinary retention)

Paradoxical bradycardia when pushed slowly or at low doses  
flushed, hot dry skin

### Drug Interactions:

Potential adverse effects when administered with digoxin, cholinergics, physostigmine

Effects enhanced by antihistamines, procainamide, quinidine, antipsychotics, benzodiazepines and antidepressants

### How Supplied:

Prefilled syringes: 1.0 mg in 10 ml of solution

Ampules: 0.4, 0.6, or 1.0 mg in 1 ml of solution

### Dosage and Administration:

#### Adult:

Bradydysrhythmias: 0.5 – 1.0 mg IV every 3-5 minutes as needed to maximum total dose of 0.04 mg / kg

Asystole: 1.0 mg IV push every 3-5 minutes as needed to maximum total dose of 0.04 mg / kg

may be given endotracheally if IV not established

2.0 mg followed by 2.0 ml of normal saline

**Pediatric:**

Bradydysrhythmias: 0.02 mg/kg IV/ET/IO

Minimum single dose 0.1 mg, maximum single dose 1.0 mg

Asystole: 0.02 mg/kg IV/ET/IO

Minimum dose 0.1 mg; maximum dose 0.5 mg for a child and 1.0 mg for adolescent

If administered via ET, follow with 2.0 ml sterile normal saline

**Duration of Action:**

Onset: immediate

Peak effect: rapid to 1-2 minutes

Duration: 2-6 hours

**Special Considerations:**

Pregnancy safety: category C

Moderate doses dilate pupils

## **Naloxone**

Generic name:

Trade name:

Narcan

Class:

Narcotic antagonist

Mechanism of action:

Naloxone is chemically similar to the narcotics. Yet, it only has antagonistic properties, and competes for opiate receptors in the brain. It also displaces narcotic molecules from opiate receptors. It can reverse respiratory depression associated with narcotic overdose.

Indications:

For the complete or partial reversal of depression caused by narcotics including the following agents: Morphine, Codeine, Heroin, Hydromorphone, Oxycodone, Fentanyl

Contraindications:

Naloxone should not be administered to a patient with a history of hypersensitivity to the drug.

Adverse reactions:

These are rare. The following have been reported: hypotension, hypertension, ventricular arrhythmias, nausea and vomiting, tachycardia, diaphoresis, blurred vision, and withdrawal (opiate).

Drug interactions:

Incompatible with bisulphite and alkaline solutions.

Dosage and administration:

0.4 mg IV, IM or SC as an initial dose, repeat dose may be given once in 3-5 mins prn.

Maximum total dose: 2mg

Duration of action:

Onset: within 2 min. Duration: 30-60 mins.

Special Considerations:

Pregnancy safety: category B. Seizures have been reported (no casual relationship established). May not reverse hypotension. Caution should be exercised when administering naloxone to narcotic addicts (may precipitate withdrawal with hypertension, tachycardia, and violent behavior).

## **Flumazenil**

### Class:

Benzodiazepine receptor antagonist, antidote

### Mechanism of action:

Flumazenil antagonizes the actions of benzodiazepines in the central nervous system. It has been shown to reverse sedation, impairment of recall, and psychomotor impairment produced by benzodiazepines. Flumazenil is not, however, as effective in reversing hypoventilation. Flumazenil does not antagonize CNS effects of ethanol, barbiturates, or opioids.

### Indications:

Reversal of excessive or prolonged benzodiazepine sedation or overdose.

### Contraindications:

Hypersensitivity to flumazenil or to benzodiazepines, cyclic antidepressant overdose, and cocaine or other stimulant intoxication.

### Adverse Reactions:

Nausea and vomiting

Dizziness

Headache

Agitation

Injection site pain

Cutaneous vasodilation

Abnormal vision

Seizures

### Drug Interactions:

Toxic effects of mixed drug overdose (especially cyclic antidepressants) may emerge with the reversal of the benzodiazepine effects.

### Dosage and administration:

For suspected benzodiazepine overdose:

Adult: 0.2 mg (2ml) IV over 15 seconds; an additional dose of 0.3 mg (3ml) may be given in 30 seconds, followed by 0.5 mg (5ml) over 30 seconds at 1 minute intervals (maximum dose of 3 mg)

Pediatric: not recommended

### Duration of Action:

Onset: 1-2 min

Duration: Related to plasma concentration of benzodiazepine

### Special Considerations:

Pregnancy Safety: Category C

To minimize the likelihood of injection-site pain, administer through an intravenous infusion established in a large vein. Be prepared to manage seizures in patients who are physically dependent on benzodiazepines to control seizures, or who have ingested large doses of other drugs. Flumazenil may precipitate withdrawal syndromes in patients who are dependent on benzodiazepines. Patients should be monitored for possible re sedation, respiratory depression, or there residual benzodiazepine effects. Be prepared to establish and assist ventilation.

## **References**

Bledsoe, B. E., Porter, R. S. & Shade, B. R. (1997). Paramedic Emergency Care. Brady Prentice Hall, NJ., 3<sup>rd</sup> Edition.

Browner, B.D., Jacobs, L. M. & Pollak, A. N. (1999). Emergency: Care and Transportation of the Sick and Injured. Jones and Bartlett Publishers, 7<sup>th</sup> Edition.

Canadian Medical Association, (2002). Prescription & Over-the-Counter Drugs for Canadians. Readers Digest.

Krause, R., Anaphylaxis, medicine from Medline excerpt from 05/15/2000. Macdonald Ambulance Service advanced skills protocol and training package.

Mosby's Paramedic Textbook, Revised Second Edition, Mike Sanders, Mosby, Inc. 2001