

Diabetic Emergencies and Altered Mental States

**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. Elizabeth Sellers and Dr. Sora Ludwig**

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

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Disclaimer

These documents were developed for improved accessibility to standardized continuing education for all paramedics in Manitoba.

This training package is consistent with the National Occupational Competency Profiles and the core competency requirements (both mandatory and optional) as identified in “An Alternative Route to Maintenance of Licensure” (ARML). 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 supersede 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 can be used in conjunction with accepted models for education delivery and assessment as outlined in “An Alternative Route to Maintenance of Licensure”. Any individual paramedics wishing to use these continuing education packages to augment their ARML program should contact their local EMS Director.

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, it is suggested that the accompanying written test only be administered at the paramedic’s current level of practice.

This package has been reviewed by the Paramedic Association of Manitoba’s Educational Subcommittee and is subject to review by physician(s) or expert(s) in the field for 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 , attention Director of Education and Standards.

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INTRODUCTION:

This module is made up of two parts. Part 1 deals with the anatomy and physiology of the endocrine system, assessment and management of diabetic emergencies. Part 2 of this module deals with the nervous system, assessment and management of patients with altered mental states.

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 level and above.

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

PART 1, DIABETIC EMERGENCIES

REVIEW OF THE ENDOCRINE SYSTEM:

The endocrine system works with the nervous system to maintain the steady state of the body. The endocrine system helps regulate growth, metabolism, reproduction, use of nutrients by cells, salt and fluid balance, and metabolic rate.

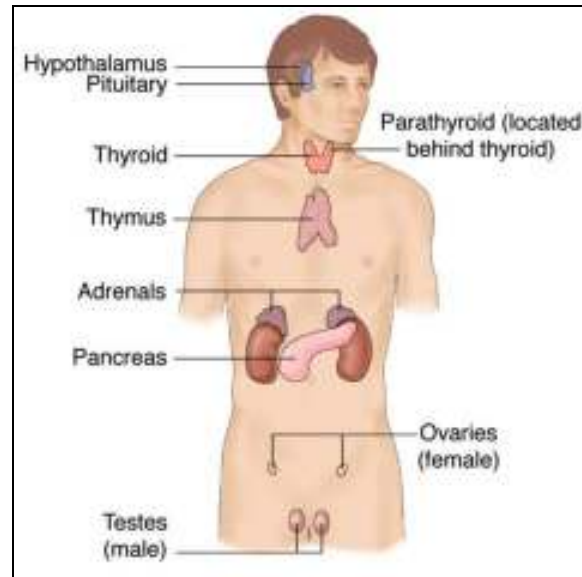
The endocrine system consists of tissues and glands that secrete hormones. Endocrine glands lack ducts; rather they release their hormones into the surrounding tissues. The hormones diffuse into capillaries and are transported by the blood. Hormones affect the activity of their target tissues, the tissues upon which they act.

Hormones can have widespread effects throughout the body. Responses to nervous system stimulation tend to be rapid and brief. In contrast, responses to hormones may require several hours or even longer, and effects may be long lasting.

ANATOMY OF THE ENDOCRINE SYSTEM:

- The main structures of the endocrine system include the following.
- Adrenal glands: two small glands that sit atop both kidneys, and have two distinct divisions, each with different functions. The *adrenal medulla*, closely related to the sympathetic component of the autonomic nervous system, secretes the catecholamine hormones *norepinephrine* and *epinephrine*. The *adrenal cortex* secretes three classes of hormones, all of them steroid hormones. They include *glucocorticoids*, the *mineralocorticoids*, and the *androgenic* hormones. These are hormones that influence the body's metabolism, blood chemicals, and body characteristics, as well as influence the part of the nervous system that is involved in the response and defence against stress.
- Hypothalamus: activates and controls the part of the nervous system that controls involuntary body functions, the hormonal system, and many body functions, such as regulating sleep and metabolism.
- Ovaries and testicles: secrete hormones that influence female and male characteristics, respectively.
- Pancreas: secretes a hormone (insulin) that controls the use of glucose by the body. The pancreas also plays a role in digestion.
- Pituitary gland: sometimes referred to as the “master gland,” its primary function is to control the other endocrine glands. It produces many hormones; their secretion is controlled by the hypothalamus.

- Thymus gland: plays a role in the body's immune system.
- Thyroid gland: produces hormones that regulate general body metabolism. These include *thyroxine* (T4), *triiodothyronine* (T3), and "C-cells", *calcitonin*.



ANATOMY OF THE PANCREAS:

The pancreas is a key gland located in the folds of the duodenum within the abdominal cavity. The pancreas is divided into three segments. The head of the pancreas lies over the vena cava, surrounding the common bile duct and usually attaches to the duodenum. The body extends horizontally across the abdomen and behind the stomach. The tail is narrow, extending to the spleen.

The pancreas is controlled in two ways. Sympathetic fibers provide pain sensation, vascular control, and enzyme secretion. The parasympathetic fibers control pancreatic exocrine and endocrine function.

Exocrine cells: called acini; aid in digestion of protein, carbohydrates, and fats by secreting pancreatic juice containing digestive enzymes.

Endocrine cells: located in the islets of Langerhans, make up 1% of pancreatic cells. These cells (located mainly in the tail), lack ducts and therefore, release secretions directly into the circulatory system.

PHYSIOLOGY OF THE ISLETS OF LANGERHANS:

There are between 500 thousand and 1 million pancreatic islets dispersed within the pancreas. Here the pancreas' endocrine functions occur. The islets of Langerhans include three types of cells: alpha cells, beta cells, and delta cells. Each secretes an important hormone.

- *Alpha* cells produce and secrete glucagon. The releasing stimuli of glucagon are low

blood glucose concentration, nutritional need, food ingestion, neural impulses (such as stress), somatostatin, increases in glycogenolysis, and other factors. The main effect of glucagon is increased blood glucose level.

- *Beta* cells produce and secrete insulin. The releasing stimuli of insulin are high blood glucose concentration, nutritional need, food ingestion, neural impulses (such as stress), somatostatin, and other factors. Insulin promotes glycogen synthesis, fat storage, protein synthesis (tissue building), and decreased glycogenolysis, and decreases blood glucose levels by increasing glucose transport into the cells.
- *Delta* cells produce and secrete somatostatin. The releasing stimuli of somatostatin are food ingestion, and neural impulses (such as stress). The effects of somatostatin are reduced insulin and/or glucagon secretion; it also impedes growth hormone release.

Diabetes Mellitus is the most common endocrine disorder, affecting many organs and leading to multiple complications. It is a complex disorder of carbohydrate, fat, and protein metabolism that is primarily a result of relative or complete lack of insulin secretion by the beta cells of the pancreas and/or the presence of insulin resistance in body tissues. Insulin is a small protein released by the pancreas when blood glucose levels rise. The primary functions of insulin are to increase glucose transport into cells, increase glucose metabolism, increase storage of glycogen in the liver, and to decrease blood glucose concentration to normal levels. Glucagon is a protein released by the pancreas when blood glucose levels fall. The two major effects of glucagon are to stimulate the conversion of glycogen to glucose in the liver; and to increase blood glucose levels by stimulating the liver to release glucose stores. Under normal conditions, the body maintains a range of glucose concentration that varies between 4.0 - 7.0 mmol/l. Diabetes mellitus is a systemic disease with many long term complications including blindness, kidney disease, nerve damage, circulation damage (atherosclerotic plaque builds up in small blood vessels), heart disease and stroke.

CLASSIFICATIONS OF DIABETES:

Type 1 Diabetes:

- previously referred to as IDDM (insulin dependant diabetes mellitus)
- characterized by inadequate production of effective insulin by the pancreas (most patients do not produce any insulin at all)
- has a heritable component
- affects 1 in every 10 diabetics and may occur anytime after birth - usually in teenagers and young adults
- requires lifelong treatment with insulin injections, exercise, and diet regulation

Type 2 Diabetes:

- previously referred to as NIDDM (non-insulin dependent diabetes mellitus)
- occurs most often in adults over 40 years of age and in those who are overweight
- characterized by a decreased production of insulin by the beta cells of the pancreas and to insulin resistance
- most patients require oral hypoglycemic medications, some may require insulin injections, exercise, and diet regulation to control their illness

Impaired Glucose Tolerance

- also referred to as IGT or pre-diabetes
- reduced entry of glucose into cells
- can only be determined after an oral glucose tolerance test

Gestational Diabetes

- occurs only during pregnancy
- is a risk factor for future Type 2 diabetes

PATHOPHYSIOLOGY OF HYPOGLYCEMIA:

Hypoglycemia (also known as insulin shock) is a complication of diabetes characterized by a less than normal amount of glucose in the blood. It is a syndrome related to blood glucose levels below 4.0 mmol/l. Hypoglycemia usually occurs in patients on insulin or some oral hypoglycemic medications, and often occurring quite suddenly. It usually is a result of strenuous exercise, an error in medication dosage, vomiting, or skipping a meal after taking insulin, and pregnancy and lactation. Hypoglycemia is a true medical emergency that has many different causes. Too much insulin or hypoglycemic medication, decreased dietary intake, unusual or vigorous physical activity, emotional stress, liver disease, adrenal or pituitary insufficiencies, and hypothyroidism are the most common causes. The lack of glucose quickly affects the nervous system because neurons cannot use fats or proteins as an energy source. The manifestations of hypoglycemia are directly related to the low blood glucose levels - not the high insulin levels. If hypoglycemia remains untreated, loss of consciousness, seizures, and death may follow.

A glucose deficit leads to a sequence of events:

Decreased blood glucose results in decreased glucose delivery to the brain; this results in neuroglycopenia. Cells break down fatty and amino acids into adenosine triphosphate (ATP) for energy, however brain cells cannot use ATP for energy. Autonomic nervous system stimulation results in the following:

Pancreas: sympathetic nerves & epinephrine rapidly stimulate glucagon secretion. Epinephrine inhibits insulin secretion.

Adrenal Glands: sympathetic nerves stimulate epinephrine secretion.

Adrenergic changes: hunger, weakness, diaphoresis, tachycardia, pallor, anxiety, tremors, nervousness, and rebound hyperglycemia.

Stomach: hypothalamus stimulates hunger; parasympathetic nerves increase gastric juices and stomach contractions.

Liver: sympathetic nerves directly stimulate glycogenolysis; epinephrine, glucagon, cortisol, and growth hormone increase glyconeogenesis; glucagon also stimulates glycogenolysis.

Muscle: hypothalamus stimulates pituitary to secrete growth hormone (delayed response), which along with epinephrine and cortisol, inhibits glucose utilization.

PATHOPHYSIOLOGY OF DIABETIC KETOACIDOSIS

Diabetic Ketoacidosis (DKA) is a serious complication of diabetes mellitus. It occurs when insulin levels become inadequate to meet the metabolic demands of the body. The hyperglycemia is from insulin insufficiency. DKA develops as blood glucose levels increase and individual cells become glucose depleted. The body begins to lose sugar to the urine, causing a significant osmotic diuresis and serious dehydration, evidenced by dry, warm skin and mucous membranes. As cellular glucose depletion continues, ketone and acid production occur, and the blood becomes acidotic. If the DKA is uncorrected, coma will follow. Unlike Hypoglycemia which usually has a rapid onset, diabetic ketoacidosis usually develops over a few days and may be initiated by anything which increases the demand for insulin in the blood (such as stress or infection or insulin omission for any reason). Ketoacidosis rarely occurs in Type 2 diabetes because some insulin is produced.

An insulin deficit leads to a sequence of events:

Initial stage

- Insulin deficit results in decreased transportation and use of glucose in many cells of the body.
- Blood glucose levels rise (hyperglycemia).
- Excess glucose spills into the urine (glucosuria) as the level of glucose in the filtrate exceeds the capacity of the renal tubular transport limits to reabsorb it.
- Glucose in the urine exerts osmotic pressure in the filtrate, resulting in a large volume of

urine to be excreted (polyuria) with loss of fluid and electrolytes (e.g., sodium) from the body tissues.

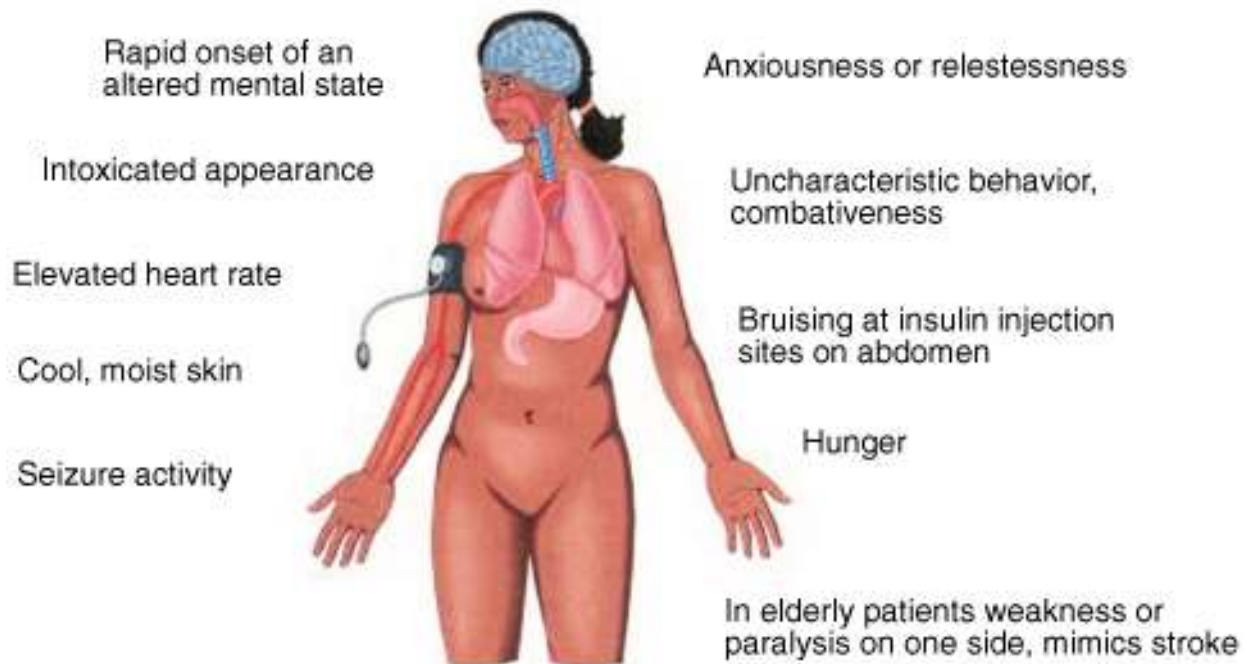
- Fluid loss through the urine and high blood glucose levels draw water from the cells, resulting in dehydration.
- Dehydration causes excessive thirst (polydipsia).
- Lack of nutrients entering the cells stimulates appetite (polyphagia).

If the insulin deficit is severe or prolonged, the process continues to develop, resulting in additional consequences. This occurs more frequently in persons with IDDM.

Progressive Effects

- Lack of glucose in cells results in catabolism of fats and proteins, leading to excessive amounts of fatty acids and their metabolites known as ketones or ketoacids in the blood. Ketones consist of acetone and two organic acids - beta-hydroxybutyric acid and acetoacetic acid. Because the liver and other cells are limited in the amount of lipids, fatty acids, or ketones they can process completely within a given time, excessive amounts of ketones in the blood cause **ketoacidosis**.
- The ketoacids bind with bicarbonate buffer in the blood, leading to decreased serum bicarbonate and eventually to a decrease in the pH of body fluids.
- Some ketoacids are excreted in the urine (ketonuria), but as dehydration develops, the glomerular filtration rate in the kidney is decreased, and excretion of acids becomes more limited, resulting in decompensated metabolic acidosis, which has life-threatening potential.

PATIENT WITH ALTERED STATUS AND HISTORY OF DIABETES COMMON SIGNS AND SYMPTOMS



Hyperglycemic Hyperosmolar Non-ketotic Coma (HHNK) is a diabetic coma in which the level of ketone bodies is normal. Hyperglycemia (above 40.0 mmol/l), hyperosmolarity and severe dehydration in the absence of ketoacidosis are common signs. The hyperglycemia produces a hyperosmolar state followed by an osmotic diuresis, dehydration, and electrolyte losses. Therefore, these patients typically have a greater hyperglycemia because they are more dehydrated and have 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. There are several precipitating factors of HHNK. They include:

- Type 2 diabetes
- Infection
- Myocardial infarction
- CVA
- Inability to obtain fluids e.g. immobility
- Pre-existing cardiac or renal disease
- Inadequate insulin requirements (stress, infection, trauma, burns)
- Medication use (glucocorticoids, sympathomimetics)
- Supplemental parenteral and enteral feedings
- Acute abdominal event e.g. acute pancreatitis, diverticulitis, G.I. bleed

PROMINENT NEUROLOGICAL FINDINGS IN HHNK:

FOCAL	DIFFUSE
Focal seizures	Seizures
Todd's paralysis	Lethargy
Hemiparesis	Confusion
Aphasia	Stupor
Babinski's reflex	Coma
Hyperreflexia	Delirium

SIGNS & SYMPTOMS OF DIABETIC EMERGENCIES

HYPOGLYCEMIA	DIABETIC KETOACIDOSIS
weakness & light-headedness	polyuria
headache	polydipsia
mental confusion	nausea & vomiting
fatigue, apathy	tachycardia
memory loss	Kussmaul respirations
incoordination; ataxic gait	warm, dry skin
slurred speech	weight loss
irritable, nervous, belligerent	restlessness to coma
seizures and coma	lethargy, confusion
weak, rapid pulse	fever, abdominal pain (mostly in kids)
cool, clammy skin	serum bicarbonate low
tremors	serum pH low
dilated pupils	appearance of intoxication
rapid onset	slow onset
pulse - normal to tachycardic	dehydration
respirations - normal to weak	hypotension
blood glucose < 4.0 mmol/l	seizures

PATIENT ASSESSMENT IN DIABETIC EMERGENCIES

When assessing diabetic patients, the paramedic must be aware of potential conditions that can cause rapid deterioration and death. These conditions are frequently identified by history, physical findings, and rapid blood glucose determination. The signs and symptoms of hypoglycemia are rapidly reversible. Its diagnosis and management are based on obtaining a history of diabetes dependent on insulin or medication and on observing altered mental status. Because of the easily reversible nature of this disease, management may precede a complete patient assessment.

The main distinction to make when encountering a diabetic emergency, is whether the patient is hypoglycemic or in diabetic ketoacidosis. To differentiate we must look at the presenting signs and symptoms, as well as history, which is the key component in diabetic assessments. The following is a list of important questions to ask a patient with a history of diabetes, in addition to the routine SAMPLE history questions.

1. Did the patient take their medications?
2. Did the patient eat (or skip) any regular meals?
3. Did the patient vomit after eating a meal?
4. Did the patient do any unusual exercise or physical activity?
5. Was the onset of symptoms gradual or rapid?
6. How long has the patient been experiencing these signs & symptoms?
7. Any other signs & symptoms associated with altered mental status?
8. Any period in which the patient regained normal mental status and then deteriorated again?
9. Did the patient suffer a seizure?
10. Has the patient experienced excessive thirst or urination?
11. Has the patient had a similar episode in the past? If so, what were the results?

ASSESSMENT FINDINGS

The physical examination begins with assessment of patient airway and respiratory status, mental status, skin colour, and temperature. Many diabetic patients wear Medic Alert tags that can provide diagnostic clues in patients who are unresponsive.

The patient's rate and rhythm of respirations should be assessed. A slight increase in respiratory rate is seen early in DKA. Kussmaul respirations (deep, rapid and intense respirations) may develop later. Hypoglycemic patients may have slowed or normal respiratory rates.

Tachycardia is commonly seen in hypoglycemic patients and those in DKA. Irregular or bradycardic rates may represent underlying cardiac disease or a serious dysrhythmia caused by electrolyte imbalance. Blood pressure may be decreased secondary to dehydration or underlying cardiac disease. Orthostatic vital signs can be less reliable in a diabetic because of diabetic neuropathy.

The patient's skin should also be assessed for tenting or loss of elasticity, both conditions suggesting dehydration. Cool, clammy skin with diaphoresis often accompanies hypoglycemia because of sympathetic nervous system activation. Hypothermia is a common presentation.

Facial features suggestive of dehydration include sunken eyes, a furrowed tongue, and dry mucous membranes.

Abdominal examination may show non-specific pain, tenderness, or distention which occur in DKA. Focal findings such as right lower quadrant tenderness may also be present. The paramedic should remember that infection is a common cause of DKA and should be considered. Neurological evaluation is important and should focus on determination of the patient's mental status. Seizures may occur which are secondary to hypoglycemia, and usually resolve with administration of glucose. Hypoglycemic patients may be psychotic, violent, or hyperactive, although DKA patients can appear apathetic.

MANAGEMENT OF THE DIABETIC PATIENT

Establishment of an adequate airway is always a first priority, and diabetes is no exception. Airway should be assessed while patient history is obtained. Any patient with respiratory distress requires airway stabilization and administration of high-flow oxygen via non-rebreather mask. Cardiac monitoring and pulse oximetry should be used if available and the appropriate training permits.

A rapid blood sugar determination is ideal for all known diabetic patients and all patients with altered mental status, preferably prior to glucose administration. Any diabetic who is awake and oriented with a blood sugar of less than 4.0 mmol/l should be given oral glucose.

If the patient is alert and refusing treatment or transport, sugar can be administered orally and then recommend complex carbohydrates. A quick boost of simple sugar (glucose) without a more prolonged carbohydrate load will lead to repeat hypoglycemia in the patient.

Any diabetic adult who is not awake and oriented, or a patient with a blood glucose reading of less than 4.0 mmol/l should receive 50 cc of IV 50% dextrose (D50). If the patient is still symptomatic 10 minutes following dextrose administration then a repeat blood glucose reading should be obtained.

Intravenous glucose should also be administered to unresponsive patients with an unknown blood glucose level. This administration will not harm hyperglycemic patients as long as definitive therapy is initiated promptly.

50% Dextrose is extremely irritating to the peripheral veins and should be administered slowly over 3 to 5 minutes, with maintenance of IV continuing to run open. Patency of the intravenous line should be ensured prior to the administration of 50% Dextrose. Most patients should be functioning at their normal, baseline mentation within 5 to 10 minutes of intravenous dextrose administration. Oral glucose takes more time to produce the same effects. Rarely, patients who are hypoglycemic for extended amounts of time sustain irreversible brain injury.

Glucagon, 1mg IM/SC (intramuscular/subcutaneous), should be given to a hypoglycemic patient if IV access is unattainable or unsuccessful.

Hypotensive patients and patients with signs of dehydration or hypoperfusion should be administered isotonic IV fluids, such as normal saline (NS) or lactated Ringer's (LR) solution, to replace fluid volume. If the patient is hyperglycemic, especially with a blood sugar greater than

40 mmol/l, the patient is considered to be dehydrated. Fluid replacement is the mainstay of therapy for DKA and NKHC and should be undertaken aggressively because of the huge volume of fluid already lost. Ultimately, these patients will require insulin in addition to continuous volume replacement. Although the patient may be acidotic, bicarbonate should not be given; the acidosis will correct with fluid and electrolyte replacement and insulin. For fluid replacement, follow local protocols. The patient's ECG should also be monitored, because decreased potassium levels can lead to serious cardiac dysrhythmias.

Oral glucose is also an effective intervention when dealing with a diabetic patient and IV access is not possible. Glucose dosage for adults (over the age of 12) is 50 gms, while a child is 25 gms. It is important when administering oral glucose to watch the patency of the airway closely, and to have suction nearby should intervention be required. Oral glucose should only be administered if the patient has an in-tact gag reflex, and the level of consciousness permits (exception if certified in Hypoglycemia (EMR or EMT) Protocol).

It is important to remember to document well after administering any medications. The following information must be charted on the run sheet:

- Date and time
- Indications for initiating hypoglycemia protocol
- Blood glucose concentration (both prior to and following administration of glucose/dextrose)
- Dosage and route of administration
- Vital signs prior to and following administration
- Patients reaction to glucose/dextrose (any improvement or deterioration)
- Signature & license number

GLUCOMETER

Blood glucose values

The body tries to keep glucose in the blood within certain levels to maintain homeostasis. Although the range of blood glucose levels may vary between individuals, 4.0 - 7.0 mmol/l is considered to be normal.

Blood Glucose Reading	Interpretation	Treatment
0.0 - 2.9 mmol/L	Very Low	Administer glucose, treat signs & symptoms, initiate load & go
3.0 - 3.9 mmol/L	Low	Administer glucose, treat signs & symptoms
4.0 - 7.0 mmol/L	Normal	Treat signs & symptoms
7.1 - 19.9 mmol/L	High	Treat signs & symptoms
Above 20.0 mmol/L	Very High	Treat signs & symptoms, initiate load & go

INDICATIONS FOR BLOOD GLUCOSE TESTING

1. Patients with altered level of consciousness (may include intoxication).
Level of consciousness changes:
 - lethargy
 - agitation
 - confusion
 - seizures
 - coma
2. Patients with seizures
3. Patients with diabetes

PROCEDURE

NOTE: The exact procedure will depend on the type of glucometer you are using. It is vital to be totally familiar with the specific glucometer your service uses. Before taking a reading, be sure to utilize Routine Practices; GLOVES MUST BE WORN.

1. Determine that the patient meets the indications for blood glucose testing.
2. Gather the equipment and explain the procedure to the patient.
3. Prepare lancet or lancing device, such as Glucolet or Penlet.
4. Turn on the glucometer and insert a test strip. Machines differ in that some start up when the test strip is inserted while others require the glucometer to be turned on prior to test strip insertion.
5. Clean the fingertip that will be used to gather the blood sample with an alcohol swab and dry thoroughly.
6. If using a lancet, grasp the lancet fairly close to the point so as to control the depth of the puncture. Then, using a quick and controlled motion, prick the prepared fingertip. Immediately dispose of the lancet in a sharps container as it is now considered a contaminated sharp.

If using a lancing device, press it firmly against the selected site and trigger the spring release mechanism. These devices normally work very well, but occasionally you may come across patients with very thick skin that the device may not penetrate. In these situations you may have to use the lancing insertion as you would a regular lancet. Some of the lancing devices have depth regulators that may overcome this problem. This requires assessment of each patient's skin prior to lancing so as to determine the appropriate setting. The lancing device insert should

be immediately disposed of in a sharps container.

7. After the finger has been pricked, gently squeeze the finger proximal to the puncture site to increase the amount of blood presented. Once the necessary amount of blood is available, apply it to the test strip.

There are two main types of test strips. One requires the blood droplet to be placed on the strip while in the other type the end of the test strip is inserted into the blood which is automatically drawn into the strip. Different glucometers may require a different amount of blood to acquire accurate readings. Getting enough blood the first time avoids having to re-do the test unnecessarily. **KNOW YOUR EQUIPMENT.**

8. Once the sample has been obtained, cover the puncture site with sterile gauze. It will take approximately one minute for the glucometer to give its reading. This is a good time to reassess your patient.

9. If the patient was treated with glucose administration and transport time is over 10 minutes, then a second test would be in order. This would check the efficiency of the glucose treatment and may indicate that more glucose should be administered.

DOCUMENTATION

When blood glucose testing is done, certain documentation is required, including:

- Patient's condition prior to testing.
- Blood glucose level, recorded as mmol/L.
- Any treatment rendered based on readings.
- The patient's response to treatment.
- Any difficulties encountered during or after testing.
- Subsequent readings must be recorded as well as the times for all treatment.
- Signature and license number of paramedic performing any transfer of function skills.

GLUCOMETRY SKILL CHECK LIST

Acceptable Behaviour	A	U	NI
Identifies the indication for blood glucose testing			
Assesses patient history and vital signs			
Gathers equipment (including putting on gloves if not already done)			
Explains procedure to the patient			
Prepares lancing device			
Prepares glucometer (as per device design)			
Cleans selected finger on the patient with alcohol swab and dries thoroughly with a sterile gauze			
Lances the prepared finger using a quick and controlled motion to the depth sufficient to pierce the skin and cause bleeding			
Disposes of lancing device appropriately			
Gently squeezes lanced finger proximal to the puncture site			
Applies blood to glucometer testing source			
Covers puncture site with sterile gauze and controls bleeding			
Reassesses the patient while waiting for blood sample analysis			
Interprets blood glucose analysis results			
Initiates appropriate treatment based on patient's signs and symptoms and blood glucose reading			
Reports and records blood glucose analysis and treatment protocol			

PART 2, ALTERED MENTAL STATES

REVIEW OF THE NERVOUS SYSTEM

The nervous system coordinates the activities of the other body systems so the body functions smoothly. The nervous system also serves as the body's link with the outside world. This system enables us to detect stimuli (changes within the body or in the outside world) and to respond to them. Together with the endocrine system, the nervous system works continuously to preserve homeostasis.

The brain is the communication and control center of the body. It receives many kinds of input, processes and evaluates it, decides on the response or action taken, and then initiates the response. Responses include both involuntary (reflex) activities required to maintain homeostasis in the body which are regulated by the autonomic nervous system, and voluntary actions that are controlled by the somatic nervous system. With both reflex and voluntary activities, the individual is often not aware of the amount and diversity of input received or the integration or assessment of that input, but only of the response.

The nervous system is the body tissue that records and distributes information in the body using electrical and chemical transmission. It has two parts. The "central" nervous system is comprised of the brain and spinal cord. The "peripheral" nervous system is the nerve tissue that transmits sensation and motor information back and forth from the body to the central nervous system.

CENTRAL NERVOUS SYSTEM

- the part of the nervous system which consists of the brain and spinal cord, to which sensory impulses are transmitted and from which motor impulses pass out, and which coordinates the activity of the entire system.
- these nerves continually inform the CNS of changing conditions and then transmit its "decisions" to appropriate muscles and glands that make the adjustments needed to preserve homeostasis.

PERIPHERAL NERVOUS SYSTEM

- the part of the nervous system that is outside the central nervous system and comprises the cranial nerves excepting the optic nerve, the spinal nerves, and the autonomic nervous system.
- consists of the cranial nerves (12 pairs) and peripheral nerves (31 pairs) ; the peripheral nervous system has both involuntary and voluntary components.
- may be subdivided into somatic and autonomic divisions; receptors and nerves concerned with changes in the outside environment are somatic, while those that regulate the internal environment are autonomic.

ANATOMY OF THE NERVOUS SYSTEM

The brain is the portion of the central nervous system that is located within the skull. It functions as a primary receiver, organizer, and distributor of information for the body. It has two (right and left) hemispheres. The brain consists of many parts which function as an integrated whole. The major divisions, named in ascending order beginning with the most inferior part are:

1. Brainstem:

- a. The medulla oblongata is located between the brain and spinal cord. It marks the divisions between the spinal cord and the brain. It contains the vital control centers regulating respiration, cardiovascular function, and vasomotor activity.
- b. The pons is between the midbrain and the medulla oblongata, composed of bundles of afferent and efferent fibers - connections between the brain and the spinal cord.
- c. The midbrain extends from the pons to the hypothalamus and integrates several different kinds of reflexes including visual and auditory reflexes.

2. Cerebellum:

- a. Located dorsal to the pons and medulla, below the occipital lobe.
- b. Consists of two hemispheres closely related to the brainstem and higher centers.
- c. The cerebellum coordinates fine motor movement, posture, equilibrium, and muscle tone.

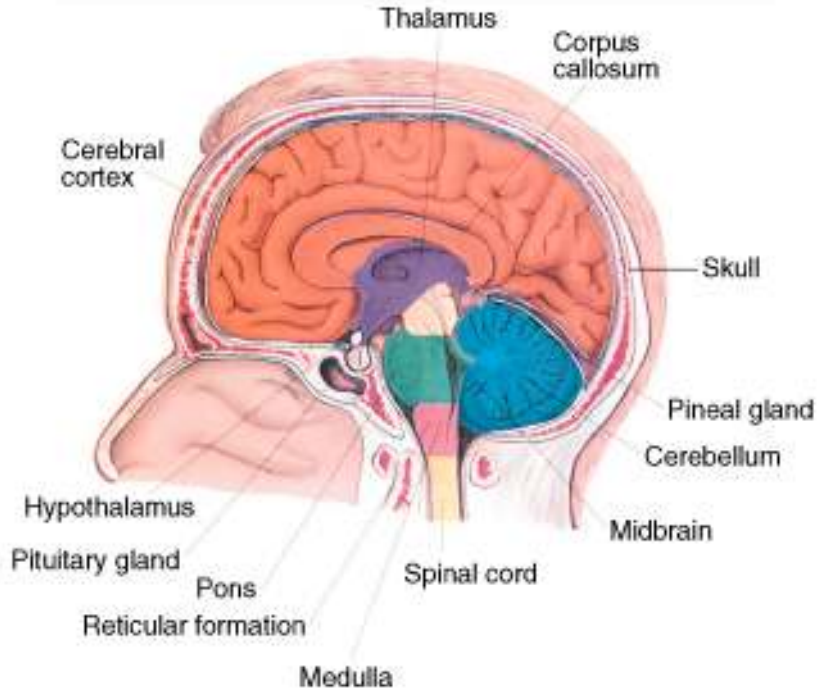
3. Diencephalon:

- a. hypothalamus: located superior to the pituitary gland and inferior to the thalamus, the hypothalamus is the area of the brain that controls body temperature, hunger and thirst.
- b. thalamus: located superior to the hypothalamus and inferior to the cerebrum, the main function of the thalamus is sensation. The thalamus integrates the impulses so that the cerebrum is able to interpret the sensation quickly.

4. Cerebrum:

- a. The largest portion of the brain, consisting of right and left hemispheres.
- b. Responsible for memory, thought, speech, voluntary movement, and sensory perception.

MAJOR REGIONS OF THE CENTRAL NERVOUS SYSTEM



Brain cells require a continuous supply of oxygen and glucose. The brain is so dependent on its blood supply that when it is deprived of it, consciousness may be lost very quickly, and irreversible damage may occur within a few minutes. In fact, the most common cause of brain damage is a stroke, or cerebrovascular accident (CVA).

Blood flow to the brain is provided by two systems - the carotid system (anterior), and the vertebral system (posterior). Both systems join at the Circle of Willis before entering the substance of the brain. The system is designed so that an interruption to one part of the brain will not cause significant loss of blood flow to all the tissues. Venous drainage of the brain is through the venous sinuses and the internal jugular veins. Besides blood flow, cerebrospinal fluid (CSF) bathes the brain and spinal cord.

The spinal cord is 17 - 18 inches long. It leaves the brain through the foramen magnum, down the spinal canal. The spinal cord is surrounded and protected by the spinal column, made up of 33 vertebrae, which are broken up into 5 sections: cervical spine (7 vertebrae), thoracic spine (12 vertebrae), lumbar spine (5 vertebrae), sacrum (5 vertebrae), and coccyx (4 vertebrae). The spinal cord ends at the level of the first lumbar vertebra. The spinal cord has two main functions. It controls many reflex activities of the body, and it transmits information back and forth from the nerves of the peripheral nervous system to the brain.

The nervous system is made up primarily of two types of nerves. There are 12 pairs of **cranial nerves** which originate in the brain and supply nervous control to the head, neck, and certain thoracic and abdominal organs. There are 31 pairs of **peripheral nerve fibers** which exit the spinal cord as it descends and enters the peripheral nervous system. The dorsal roots contain

afferent fibers, the ventral roots contain *efferent* fibers. Afferent fibers carry impulses toward the center of the body. Sensory nerves send messages toward the brain and are thus afferent. Efferent fibers carry conducted impulses away from the brain or spinal cord to the periphery.

PROTECTION OF THE CENTRAL NERVOUS SYSTEM

The central nervous system is well protected. The soft, fragile brain and spinal cord are the most carefully protected organs in the body. Both are encased in bone, covered by three layers of connective tissue, and bathed in a cushioning fluid.

The three connective tissue layers covering the brain and spinal cord are the **meninges**. The outermost of the meninges is the **dura mater**, a tough, double-layered membrane. Inside the skull, the two layers of the dura mater are separated in some regions by large blood vessels called sinuses. These vessels receive blood leaving the brain and deliver it to the partitions (septa) that subdivide the cranium into compartments. The largest of these partitions (the falx cerebri) dips down between the cerebral hemispheres.

The second of the meninges is the **arachnoid**, a thin, delicate membrane. Thread-like fibers of the arachnoid extend like the threads of a web to the innermost meningeal layer, the **pia mater**. The pia mater is a very thin membrane that adheres closely to the brain and spinal cord, following each curve or indentation of the tissue. It has many blood vessels.

The shock-absorbing **cerebrospinal fluid** (CSF) fills the ventricles, the cavities within the brain, and the spaces below the arachnoid layer (subarachnoid space) in the brain and spinal cord. Most of the CSF is produced by clusters of capillaries, the choroid plexuses, which project from the pia mater into the ventricles. The CSF circulates through the ventricles, and then passes into the subarachnoid space. Finally, it is reabsorbed into the blood through structures called arachnoid granulations. These structures project from the arachnoid layer into large blood sinuses within the dura mater.

The brain actually floats in the CSF. The CSF protects against mechanical injury. It dissolves and transports substances filtered from the blood and serves as a medium for the exchange of nutrients and waste products between the blood and the brain. A normal volume of CSF is essential to normal nervous system function.

AUTONOMIC NERVOUS SYSTEM

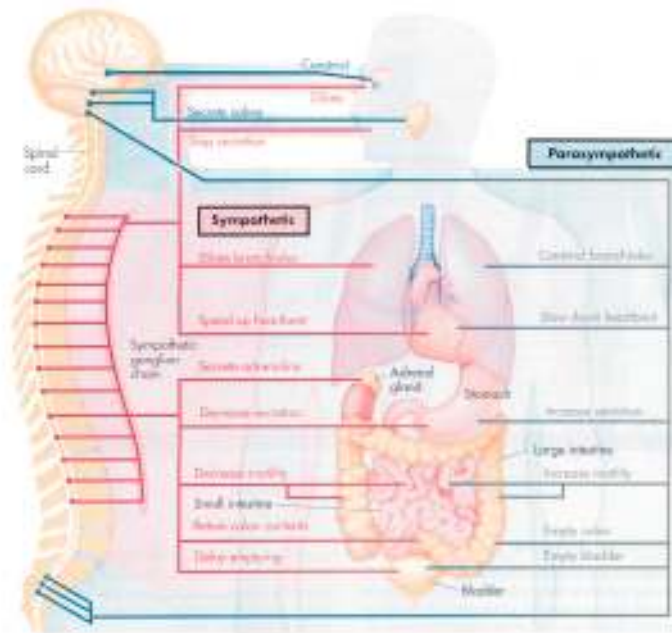
The autonomic nervous system is the involuntary component of the peripheral nervous system. It is responsible for the unconscious control of many body functions and has two functional divisions - the sympathetic and parasympathetic nervous system. The autonomic system provides motor and sensory innervation to smooth muscle, cardiac muscle, and glands.

The sympathetic nervous system, which is often referred to as the “fight-or-flight” system, prepares the body for stressful situations. Stimulation causes increased heart rate and blood pressure, pupillary dilation, rise in the blood sugar, as well as bronchodilation. Epinephrine and norepinephrine mediate its actions.

The parasympathetic nervous system, which is sometimes called the “feed-or-breed” system, is

responsible for controlling vegetative functions. When stimulated, it causes a decrease in heart rate, an increase in digestive activity, pupillary constriction, and a reduction in blood glucose. During rest the parasympathetic system dominates. Acetylcholine is the neurotransmitter for the parasympathetic nervous system.

THE AUTONOMIC NERVOUS SYSTEM



SEIZURES

A seizure is a sudden and temporary alteration in behaviour caused by massive electrical discharge in a group of nerve cells in the brain. The abnormal electrical discharge typically produces changes in mental activity and behaviour ranging from brief trance-like periods of inattention to unresponsiveness and the jerky muscle spasms known as a convulsion. A seizure is not a disease in itself but rather a sign of an underlying defect, injury, or disease. A common cause of seizures is *epilepsy*, a chronic brain disorder characterized by recurrent seizures.

There are six common types of seizures.

- **Absence:** or *petit mal*, usually occurs in children between 4 & 12 years of age, although they can occur in adults. The patient will have a blank stare and repeat behaviours (such as lip smacking), typically lasts from 1 to 10 seconds.
- **Focal:** characterized by dysfunction of one area of the body. Focal seizures begin as localized tonic-clonic movements and frequently spread and appear as full tonic-clonic seizures.
- **Psychomotor:** characterized by distinctive auras which include unusual smells, tastes, sounds, or the tendency of objects to look either very large and near, or small and distant. Psychomotor seizures are a form of focal seizures lasting approximately 1 to 2 minutes.

- **Tonic Clonic:** also called *grand mal* or generalized motor seizures, causes alternating tonic (contractions) and clonic (successive contractions and relaxations) movements of the extremities beyond the patient's control.
- **Hysterical:** stem from psychological disorders. The patient presents with sharp and bizarre movements that can often be interrupted with curt commands. There is usually no postictal period.
- **Febrile:** a tonic clonic seizure resulting from sudden high fevers, particularly in children.

PARTIAL VS. GENERALIZED SEIZURES:

Partial seizures can be divided into simple partial and complex partial seizures. *Simple partial seizures* occur when a localized area of the brain becomes involved with abnormal electrical discharge. The patient does not normally lose consciousness nor is there any alteration in baseline mentation. During partial seizures, motor movements are simple, such as jerking or twisting of the head to one side. There is usually no postictal period. The patient later may be able to describe the attack.

Complex partial seizures usually do cause altered mental status. The patient may be observed to be wandering around aimlessly, similar to sleepwalking. Automatisms, such as lip-smacking and fumbling, may be observed. The patient may experience inappropriate emotional feelings, such as crying or laughter, may disrobe or may run away for no apparent reason. After the event, the patient may appear postictal and may not be able to recall the episode.

Generalized seizures can be divided into absence and tonic clonic seizures, as discussed on the previous page.

Status epilepticus or a status seizure typically occurs when a seizure lasts for more than 5 minutes, or when two or more seizures occur sequentially without returning to a normal level of consciousness. A status seizure represents a true life-threatening condition.

COMMON CAUSES OF SEIZURES:

- | | |
|-----------------|---------------------------|
| ⇒ High Fever | ⇒ Hypoxia |
| ⇒ Infection | ⇒ Stroke |
| ⇒ Poisoning | ⇒ Drug/alcohol withdrawal |
| ⇒ Hypoglycemia | ⇒ Dysrhythmias |
| ⇒ Hyperglycemia | ⇒ Hypertension |
| ⇒ Head injury | ⇒ Pregnancy complications |
| ⇒ Shock | ⇒ Idiopathic (unknown) |

SIGNS & SYMPTOMS:

Although specific types of seizures result in specific signs and symptoms, the following are commonly associated with some type of seizure behaviour.

- Loss of consciousness
- Blank stares
- Eye twitching & lip smacking
- Nausea
- Twitching
- Preceding aura (may be a smell, visual, or auditory)
- Muscle contraction (Tonic) & relaxation (Clonic) to one part of the body or the whole body
- Clenched jaw, respirations cease during active seizure
- Increased salivation
- Incontinence
- Post seizure pt. will be confused, fatigued, and achy
- Repeated motor activity
- Tingling sensation
- Inappropriate behaviour
- Hallucinations

ASSESSMENT:

Scene size-up and general impressions are an important factor in the assessment of seizure patients. Because seizures could be a sign of head injury, look for a mechanism or injury that may suggest blunt trauma or penetrating injury to the head. Also, check the environment for any evidence of poisoning, such as pill bottles and syringes. Look for prescription medications that may indicate a potential history of epilepsy, diabetes, or heart disease. Form a general impression of the patient as you begin the initial assessment. Whether you find the patient actively seizing or in a postictal state, consider both of these to be an altered mental state which warrants close assessment of the airway, breathing, and circulation. The patient who is not responding to verbal stimuli following the seizure episode, the patient who is actively seizing, or the patient who has suffered more than one consecutive seizure without an intervening period of responsiveness is at the greatest risk for airway, breathing, and circulation compromise. Although the postictal patient is often confused, disoriented, weak and exhausted, typically these patients have an open airway, are breathing on their own, and have adequate circulation.

Physical assessment of a patient who is actively seizing is very difficult. Most commonly, the number one priority in a patient who is seizing is to protect the patient from harming themselves by moving objects away from them and ensuring there are no immediate hazards around. Protect the patient from falling unsupported to the ground, and/or striking objects during a seizure. Position the patient on his or her side with the head supported in a neutral in-line position. Although it is important to protect the patient from striking his or her head and other parts of the body, **do not restrain the patient**. Restraining the head could result in cervical spine injuries. Establish ABC's and administer high flow concentration oxygen. It is important to have suction readily available. Maintain the patient's dignity by removing bystanders and covering the patient

if possible. Note and record the duration and pattern or characteristics of the seizure(s).

Because seizures generally only last a couple of minutes, commonly upon arrival the patient is in a **postictal state** and is not actively seizing. The postictal state follows the seizure and is the recovery period for the patient. During this period, the patient may be unresponsive, very sleepy, weak, and disoriented. Because such a large number of muscles contract during a grand mal seizure (generalized tonic-clonic seizure), patients are often extremely tired. The patient will slowly but progressively regain complete responsiveness and orientation. This phase may last up to 30 minutes.

Post Seizure: Reassess airway, breathing, and circulation. Be sure the patient has an open, patent airway - have suction ready. Continue to administer oxygen until the patient is fully alert. Perform a detailed physical assessment (secondary survey) to ensure the patient was not injured during or as a result of the seizure. Pertinent medical history is important in assessing the patient. In addition to the routine SAMPLE history questions, it is important to ask:

- If the patient has a known seizure disorder
- What medications were taken, and when
- Medical identification
- Suspected alcohol / drug abuse
- If the patient has suffered recent trauma or illness
- If the patient has a fever (or has had recently)
- If the patient has a history of Diabetes

If it is within your scope of practice, obtain a blood glucose reading and follow hypoglycemia protocol if necessary. Note any fever, particularly in children under the age of six. Treat any other injuries found on physical exam (secondary survey). Post seizure patients may be hypoxic, oxygen saturations should be monitored within ones scope of practice, and ventilations should be assisted if required. High flow oxygen concentration to the patient should be maintained and frequent re-assessment of ABC's and vital signs should be priority. If the patient is suspected of having a febrile seizure (common in children), remove clothing and cool the patient down while being cautious not to allow the child to become hypothermic.

Status epilepticus is a dire medical emergency that requires aggressive airway management, positive pressure ventilation with supplemental oxygen, and immediate transport. The longer the delay in treatment is, the greater the chance of the patient suffering permanent brain damage.

For any patient who has a generalized seizure lasting longer than five (5) minutes, the following medications should be considered.

Diazepam

Adult: (16 years old or greater)

- 5 mg IV as an initial dose
- repeat IV dose may be given every 3 minutes
- maximum total dose: 20 mg

Adolescent: (10 - 15 years old)

- 2.5 mg IV as an initial dose
- repeat IV dose may be given every 3 minutes
- maximum total dose: 10 mg

Pediatric: (0 - 9 years old)

- 0.2 mg / kg IV as initial dose (max 2.5 mg as initial dose)
- repeat IV dose may be given every 3 minutes
- maximum total dose: 5 mg
- Diazepam administration should be stopped if seizure stops, maximum dose is reached, or there is evidence of respiratory depression. See pharmacology page 37 for contraindications.

Lorazepam

Adult & Adolescent: (10 years and greater)

- 2 mg intrabucally as an initial dose
- repeat dose may be given every 10 - 15 minutes
- maximum total dose: 4 mg

Pediatric: (9 years and younger)

- not indicated in pediatric patients

DOCUMENTATION

When any of the previous medications have been administered, certain documentation is required, including:

- Patient's condition prior to testing (presenting signs & symptoms, including vital signs and LOC)
- History of seizure, its features, and duration.
- Indications for protocol use.
- Dose and time(s) for each drug dose administered and resulting clinical effects.
- Repeat assessment and vital signs, as indicated.
- Changes from baseline, if any, that occur during treatment or transport.
- Amount of diazepam or lorazepam discarded, if any.
- Signature and license number or paramedic performing any transfer of function skills. A second signature is required from another crew member or health care staff, witnessing discarding of un-used diazepam or lorazepam (if applicable).

CEREBROVASCULAR ACCIDENT & TRANSIENT ISCHEMIC ATTACK

Cerebrovascular Accident (CVA), most commonly referred to as a stroke or “brain attack“, is a general term that describes injury or death of brain tissue due to interruption of cerebral blood flow resulting in neurological deficit.

Transient Ischemic Attack (TIA), sometimes referred to as a “mini-stroke”, is a temporary interruption of blood supply to the brain caused by small emboli that result in transient stroke-like symptoms. These attacks may last a few minutes or for several hours. No residual brain or neurological damage accompanies the attack. A TIA is thought to be the most important indication of impending stroke; about 5% of patients with a TIA will develop a complete stroke within 1 month if untreated.

TYPES OF CEREBROVASCULAR ACCIDENTS

	Thrombus	Embolus	Hemorrhage
Predisposing Condition	Atherosclerosis in cerebral artery	Atherosclerosis or systemic source	Hypertension, Atherosclerosis
Onset	Gradual, may be preceded by TIA's	Sudden	Sudden, occurs often with activity
Increased ICP Effects	Minimal, Localized	Minimal, Localized unless multiple emboli are present	Present, widespread and severe - often fatal

There are two categories of stroke. **Ischemic** stroke occurs when the blood supply to a limited portion of the brain is inadequate and death of nervous tissue follows. Infarction can be caused by an embolism, by blood vessel occlusion due to atherosclerosis, or by pressure from a mass within the brain itself. Ischemic strokes account for approximately 80% - 85% of all strokes. The onset of stroke from cerebral thrombosis usually is associated with a long history of vessel disease, therefore the majority of these patients are older and have evidence of atherosclerotic disease in other areas of the body (such as angina pectoris, or previous strokes). A stroke caused by an embolus results from an occlusion of any intracranial vessel by a fragment of a foreign substance arising outside of the central nervous system. Common sources of cerebral emboli include: atherosclerotic plaques (originating from large vessels of the head, neck, or heart); thrombi that develops on the valves or in the chambers of the heart (very common in patients with valvular disease and atrial fibrillation). Although not as common, air embolism after thoracic injury, fat embolism after long bone injury, and women taking oral contraceptives have also been known to be causes of cerebral emboli. Five minutes or more of ischemia causes irreversible cell damage.

Cerebral hemorrhage accounts for 15% to 20% of all strokes. A hemorrhage may occur

anywhere within the cranial vault, including the epidural, subdural, subarachnoid, and intraventricular spaces. Unlike ischemic stroke, which have relatively high survival rates, cerebral hemorrhages are fatal in 50% to 80% of cases. Hemorrhagic strokes are commonly caused by cerebral aneurysms and hypertension.

Both forms of stroke can be life threatening. Ischemic stroke however, rarely leads to death within the first hour. Hemorrhagic stroke can be rapidly fatal. Understanding the various signs & symptoms of each type of stroke will better prepare the paramedic to anticipate the course of patient care.

SIGNS & SYMPTOMS OF CVA

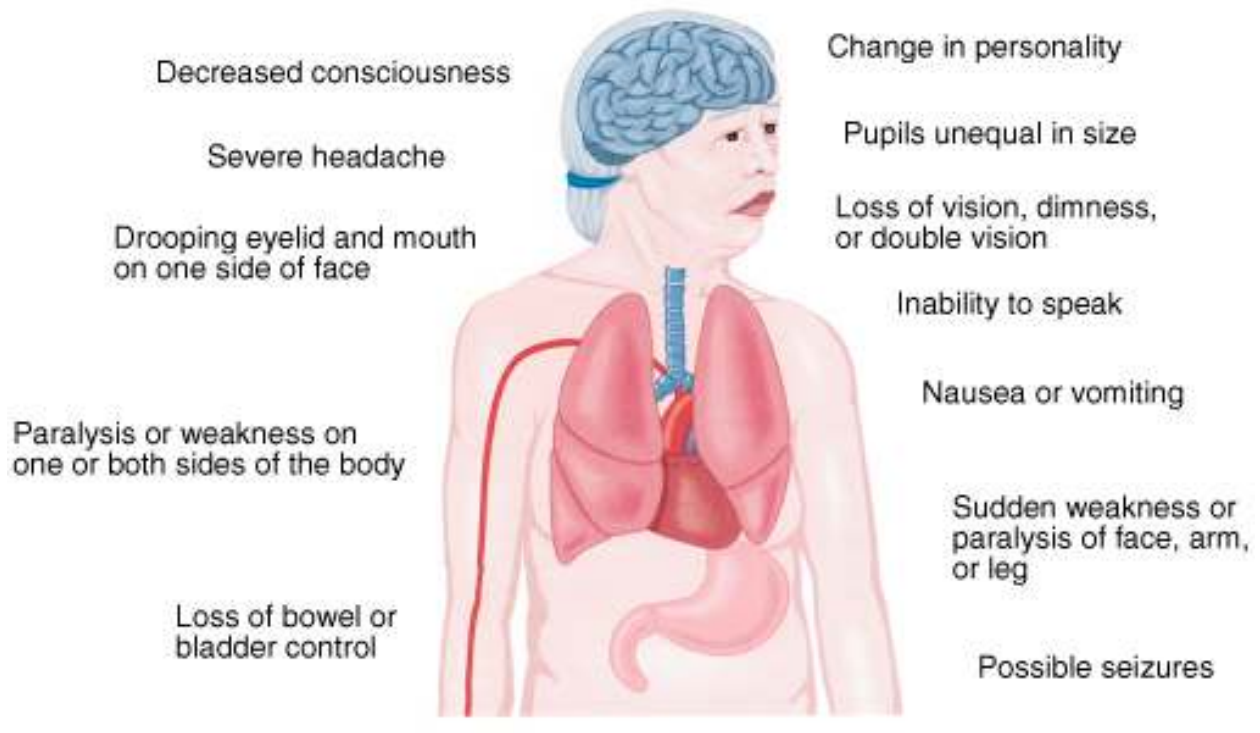
Ischemic Stroke	Hemorrhagic Stroke
Convulsions	Nausea
Incontinence	Vomiting
Double vision (diplopia)	Progressive deterioration in mental status
Numbness of the face	Headache
Slurred speech (dysarthria)	Loss of consciousness
Headache	Seizure
Dizziness or vertigo	Cushing's Reflex (hypertension & bradycardia)
Weakness / paralysis on one or both side(s) of the body	Unequal pupils

Risk factors for a stroke can be divided into two categories; modifiable, and non-modifiable. Modifiable risk factors include: high blood pressure, cigarette smoking, transient ischemic attacks, heart disease, carotid bruit, diabetes mellitus, and sickle cell anemia. Non-modifiable risk factors include age, gender (men are at a greater risk than women), race (African-Americans are at a greater risk), prior stroke, and heredity. The five most common risk factors associated with CVA include hypertension, diabetes mellitus, atherosclerosis, hyperlipidemia (excessive fat in the blood), and cardiac disease.

It is important to realize that specific signs & symptoms depend on the location of the obstruction, the size of artery involved, and the functional area of the brain affected.

Signs and symptoms of a transient ischemic attack (TIA) are the same as those that characterize stroke and include weakness, paralysis, numbness of the face, and speech disturbances. Although symptoms may only last from 5 to 10 minutes, most patients that experience a TIA are hospitalized for observation, evaluation, and treatment.

GENERAL SIGNS AND SYMPTOMS OF STROKE



PATIENT ASSESSMENT & FOCUSED HISTORY:

When called to assist a patient with a possible stroke, you should first determine whether the patient is responsive and breathing. You should make a special effort to find out from family or friends when the patient last appeared to be normal. This will aid in the decision at the hospital whether or not thrombolytic therapy can be used.

Initial Assessment: Upon arrival, you should check and care for immediate problems with the patient's airway, breathing, and circulation. Maintenance of a patent airway is essential, as the patient may continue to deteriorate and the airway may become compromised. It may be necessary to insert an airway adjunct to ensure the airway is kept open. If the patient is conscious, the nasopharyngeal airway is preferred because it is better tolerated by the patient. Since vomiting is associated with a brain injury, be prepared to remove secretions by suctioning. As the patient deteriorates, breathing may become inadequate. If the rate or quality of breathing is inadequate, begin positive pressure ventilation with supplemental oxygen. It is important to recognize early signs of respiratory failure. Inadequate breathing could drastically increase injury to the brain because of decreased oxygen flow to the brain and a build-up of carbon dioxide. If breathing is adequate, continue oxygen therapy via non-rebreathe mask at 15 lpm. The patient's cardiovascular state should be monitored closely to rule out the possibility of cardiac compromise. Cardiac dysrhythmias are frequent, and therefore the patient's ECG and blood pressure require constant monitoring.

Focused History: Along with the routine SAMPLE history, the following are important components in a CVA patient history:

- Previous neurological symptoms (TIA's)
- Previous neurological deficits
- Initial symptoms & their progression
- Alterations in level of consciousness
- Precipitating factors
- Presence of dizziness or palpitations
- Significant past medical history including hypertension, diabetes mellitus, smoking, cardiac disease, oral contraceptive use, sickle cell disease, and previous CVA.

If the patient is unable to protect his own airway because of a reduction in mental status, place the patient in the recovery position. The responsive patient should be placed in a supine position with the head and chest elevated.

Since the patient cannot move the paralyzed extremity, it is vital that the paramedic protect the extremities from any injury, particularly when moving the patient.

While en route to the hospital, it is appropriate to perform a detailed physical exam (secondary survey) if the patient's condition and time permits. As soon as possible, perform a neurological exam as part of the focused physical exam. You should perform at least three key physical tests on patients you suspect of having had a stroke: tests of speech, facial movement, and arm movement. Ask the patient to show their teeth or to smile, this will test facial droop. Normally both sides of the face move equally well. Ask the patient to close their eyes and hold both arms out with palms up. If one arm does not move, or one arm drifts down compared with the other side, it is possible that the patient is having a new stroke. Lastly, ask the patient to repeat a phrase such as "You can't teach an old dog new tricks". If the patient slurs words, uses inappropriate words, or is unable to speak, consider the possibility of a stroke. These three neurological tests are often referred to as the "Cincinnati Prehospital Stroke Scale". Patients with one of these 3 findings - as a *new* event - have a 72% probability of an ischemic stroke; if all 3 findings are present the probability of an acute stroke is more than 85%. Using this scale can help the paramedic identify a stroke patient who requires rapid transport & enables pre-arrival notification of the receiving hospital.

Patients who suffer a CVA may also have sustained an injury. During the detailed exam, the sensory and motor function should be reassessed closely in all extremities. If the patient is unconscious, rule out hypoglycemia by obtaining a blood glucose reading. Document and report any changes from the earlier focused history and rapid assessment in the patient's speech, sensory, and motor function.

Perform an ongoing assessment every 5 minutes. Pay special attention to the status of the airway, breathing, circulation, and mental status. This is extremely important since many non-traumatic brain injured patients deteriorate rapidly and significantly. Repeat and record the baseline vital signs. Note any changes in the patient's condition and communicate these to the receiving medical facility. Be prepared to support the patient's respiratory and circulatory functions.

NOTE

- Patients who have had a CVA may benefit from early thrombolytic therapy. Therefore, on-scene time should be kept to a minimum and transport initiated as early as possible to the nearest appropriate health care facility.
- Talk to the patient and keep them informed. While patients who have suffered a stroke may be unable to speak, they may be aware of their surroundings - they should have treatments and actions explained to them and they should be reassured.
- Strokes not only occur in elderly patients, they can and do occur in every age group and in both sexes.
- Signs & Symptoms of TIA's are very similar, and should be treated as a CVA to prevent causing permanent neurological damage by delaying treatment or transport.

NEW ADVANCES IN ACUTE STROKE MANAGEMENT

In 1996 the AHA recommended the use of fibrinolytic therapy within 3 hours of symptom onset for selected patients with ischemic stroke. After an in-depth review of the available evidence, the AHA Stroke Task Force concluded that fibrinolytic therapy was supported by sufficient evidence to receive a Class IIb recommendation.

The Stroke Expert Panels from the Guidelines 2000 Conference made no changes to the Stroke Algorithm. Research has continued to accumulate in support of the effect of fibrinolytic therapy when given to carefully selected patients within 3 hours of the onset of symptoms of acute ischemic stroke. This is a Class I recommendation, with the important caveat that it must be given according to the NINDS protocol, which requires a careful search for indications and contraindications.

Intra-arterial fibrinolytic therapy may be beneficial for patients with middle cerebral artery occlusion 3 to 6 hours after the onset of symptoms (Class IIb recommendation).

Intravenous fibrinolytic therapy within 3 to 6 hours of onset of symptoms (this recommendation received an Indeterminate rating because of insufficient evidence to support its efficacy).

The following actions must take place to provide the patient with the best possible outcome related to Fibrinolytic therapy.

- recognize the signs of transient ischemic attack (TIA) and stroke
- perform a rapid neurologic examination that includes the elements of the Cincinnati Prehospital Stroke Scale
- determine (if possible) the time of symptom onset
- provide rapid transport to an ED capable of caring for patients with acute ischemic stroke
- assess and support cardiorespiratory function as needed during transport
- obtain serum glucose levels and treat accordingly
- notify the receiving hospital early that a possible stroke victim is in transport
- support fluid status
- understand the potential risks and benefits of fibrinolytic therapy for selected patients with acute ischemic stroke who present within 2 hours of symptom onset
- understand the inclusion and exclusion criteria for fibrinolytic therapy

SYNCOPE

Syncope, or fainting, is a sudden and temporary loss of consciousness. It occurs when there is a temporary lack of blood flow to the brain and the brain is deprived of oxygen for a brief period. Syncope usually occurs when the patient is standing up or when the patient suddenly stands up from a sitting position. Some patients feel as though everything is going dark, and then they suddenly become unresponsive. The collapse that follows puts the body in a horizontal position, allowing blood circulation to the brain to improve. As a result, the patient generally recovers rapidly.

There are many causes for syncope, the most common ones including stroke, neoplasms of the CNS, cardiac abnormalities, and orthostatic hypotension. The focus of assessment in the syncope patient should be to obtain an extensive patient history, which should help the paramedic understand what the cause of this episode could be, and possibly prevent another episode.

In caring for a patient who has experienced a syncopal episode, assume that there could be head or spine injury and treat accordingly. Loosen any restricting clothing and administer high flow concentration oxygen via non-rebreathe mask. Place the patient in a supine position with the lower extremities elevated to allow for improved blood flow to the brain. Place the patient on supplemental oxygen and monitor vital signs closely. It is important to recognize that syncope could be a sign of a serious illness or injury. If the patient is not responding appropriately upon your arrival, consider the condition an altered mental status. Perform an initial assessment, rapid assessment and manage the patient's airway, breathing, and circulation as in any altered mental status patient. If the patient has not fallen "flat", make sure to position in a supine position for assessment, maintaining manual stabilization of the head and spine if spinal injury is suspected.

Diagnosis of syncope is difficult. There are many cases of unresponsiveness, weakness, dizziness, and other conditions that may appear to be syncope but are not. Syncope is an actual loss of consciousness. Signs and symptoms such as dizziness, vertigo, weakness, and other conditions should be differentiated from syncope by an accurate history. These conditions may be indicative of serious illness and should be explored. Remember that the loss of consciousness with syncope is brief. A prolonged state of unresponsiveness is not syncope. The transient nature of syncope prompts many patients to believe that transportation to the hospital is not necessary. Make every effort to persuade patients to accept care and transport as many causes of syncope (i.e. dysrhythmia) may recur, and if not treated, can result in death.

ALTERED MENTAL STATUS: UNKNOWN ETIOLOGY

An altered mental status is a significant indication of injury or illness in a patient. The alteration may range from simple disorientation to complete unconsciousness in which the patient is not responsive, even to painful stimuli. A change in the patient's mental status is an indication that the central nervous system has been affected in some manner. Causes may include trauma, where the brain is injured from a blunt force or penetrating object, or non-traumatic causes such as alterations in the patient's blood sugar level or blood oxygen level. In any patient with an altered mental status, it is vital that you manage any life-threatening injuries or conditions,

recognize the mental status change, document it, and continue to monitor the patient for further deterioration.

ASSESSMENT: ALTERED MENTAL STATUS WITH UNKNOWN HISTORY

Perform a scene size-up to begin to find out why the patient has an altered mental status. Based on the dispatch information and a scan of the scene, it is important to determine if the patient has been injured or is suffering from a medical illness. As you are approaching the patient inspect the scene for a mechanism of injury that would be significant enough to cause an alteration in mental status. This information may be gathered from your dispatch information, the patient, the relatives, or bystanders at the scene.

If no mechanism of injury is apparent, you would then suspect that the altered mental status is a result of a medical illness. As you are approaching the patient, look for clues that may indicate the nature of the illness. Alcohol bottles, drug paraphernalia, home oxygen tanks, and chemicals may help explain the cause. The patient's medications may provide the most valuable information to you. Have a family member gather the patient's medications while you are performing your assessment. It is important to look at both prescription and non-prescription medications.

Perform the initial assessment, stabilizing the spine if injury is possible. Pay particular attention to the patient's airway and breathing. Severe alterations in mental status resulting in unresponsiveness may cause the patient to lose the ability to maintain his own airway. The jaw and tongue become relaxed, fall back, and block the airway. Also, an unresponsive patient commonly has no gag reflex and, therefore, is unable to keep his airway clear of secretions, blood, and vomitus. The breathing rate and depth may be inadequate, so be prepared to assist with ventilations. All patients with altered mental status must receive high flow oxygen therapy because ensuring an adequate supply of oxygen to the brain is important in maintaining or restoring mental function. It is also important to recognize that the poor breathing status or blocked airway may be the cause of the altered mental status, as well as the result.

Conduct the focused history and physical exam. Baseline vital signs must be obtained at this time in order to aid in monitoring for further deterioration or improvement in the patient's condition. A blood glucose test should be taken to rule out the possibility of a diabetic related altered mental status. During transport, persons suffering from altered mental status should be transported in the recovery position, injuries permitting.

While we often use the patient as our primary historian during patient history, it is important to remember with altered mental status that if the patient is disoriented, or suffering a severely depressed mental status, he may be unable to provide the necessary answers. This is where family and bystanders could be of use. During the SAMPLE history, it is important to ask the following questions:

- What were the signs and symptoms the patient was complaining of prior to the alteration in the mental status?
- Did the signs and symptoms seem to get progressively worse or better?

- Does the patient have any known allergies?
- What medications, prescription and non-prescription, is the patient taking?
- What is the patient's past medical history? When was the last time he has seen a doctor for his medical condition?
- When did the patient last have something to eat or drink? What did he eat or drink? Did he take any drugs or ingest any alcohol?
- What was the patient doing prior to the onset of the altered mental status?
- Was the onset of signs and symptoms gradual or sudden?
- Did the patient suffer from a seizure, severe headache, or confusion prior to the alteration in the mental status?
- How long has the patient been sick or suffering from these signs and symptoms? When was the patient last well?

SIGNS & SYMPTOMS: ALTERED MENTAL STATUS, UNKNOWN ETIOLOGY

The signs and symptoms associated with an altered mental status will vary depending on the cause. Common signs and symptoms of altered mental status associated with trauma are:

- Obvious signs of trauma; deformity, contusions, abrasions, punctures or penetrations, burns, tenderness, lacerations, or swelling
- Abnormal respiratory pattern
- Tachycardia or Bradycardia
- Unequal pupils
- Hypertension or Hypotension
- Raccoon eyes (dark discoloration around the eyes)
- Battle signs (discoloration behind the ears)
- Pale, cool, moist skin
- Decorticate posturing (arms flexed, legs extended) or Decerebrate posturing (arms and legs extended)

Common signs and symptoms of altered mental status associated with *non-trauma* or *medical illness* are:

- Abnormal respiratory pattern
- Dry or moist skin
- Cool or hot skin
- Pinpoint, mid-size, dilated, or unequal pupils
- Stiff neck
- Lacerations to the tongue indicating seizure activity
- Cushing's Reflex (high systolic blood pressure and low heart rate)
- Loss of bowel or bladder control

There are many different medical conditions and injuries that could lead to an altered mental status. Some of the more frequent causes are:

- Shock
- Poisoning or drug overdose

- Post seizure (the patient has suffered a seizure and is just beginning to recover)
- Infection
- Traumatic head injury
- Decreased oxygen levels due to an inadequate airway or breathing
- Alcohol intoxication
- Stroke (cerebral vascular accident)
- Diabetes

The following mnemonic (AEIOU TIPS) may help in determining the cause of altered mental status.

A acidosis, alcohol
E epilepsy
I infection
O overdose
U uremia (kidney failure)
T trauma, tumor
I insulin (hypoglycemia or DKA)
P psychosis
S stroke

PHARMACOLOGY

DEXTROSE (D50W)

Class

- hypertonic carbohydrate solution

Mechanism of Action

- rapidly increases serum glucose levels
- short-term osmotic diuresis

Indications

- hypoglycemia
- altered level of consciousness
- coma or seizure due to hypoglycemia

Contraindications

- intracranial hemorrhage
- delirium tremens

Adverse Reactions / Side Effects

- extravasation leads to tissue necrosis
- warmth, pain, burning, thrombophlebitis, rhabdomyositis

Interactions

- sodium bicarbonate
- coumadin

Dosage and Administration

- adult: 12.5-25 gram slow IV; may be repeated as necessary
- pediatric: 0.5-1 gram / kg / dose slow IV; may be repeated as necessary
note: when administering to a pediatric patient, D25W should be used; if not available, D50W should be diluted 1:1 with sterile water

Duration of Action

- onset: less than 1 minute
- peak effects: variable
- duration: variable

Special Considerations

- consider administering thiamine (if available and protocol permits) prior to D50W in known alcoholic patients
- administer blood glucose testing prior to administering
- do not administer to patients with known CVA unless hypoglycemia is documented

DIAZEPAM

Class

- benzodiazepine, sedative-hypnotic, anticonvulsant

Mechanism of Action

- potentiates effects of inhibitory neurotransmitters
- raises seizure threshold
- induces amnesia and sedation

Indications

- acute anxiety states, acute alcohol withdrawal, muscle relaxant, seizure activity, agitation
- sedation for medical procedures (cardioversion, fracture reduction)
- delirium tremens

Contraindications

- hypersensitivity, glaucoma
- coma, shock
- substance abuse, head injury

Adverse Reactions / Side Effects

- respiratory depression, hypotension, drowsiness, ataxia
- reflex tachycardia, nausea, confusion, thrombosis, and phlebitis

Interactions

- incompatible with most drugs, fluids

Dosage and Administration

- seizure activity: adult: 1.5-5mg IV q 3-5 minutes prn
- maximum dose - 30 mg
- seizure activity: pediatric: 0.2 mg/kg / dose every 15-30 minutes, no faster than 3 mg over 5 minutes
- maximum dose - 5 mg/kg
- sedation for cardioversion: 5-15 mg IV over 5-10 minutes prior to cardioversion

Duration of Action

- onset: 1-5 minutes
- peak effect: minutes
- duration: 20-50 minutes

Special Considerations

- short duration of anticonvulsant effect
- pregnancy safety: category D
- reduce dose 50% in elderly patient

GLUCOSE

Class

- hyperglycemic

Mechanism of Action

- provides quickly absorbed glucose to increase blood glucose levels

Indications

- conscious patients with suspected hypoglycemia

Contraindications

- decreased level of consciousness
- absent gag reflex
- nausea, vomiting

Adverse Reactions / Side Effects

- nausea, vomiting

Interactions

- none

Dosage and Administration

- orally, sublingually

Duration of Action

- onset: immediate
- peak effect: variable
- duration: variable

GLUCAGON

Class

- hyperglycemic agent, pancreatic hormone, insulin antagonist

Mechanism of Action

- increases blood glucose by stimulating glycogenesis
- unknown mechanism of stabilizing cardiac rhythm in beta- or calcium-channel-blocker overdose
- minimal positive inotrope and chronotrope
- decreases GI motility and secretions

Indications

- altered level of consciousness when hypoglycemia is suspected
- may be used as inotropic or chronotropic agent in beta- or calcium-channel-blocker overdose

Contraindications

- hyperglycemia
- hypersensitivity

Adverse Reactions / Side Effects

- nausea, vomiting
- tachycardia, hypertension

Interactions

- incompatible in solution with most other substances
- no significant drug interactions with other emergency medications

Dosage and Administration

- adult: 0.5-1 mg IM, SC or slow IV; may be repeated q20 minutes PRN
- pediatric: 0.03-0.1 mg/kg / dose (not to exceed 1 mg); may be repeated q20 minutes IM, IO, SC, IV

Duration of Action

- onset: 1 minute
- peak effect: 30 minutes
- duration: variable (generally 9-17 minutes)

Special Considerations

- pregnancy safety: category C
- ineffective if glycogen stores depleted
- should always be used in conjunction with 50% dextrose whenever possible
- if patient does not respond to second dose of glucagon, 50% dextrose must be administered

INSULIN

Class

- Protein

Mechanism of Action

- Insulin promotes fat storage, increases glycogen synthesis, stimulates protein synthesis (tissue building), decreases glycogenolysis, and decreases glucose levels by increasing glucose transport into the cells

Indications

- Type I Insulin Dependant Diabetes Mellitus

Contraindications

- Hypoglycemia

Adverse Reactions / Side Effects

- redness, swelling & itching at injection site
- edema and refraction anomalies may occur

Dosage and Administration

- Dosage is determined by the Physician in accordance with the needs of the patient
- Administered by injection

Duration of Action

- onset: 0.5 - 2.5 hours (variable)
- peak effect: 2.5 - 24 hours (variable)
- duration: 8 - 24 hours (variable)

Special Considerations

- stress or illness may increase insulin requirements
- hypoglycemia may occur if patients overdose, exercises more then usual, or skips a meal
- diabetic coma may develop if patient takes less insulin then needed

LORAZEPAM

Class

- benzodiazepine, sedative, anticonvulsant

Mechanism of Action

- anxiolytic, anticonvulsant and sedative effects
- suppresses propagation of seizure activity produced by foci in cortex, thalamus and limbic areas

Indications

- patient who has generalized seizure lasting longer than 5 minutes
- initial control of status epilepticus or severe recurrent seizures
- severe anxiety
- sedation

Contraindications

- hypersensitivity
- focal seizure with no alteration in consciousness
- acute narrow-angle glaucoma
- coma, shock
- suspected drug abuse

Adverse Reactions / Side Effects

- respiratory depression, apnea, drowsiness, sedation, ataxia, psychomotor impairment
- restlessness, delirium, confusion
- hypotension, bradycardia

Interactions

- may precipitate CNS depression if patient is already taking CNS depressant medications

Dosage and Administration

- adult and adolescent (10-15 years of age)
 - 2 mg intrabucally (initial dose)
 - repeat q 10-15 minutes
 - maximum dose: 4 mg
- pediatric: (0-9 years) not indicated

Note: hold lorazepam if seizure stops, maximum dose is reached, or there is evidence of respiratory depression.

Duration of Action

- onset: 1-5 minutes
- peak effect: variable
- duration: 6-8 hours

NALOXONE

Class

- narcotic antagonist

Mechanism of Action

- competitive inhibition at narcotic receptor sites
- reverse respiratory depression secondary to depressant drugs
- completely inhibits the effect of morphine

Indications

- opiate overdose or decreased level of consciousness due to opiate use
- complete or partial reversal of CNS and respiratory depression induced by opioids
 - narcotic agonist
 - morphine, heroin, hydromorphone (dilaudid), methadone, meperidine (demerol)
 - fentanyl (sublimase), oxycodone (percodan), codeine, propoxyphene (darvon)
 - narcotic agonist-antagonist
 - butorphanol (stadol), pentazocine (talwin), nalbuphine (nubain)
- coma of unknown origin

Contraindications

- use with caution in narcotic-dependent patients
- use with caution in neonates of narcotic-dependent mothers

Adverse Reactions / Side Effects

- withdrawal symptoms in the addicted patient
- tachycardia, hypertension, dysrhythmias, nausea, vomiting, diaphoresis

Interactions

- incompatible with bisulphite and alkaline solutions

Dosage and Administration

- adult: 0.4 IV, IM, SC, or ET (diluted)
 - minimum recommended dose - 0.4 mg
 - repeat at 3-5 minute intervals as needed
 - maximum dose 2 mg
- pediatric: 0.1 mg/kg / dose IV, IM, SC, ET (diluted)
 - maximum of 0.8 mg
 - if no response in 10 minutes, administer an additional 0.1 mg/kg dose

Duration of Action

- onset: within 2 minutes
- peak effect: variable duration: 30-60 minutes

OXYGEN

Class

- Gas

Mechanism of Action

- reverses hypoxemia

Indications

- suspected or confirmed hypoxemia
- ischemic chest pain
- respiratory insufficiency
- confirmed or suspected carbon monoxide poisoning
- all causes of decreased tissue oxygenation
- decreased level of consciousness

Contraindications

- None (use caution with COPD patients, and those suffering from hyperventilation)

Adverse Reactions / Side Effects

- prolonged use of non-humidified oxygen may cause drying of mucous membranes

Interactions

- In very rare occurrences, oxygen may increase the toxicity of certain uncommon herbicides if ingested by a patient.

Dosage and Administration

- determined by the underlying problem

Duration of Action

- onset: immediate
- peak effect: not applicable
- duration: less than 2 minutes

Special Considerations

- be familiar with litre flow and each type of delivery device used
- supports combustion

GLOSSARY OF TERMS

Aphagia: inability to eat

Glycosuria: Condition characterized by the presence of glucose in the urine.

Glycogenolysis: The chemical change of glycogen to glucose

Gluconeogenesis: the formation of glucose from non-carbohydrates such as protein or fat

Hemiparesis: weakness affecting one side of the body

Homeostasis: the state of equilibrium in the body with respect to various functions and to the chemical composition of the fluids and tissues; the process through which such bodily equilibrium is maintained.

Hyperreflexia: a condition in which the deep tendon reflexes are exaggerated

Polyuria: excessive excretion of urine

Polydipsia: excessive thirst

Polyphagia: excessive hunger

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