EMS “Live” In-Station Continuing Education

CE Provider:	University of Texas Southwestern Medical Center at Dallas Department of Emergency Medicine Division of Emergency Medical Services
Course Title:	Diabetic Emergencies and Bleeding Control
Course Approval Number:	UTSW – 8M2016 TDH – 101606
Date:	August 2016
National Registry Content Area and Hours:	2.0 hours: Mandatory: Trauma 2.0 hours: Flexible: Medical Emergencies
National Continued Competency Program (NCCP) Content Area and Hours:	0.5 Hours: National Component – Trauma: Tourniquets 3.5 Hours: Local or Individual Component
TDH Content Area and Hours:	1.0 hour: Preparatory 0.5 hour: Patient Assessment 1.5 hour: Trauma 1.0 hour: Medical
Skills Proficiency Verification:	Patient Assessment – Trauma Patient Assessment – Medical Hemorrhage Control
Class Location:	_____
Instructor Name:	_____
Student Name:	_____

In order to accrue the CE hours required for recertification, the student must attend and participate in the live CE component represented by this module and complete any required skill demonstrations.

STUDENT VERSION

This form shall serve as a written record of the participant's successful completion of the EMS educational activity as outlined in the Texas Administrative Code, Title 25, Part 1, Chapter 157, Subchapter C and as outlined in CECBEMS Standards and Requirements for Organization Accreditation.

Diabetic Emergencies and Bleeding Control

Cognitive Objectives: Upon successful completion of this course, the student will be able to:

- Describe the normal relation between insulin, glucagon, and glucose levels in the blood.
- Describe the pathophysiology of diabetes mellitus.
- Correlate abnormal findings in assessment with clinical significance in the patient with a diabetic emergency.
- Discuss the management of diabetic emergencies
- List the indications and contraindication for the use of a tourniquet
- Describe the procedure for applying a tourniquet
- Describe complications associated with the use of a tourniquet
- Differentiate between the types of hemostatic agents
- Describe how the application of a hemostatic agent before your arrival will affect your care.

Psychomotor Objectives:

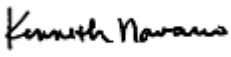
- No psychomotor objectives listed for this CE module

Affective Objectives:

- No affective objectives listed for this CE module

This continuing education activity is approved by the University of Texas Southwestern Medical Center at Dallas, an organization accredited by the Continuing Education Coordinating Board for Emergency Medical Services (CECBEMS).

You have participated in a continuing education program that has received CECBEMS approval for continuing education credit. If you have any comments regarding the quality of this program and/or your satisfaction with it, please contact CECBEMS at: CECBEMS -5111 Mill Run Road -Dallas, Texas 75244 - (972) 387-2862 lsibley@cecbems.com. CECBEMS is an organization established to develop and implement policies to standardize the review and approval of EMS continuing education activities. The cosponsoring organizations of CECBEMS are the National Association of Emergency Medical Technicians, the American College of Emergency Physicians, the National Association of Emergency Medical Services Physicians, the National Association of State Emergency Medical Services Directors, the National Council of State Emergency Medical Services Training Coordinators, and the National Registry of Emergency Medical Technicians, and the National Association of EMS Educators.



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For ECG practice or review, visit **www.ecglibrary.com**. To access the latest version of the protocols, for patient care alerts or updates on new medications, visit **www.biotel.ws**. References and bibliographies for all CE modules are on file and available upon request. To comment on this module, good or bad, e-mail **kenneth.navarro@utsouthwestern.edu**

Diabetic Emergencies and Bleeding Control

Diabetes mellitus is a chronic disease in which the body has trouble metabolizing glucose. Diabetes results from little or no insulin production in the body or may develop when the muscle, fat, or liver cells respond poorly to the available insulin. A less common form, diabetes insipidus is caused by the lack of a certain hormone which regulates the production of urine. This CE module will concentrate on the more common diabetes mellitus.

At the turn of the last century, a 10-year-old child diagnosed with diabetes could not reasonably expect to live long enough to celebrate his 12th birthday (MacCracken & Hoel, 1997). Diabetes care has significantly improved since then, but is still the seventh leading cause of death in the United States (American Diabetes Association, 2016). Diabetes is most commonly found in adults 45 years or older, people who are overweight or physically inactive, persons with a family history of diabetes, and people of minority ethnicity. Diabetes is also slightly more common in men than women (Centers for Disease Control and Prevention, 2015).

By 2050, estimates are that one-fifth to one-third of the United States population will suffer from diabetes (Boyle, Thompson, Gregg, Barker, & Williamson, 2010).

Anatomy and Physiology

Since diabetes is a disease that affects the body's ability to use glucose, let's start by examining glucose and how it is controlled by the body. Glucose is a simple sugar which provides energy to most cells of the body. As you eat, complex carbohydrates are broken down into the simple sugars, which are absorbed across the small intestine and distributed by the bloodstream throughout the body. Individual cells require a constant supply of glucose in order to perform their cellular functions.

The body tries to insure a constant supply of glucose by storing it as glycogen when it is plentiful and releasing it when fresh glucose is in short supply. Without this storage and releasing mechanism, the cells would be bathed in glucose as you eat, but starve in between meals. To maintain a steady supply of glucose, the body relies on two hormones that work in direct opposition to each other. These two hormones, insulin and glucagon, are produced by special cells located within the pancreas.

Insulin is made and secreted by the beta cells of the Islets of Langerhans within the pancreas. Insulin stockpiles nutrients right after a meal by allowing cells to absorb glucose, thereby reducing its concentration in the bloodstream. Glucagon is made and secreted by the alpha cells of the islets. Glucagon has the opposite effects of insulin by stimulating the liver and muscles to break down the

stored nutrients (glycogen) to release the glucose.

Normally, the levels of insulin and glucagon are counterbalanced in the bloodstream. For example, as you eat, the presence of glucose in the intestines stimulates the beta cells to release insulin into the blood and inhibits the alpha cells from secreting glucagon. Insulin levels in the blood rise which causes cells, particularly liver, fat, and muscle cells to absorb the incoming molecules of glucose. This action prevents the blood-glucose level from substantially increasing within the bloodstream. In this way, your body maintains a steady blood-glucose concentration.

In contrast, when you are between meals or sleeping, your cells do not have access to a fresh supply of glucose. During these times, slight drops in blood-sugar levels stimulate glucagon secretion from the alpha cells and inhibit insulin secretion from the beta cells. Rising glucagon levels in the blood stimulates the liver to release glycogen, which is a form of stored glucose. Glycogen is converted into glucose which is used as an energy source by the cells of the body. This action prevents the blood-glucose concentration from falling drastically.

This delicate balance between insulin and glucagon secretion (and a number of other hormones) throughout the day helps to keep your blood-glucose concentration relatively constant. For an individual who does not have diabetes, a normal blood glucose level should range from under 100 mg/dL when the individual first awakens to less than 140 mg/dL about two hours after finishing a meal (American Diabetes Association, 2011).

Pathophysiology

In diabetes mellitus, the patient's body is unable to manufacture insulin or stops responding to the insulin being produced. Without insulin, the cells of the body cannot absorb glucose from the blood regardless of how high the glucose levels rise. Without glucose, the cells begin to starve.

The starving cells force alpha cells within the pancreas to secrete glucagon, which stimulates the liver and muscles to breakdown stored glycogen and release glucose into your bloodstream. However, since the cells cannot absorb glucose without insulin, the glucose levels in the blood rise even further.

As the glucose-rich blood passes through the kidneys, some of the glucose spills over into the urine. When this happens, more water and electrolytes follow the glucose out of the bloodstream and into the urine. Since the body is now making more urine than it normally does, frequent urination occurs. This causes the patient to experience an increase in thirst as he or she tries to replace the fluid being lost.

The glucose levels in the urine pulls a tremendous amount of fluid out of the bloodstream and into the urine. The blood becomes thicker, with a consistency more like molasses. It becomes

harder and harder for the heart and blood vessels to pump around the thick blood and circulation becomes impaired. The patient may complain that their hands and feet feel cold. Circulatory changes within the capillaries of the eyes begin to impact vision which, long-term, results in blindness. Poor circulation also causes numbness and pain or a burning sensation in the hands and feet, a condition called neuropathy. The extremities become prone to slow-healing wounds and frequent infections which, ultimately can lead to gangrene in the limbs with subsequent amputations.

Up to this point, the starving cells have been unable to get the glucose they need for survival. From their point of view, there does not seem to be any glucose available in the body. The brain responds by stimulating the hunger centers in hopes the patient will eat and deliver glucose. At the same time, however, the lack of insulin stimulates the breakdown of fats in fat cells and proteins in muscle, leading to weight loss. Despite the fact that diabetics eat more frequently, they often lose weight. The patient begins to feel tired and fatigues easily because the cells have nothing to burn for energy.

Early in diabetes, circulation through the capillaries is impaired. Later, larger blood vessels are affected which leads to many cardiovascular problems, including high blood pressure, heart attack, and stroke. Although these conditions also occur in nondiabetic individuals, people with diabetes are two to four times more likely to develop cardiovascular disorders or stroke (American Heart Association, 2015). One in five patients with type 2 diabetes may suddenly develop abnormal myocardial perfusion without experiencing any symptoms of pain or discomfort, a condition known as silent myocardial ischemia (Kawano et al., 2016). This is thought to be caused by worsening nerve damage to the sensory nerves of the heart caused by diabetes (Chico, Tomás, & Novials, 2005).

Kidney failure, often called end stage renal disease (ESRD), is also a frequent long-term and life threatening complication in diabetes (Narres et al., 2016). Diabetes causes the kidneys to work harder when filtering the blood. After years of this increased work, the membranes that filter the blood begin to leak small amounts of protein into the urine, a condition called microalbuminuria. Large protein molecules passing through the tubules in the nephron (filtering subunit of the kidney) create inflammation and scarring of the tubules with eventual loss of function. If this condition is recognized early and with proper management of the diabetes, treatment can prevent the kidney damage from getting worse. However, if the condition is not well managed, ESRD occurs. When ESRD reaches a certain point, the patient will require dialysis while awaiting a kidney transplant. In fact, almost half of all patients placed on renal dialysis developed end-stage renal disease because of diabetes (National Institutes of Health [NIH], National Institute of Diabetes and Digestive and Kidney Diseases [NIDDKD], 2013).

Type-I Diabetes

There are actually two primary types of diabetes mellitus. The less common form is called Type-I diabetes. It is also known as insulin-dependent diabetes. This condition was once termed juvenile-onset diabetes because it frequently occurs in children and usually begins at puberty. However, this term is no longer used to describe the disease. In Type-I diabetes, the body has stopped producing insulin, altogether.

Type-I diabetes is often considered an auto-immune disease. For some unexplained reason, the immune system begins to attack and eventually destroys the insulin producing cells of the pancreas. It is believed that a combination of genetic and environmental factors trigger this attack.

Type-I diabetics require the administration of insulin several times a day in order to replace the insulin they are not producing. The exact amount of insulin required is highly individualized and may be influenced by levels of physical activity, diet, and general health. Type-I diabetics generally monitor their own glucose levels with small hand-held meters obtained from their doctor. In this way, they can adjust the amount of insulin or food intake to keep their blood glucose levels near a normal level.

Type II Diabetes

In Type-II diabetes, formally known as adult onset diabetes, the body is still producing insulin but it may not be producing enough or the cells are ignoring the insulin altogether. The American Diabetes Association (2016)) reports that about 96 percent of diabetics are Type-II diabetics. In this disease, the insulin resistance is probably linked to obesity, especially in individuals who primarily store fat in the viscera instead of the subcutaneous tissues (Hardy, Czech, & Corvera, 2012). The exact link is still unknown.

For the type-II diabetic, the disease may initially be controlled with special diets, exercise and weight reduction. As the disease progresses, these measures may not be enough to prevent abnormal elevations of glucose in the blood. At that point, the patient may be prescribed an oral anti-hyperglycemic agent. If this fails, the patient may need supplemental insulin injections or a combination of insulin and oral medications.

Gestational Diabetes

Another type of diabetes can occur in some pregnant women and is known as gestational diabetes. It is similar to Type-II diabetes and occurs in about 2 to 5% of all pregnancies. During pregnancy, several hormones partially block the actions of insulin, thereby making the woman less sensitive to her own insulin. The condition typically develops around or the 24th week of pregnancy, and can usually be managed by special diets and/or supplemental injections of insulin. It usually

goes away after the baby is delivered. In some studies, however, nearly 40% of women with a history of gestational diabetes developed diabetes later in life.

Most women with gestation diabetes deliver healthy babies. However, complications related to the disease can arise in some patients. In many cases, the disease will produce a condition called macrosomia, in which the baby's body is larger than normal. This increases the risk of injury during vaginal deliveries and thus may require delivery by cesarean section. The most common complication of macrosomia is shoulder dystocia, a condition that prevents the baby's anterior shoulder from passing below the mother's pubic symphysis.

Diabetic Emergencies

Diabetic emergencies occur in one of two ways; either the blood glucose value falls to dangerous levels or rises to extremes.

Diabetic Ketoacidosis

Hyperglycemia is a condition when the blood glucose values rise above normal. If the hyperglycemic episode is prolonged and complicated by profound dehydration and excess acid production, a condition known as diabetic ketoacidosis (DKA) may develop. The most common causes are underlying infections, disruption of insulin treatment, and new onset of diabetes. Diabetic adolescents are especially susceptible to hyperglycemia, since hormonal levels are in flux and many adolescents exhibit erratic eating and sleeping patterns. DKA typically is characterized by blood glucose values over 300 mg/dL, although the condition can still be present at lower blood glucose levels.

Before the discovery of insulin in 1922, the mortality rate from DKA was 100%. With modern management strategies, the overall mortality rate has decreased to less than 1% (Umpierrez & Korytkowski, 2016). A rare but devastating complication of DKA in the pediatric patient is cerebral edema (Watts & Edge, 2014). This condition usually begins with 4 to 24 hours after initiating treatment. The exact mechanism that produces the cerebral edema remains unknown, but loss of autoregulatory functions of the brain that permits abnormal diffusion of intravascular fluids into the brain tissue (Jeha & Haymond, 2015). DKA complicated by cerebral edema has a mortality rate as high as 24% and those who do survive frequently have permanent neuroglial deficits (Watts & Edge, 2014).

Since patients in DKA can no longer get energy from carbohydrates and glucose, the body begins to burn fat. Fat metabolism leads to increased formation of chemicals called ketone bodies, which build in the bloodstream. Ketone bodies can poison and even kill body cells. The urinary

system attempts to remove some of the ketones by increasing urination, which can lead to profound hypovolemia. One type of ketone, called acetone, can be expelled through the lungs. This gives the breath the characteristic fruity odor associated with hyperglycemia. Ketones that build in the body for a long time lead to serious illness and coma.

The first signs of developing DKA are frequent urination and increased thirst. The patient may then show a variety of symptoms, including flushed face, dry skin, dry mouth, headache, nausea, vomiting, abdominal pain, drowsiness and lethargy, blurry vision, and tachycardia. DKA can lead to irregularities in the heart, altered mental status, and breathing problems, such as Kussmaul's respirations, as the body tries to rid itself of the excess acid. Without treatment, the patient will lapse into a diabetic coma and die.

Hypoglycemia

When the body's blood glucose value is abnormally low, hypoglycemia is said to be present. Many people believe if hypoglycemia is present, the patient must have diabetes. This is simply not the case. Many medical conditions can cause hypoglycemia and diabetes is only one of them.

Among the underlying causes of hypoglycemia are the uses of certain medications such as oral anti-hypoglycemia agents, alcohol abuse, sepsis, certain cancers, critical illnesses including kidney, liver, or heart failure, hormonal deficiencies, and disorders that result in the body producing too much insulin. The most common cause of hypoglycemia in the Type I diabetic patient is an over administration of insulin.

The correct balance between the right amount of glucose and the right amount of insulin in the body is very difficult to achieve sometimes. In any given two-day period, a patient could eat two identical meals and self-administer two identical insulin injections but still develop a life-threatening episode of hypoglycemia. Since the organ most sensitive to blood glucose levels is the brain, mental status changes are one of the first indicators of a problem. These changes can be very subtle, for example, the patient may become anxious or lightheaded. As the body attempts to compensate for the lowered blood glucose levels, adrenalin is released into the bloodstream. As a result, the patient may experience nausea or vomiting, diaphoresis, and tachycardia. The patient may also develop muscle tremor or weakness. The blood pressure remains normal or slightly elevated and the pupils are dilated, but reactive to light.

As the hypoglycemia progresses, alterations in brain function become more and more pronounced. The patient may become sleepy or extremely slow to react to stimuli. Some patients may exhibit psychotic or combative behavior. Still others will develop signs similar to a stroke, such as paresthesia or hemiplegia. If left uncorrected, seizures may develop followed by coma.

In some situations, the development of hypoglycemia is much slower. In those patients,

adrenalin is not released and the patient's energy levels decrease gradually like a child's toy when the batteries are becoming weak. The patient will become less and less active, feel very sleepy, and drift into a coma. Once the hypoglycemia has progressed to that state, it may be difficult to arouse the patient.

Patient Assessment

Before patient assessment begins, a thorough scene evaluation must take place. You must identify any conditions that could represent a hazard to you or your partner. Review the need for body substance isolation procedures. Many hypoglycemia patients will be disoriented and confused to the point of being dangerous to approach alone.

Once you access the patient, rapidly assess their airway, breathing, and circulation. In most cases, simple non-invasive airway maneuvers such as proper positioning will often suffice. If positioning alone fails to correct the problem, insert a nasopharyngeal airway.

Place the patient on a pulse oximeter to establish baseline oxygen saturation. In most cases, saturation values will be normal. However, if the patient has lost consciousness and is not breathing well, saturation will be lower than normal. In those cases, administer high-flow oxygen. The oxygen saturation of both patients will usually be within normal limits, although the hypoglycemic patient may have low saturations if unconscious with slow respirations.

Pay particular attention to the patient's breathing. An excessively deep, rapid breathing pattern called Kussmaul's respirations can occur in DKA as the body attempts to compensate for the acidosis by blowing off excessive carbon dioxide. In an ED evaluation of patients with a point-of-care blood glucose reading greater than 550 mg/dL, DKA was never present when the patient had an $ETCO_2$ greater than or equal to 35 mmHg (Bou Chebl, Madden, Belsky, Harmouche, & Yessayan, 2016). On the other hand, DKA was always present when the patient had an $ETCO_2$ less than or equal to 21 mmHg. Kussmaul's respirations are often associated with a fruity breath odor that occurs as cells burn fat and proteins for fuel.

Once you have addressed the ABCs, examine the patient's level of consciousness using the Alert, Pain, Verbal, Unresponsive (AVPU) classification system - an important step in establishing a baseline mental status. The mental status changes found in DKA are usually more subtle than those found in hypoglycemia. The DKA patient will present with drowsiness or lethargy progressing to coma over time. The hypoglycemic patient will be anxious, lightheaded, sleepy or extremely slow to react, psychotic or combative, and may progress to seizures and coma.

After the initial survey, obtain a full set of vital signs. Vital signs in the DKA will usually reveal tachycardia, a weak and thready pulse, and Kussmaul's respirations with a fruity odor on the breath.

The hypoglycemic patient will present with tachycardia, but the respiratory changes seen in DKA will be absent.

History

Whenever possible, the best source of medical history information is the patients themselves. If the patient cannot provide medical information, you must obtain a history from family members or bystanders, or use other clues.

Look for a Medic Alert tag or wallet card that can confirm a medical history. If you are at a patient's home, gather the medications. Diabetics will store their insulin in the refrigerator and will probably keep their syringes near where they sleep. Common insulins are regular insulin, NPH insulin, Lente, Ultralente, Humalog, and Lantus.

Patient can self-administer insulin in a variety of way, including through the use of a syringe, a pen, or an inhaled dry spray. In some cases, the patient may have an external insulin pump (Figure 1). This is a small device that is usually attached to the body via a needle or catheter inserted into the subcutaneous tissues of the abdomen. The device has a refillable cartridge that stores the insulin and continuously releases tiny amounts throughout the day, thus mimicking normal pancreatic function. The devices also have the ability to release larger boluses when activated by the user. This is helpful in situations where the blood glucose levels are very high.

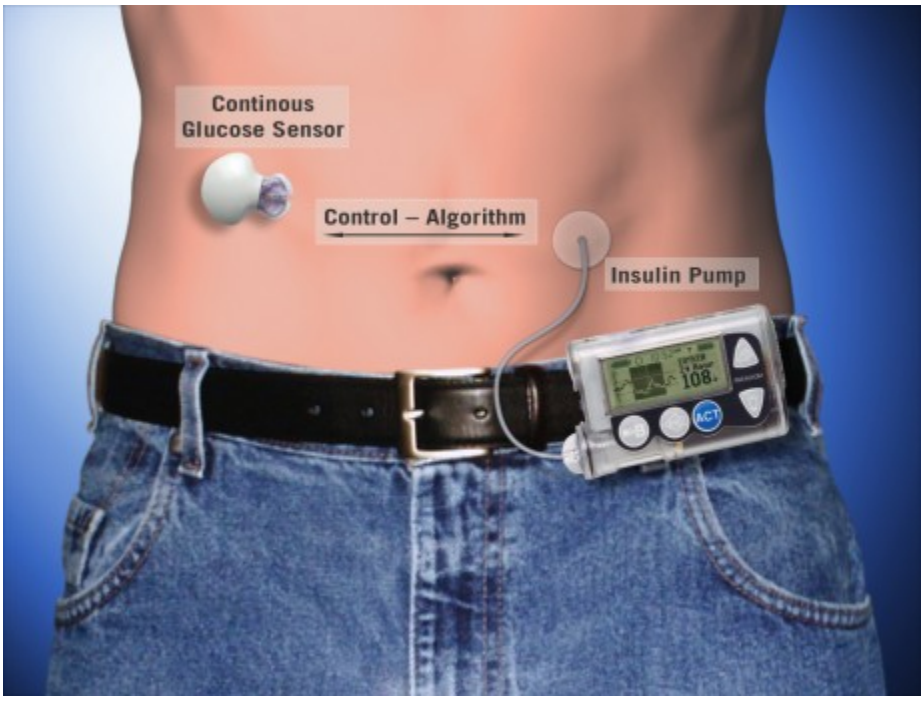

Figure 1: Insulin pump. Image retrieved from http://images.dailytech.com/nimage/21235_large_Insulin-Pump.jpg

The pump is typically worn around the patient's waistband but may also be stored in clothing pockets, undergarments or socks. Patients may temporarily disconnect the device when bathing or showering.

In most cases, it is not necessary for the paramedic to disconnect the device from the patient. If the patient is hypoglycemic, treat accordingly and leave the device in place. If the patient is unconscious and IV access is not easily obtainable, it may be appropriate to disconnect the device so the hypoglycemic patient will not continue to receive insulin. If you are unsure, contact BioTel for a consultation. Disconnecting the device is relatively simple; however, you may want to have family members assist you as they may be familiar with the operation of the device. Because most pumps use short acting insulin, the patient will need to be reconnected to the pump within a couple of hours. These devices are expensive, so make sure you document bringing the device to the hospital and who took possession of the device once you arrive at the hospital.

In addition to insulin, there are six different classes of oral medications used to treat diabetes. Complications may be specific to each class and include an increased risk for hypoglycemia, lactic acidosis, and an increased risk of heart failure.

Ask about the events that preceded the 911 call. If the patient has Type I diabetes, ask whether they have eaten and about the last time they took their insulin. Ask about recent illnesses, changes in activity or stress levels, recent changes in their insulin dose, and other health problems. DKA usually has a history of recent illness or fever, a reduction in daily activity levels, missing or taking inadequate insulin doses, eating increased amounts of food, having increased thirst (polydipsia), and/or excessive urination (polyuria). The history of the hypoglycemic patient will reveal increases in activity or stress, taking excess insulin or oral glucose control medicines, a recent change in medication doses, or missing meals.

When the central nervous system recognizes a critical drop in circulating blood glucose levels, it causes a release of epinephrine, also known as adrenalin. For this reason, hypoglycemia patients tend to present with the “fight-or-flight” stress response symptoms. Physical findings in the DKA patient generally reflect significant dehydration.

DKA usually develops over a period of a few days. Hypoglycemia may develop rapidly or slowly with the patient becoming less and less responsive.

Secondary Assessment

The skin of the DKA patient will be warm, flushed, and dry. The mucous membranes will also be dry. The hypoglycemic patient on the other hand will be pale, with clammy and diaphoretic skin. The hypoglycemic patients may also have muscle tremor, weakness, paresthesia, or hemiplegia.

Both hypoglycemia and DKA can present with nausea with vomiting. Paramedics should always be concerned about diabetic patients who vomit, regardless of the blood glucose value. Patients with DKA, especially children, may also have abdominal pain, which is not usually seen in hypoglycemia. Abdominal pain is usually associated with metabolic decompensation and may be

present in up to 75% of patients with DKA (Umpierrez & Freire, 2002).

The blood-glucose analysis of the DKA patient will likely be over 300 mg/dL. The reading in the hypoglycemic patient will be less than 110 mg/dL and in many cases, lower than 80 mg/dL.

Blood Glucose Monitoring

One very common assessment step in a patient suspected of having a diabetic emergency is to determine the patient's blood glucose level, often by using a handheld monitor. It is important to note that handheld monitors can be divided into two distinct categories (Lias, 2014). Over-the counter monitors are designed for single patient use. These monitors allow a patient suffering from diabetes to monitor their own glucose state and adjust their self-administered medication based on those results. Point-of-care (POC) monitors are used in a professional setting such as an emergency department or EMS agency. POC monitors are designed for use on many patients.

Although a number of studies demonstrate acceptable accuracy for handheld blood glucose monitors under controlled conditions (Arabadjief & Nichols, 2006; Cohen, Boyle, Delaney, & Shaw, 2006; Kuo, Hsu, Ho, Su, Wu, & Wang, 2011), accuracy is often suboptimal during actual clinical situations, which could have a significant impact on therapy (Boyd & Bruns, 2001; Budiman, Samant, & Resch, 2013; Francescato, Geat, Stel, & Cauci, 2012; Hellman, 2012; Henry, Major, & Reinsch, 2001; Kanji et al., 2005; Petersen et al., 2008; Trajanoski, Brunner, Gfrerer, Wach, & Pieber, 1996; Virdi & Mahoney, 2012). An Australian study demonstrates that meters tend to overestimate glucose levels when compared to reference values, with one common meter averaging about 25 mg/dl higher than the reference device (Cohen, Boyle, Delaney, & Shaw, 2006). In an investigation involving over 18,000 patients, Canadian researchers found glucose measurement differences greater than 90 mg/dl in one in 200 meters designed for home use (Naugler, Zhang, & Redman, 2014). This degree of measurement error could result in patient self-administration of higher than necessary insulin doses, which could lead to hypoglycemic episodes. In fact, about 40% of patients who use older meters may over administer insulin (Boyd & Bruns, 2001). If all patients suffering from Type 1 diabetes used the least accurate meter, the error would result in about 300,000 episodes of hypoglycemia per year (Budiman, Samant, & Resch, 2013).

For critically ill patients, POC glucose testing may overestimate glucose levels, especially in patients receiving blood pressure support through the use of vasopressors (Critchell et al., 2007), in patients with diabetic ketoacidosis or hyperglycemic hyperosmolar syndrome (Corl et al., 2013). Capillary samples taken from patients with severe edema may underestimate by as much as 46% of reference value (Petersen et al., 2008). In these cases, the sample may be more representative of tissue glucose levels rather than capillary levels.

The margin of error may be increased by poor or missing calibration, temperatures outside the

intended range, outdated strips, improper technique, poor timing, insufficient sample size, and contamination. Contamination is especially serious since it can happen so easily and is likely to result in episodes of hypoglycemia going unrecognized and untreated. Even a very tiny amount of glucose contamination can seriously alter a reading.

By taking a few precautions, EMS personnel may increase the accuracy of the glucose values. First, always use test strips recommended by the glucometer manufacturer. It is unclear whether glucose readings are accurate if obtained with generic test strips. In side-by-side comparisons against a known sample, researchers have demonstrated that different test strips will give noticeable variations in blood glucose values (Lenhard, DeCherney, Maser, Patten, & Kubik, 1995).

Next, glucometers are calibrated for capillary samples, NOT venous samples. If venous samples are used directly from the IV site, for instance, you may get a reading; however, the accuracy of the value is questionable. In healthy volunteers, a poor correlation has been demonstrated between glucometer values using venous samples and values obtained from capillary blood. For the most accurate reading possible, you should obtain your blood sample from a fingerstick source.

Next, after swabbing the sample finger with alcohol, you must wipe the area dry with a sterile gauze pad. This allows any residual alcohol to be removed and avoids contamination of the sample blood. For added accuracy, the first drop of blood available from the fingerstick should also be wiped with the gauze pad. This ensures that the second blood drop is the most accurate and least contaminated sample that can be taken.

Finally, most glucometers are not calibrated for use in neonates, who are generally defined as babies in the first 28 days of their life. If blood glucose analysis is performed in neonates, the accuracy is questionable. If those babies show signs of hypoglycemia, dextrose should be administered even if the glucometer indicates normal blood glucose levels. Contact BioTel for consultation in those situations.

The United States Food and Drug Administration has recently recommended implementation of two separate sets of standards for glucose meters, one for over-the-counter meters and one form POC meters (American Association for Clinical Chemistry, 2014). These new standards will likely have a greater impact on the POC meters and improve the accuracy of glucose measurement in the prehospital environment.

Words of Wisdom from the Medical Directors: Diabetic Ketoacidosis

Dr. Salazar: Any patient with a blood glucose of 250mg/dL or higher, especially if symptomatic, should be considered to have DKA until proven otherwise. These patients need an IV and IV fluids. Medics often tell me that patients in DKA always have extremely high glucose levels. That is incorrect. Always look for a trigger. One useful mnemonic is the Four Is:

- Infection
- Insulin non-compliance
- Ischemia (stroke)
- Intrauterine pregnancy

Dr. Isaacs: Any patient refusing transport with a POC glucose greater than 250mg/dL or who was hypoglycemic, altered and received either oral glucose, D50 or glucagon really should be signing "AMA" and have all the appropriate documentation that goes along with an AMA refusal. Glucagon should NOT be administered unless the patient is hypoglycemic, altered, and reasonable attempts to obtain IV access have been exhausted.

Management

Hypoglycemia

Caring for hypoglycemia patients requires greater urgency than for hyperglycemic DKA patients because the body and especially the brain depend heavily on glucose. The management goal for hypoglycemia is fairly obvious. You must raise the level of glucose in the circulating blood before permanent brain damage can occur.

Medics should start with oral glucose, if the hypoglycemia patient is still conscious and can provide airway self-protection. Oral glucose comes packaged in a tube containing 15 grams of glucose in gel form. Standard administration doses are 0.5 grams per kilogram. Most adults will show improvement with two or three tubes.

Place the gel under the tongue or in the pouch of the cheek. Encourage the patient to hold the gel in the mouth for as long as possible in order to maximize absorption. Take care to prevent the patient from aspirating the glucose and always have suction available.

If the patient is unconscious, treat the hypoglycemia with intravenous dextrose. Dextrose for the adult comes packed in 50 mL syringes containing 25 grams of dextrose. This D50 is virtually free of adverse effects but it is not without its hazards. D50 is a very concentrated form of sugar and if it infiltrates into the tissues from the IV site, extensive tissue necrosis is possible with widespread damage. Your IV line must be patent in a large, stable vein before administration of dextrose in any

concentration.

Although 50% dextrose has been used for decades in the treatment of hypoglycemia, it is beginning to fall out of favor for a number of reasons, including hyperglycemia overshoot as well as histamine release. Blood has a property known as osmolarity, which is the concentration of solutes dissolved in the plasma. Normal "concentration" of the blood, when measured in osmolarity, is about 300 milliosmoles per liter (mOsm/L). A vein can typically withstand concentrations of solutions up to around 900 mOsm/L before damage occurs to the vein. When the osmolarity of an administered solution exceeds 900 mOsm/L, inflammation of the vein (phlebitis) can occur. The osmolarity of 50% dextrose is about 2,525 mOsm/L (because of the high concentration of sugar), more than enough to irritate and disrupt the inner lining of blood vessels (Kuwahara, Asanami, & Kubo, 1998).

Reducing the concentration of infused dextrose will significantly reduce the likelihood of damage to the vein in which it is being infused. For example, a 10% dextrose solution has an osmolarity of around 506 mOsm/L, which is well within the range for safe peripheral administration (Kuwahara, Asanami, & Kubo, 1998). The next edition of the BioTel Treatment Guidelines will likely recommend a 10% dextrose solution for all patients instead of a 50% solution. Stay tuned.

Currently, however, the BioTel Treatment Guidelines recommend using a 50% dextrose concentration for dextrose 50% is usually reserved for adult patients only. The standard IV dose is 1 mL of D50 per kilogram of body weight. This allows for 500 mg of dextrose to be given per kilogram. A rapid return of consciousness is diagnostic of hypoglycemia. Carefully monitor the patient's level of consciousness. As a hypoglycemia patient regains consciousness, a phase of combativeness and mild confusion may follow and can result in movement that compromises the IV line.

According to the BioTel Treatment Guidelines, at what age can minors start receiving D50?
ANSWER:

The administration of D50 is not recommended for children. Instead, the D50 should be diluted to a concentration of 25% dextrose. This is easily accomplished by wasting half of the contents of a D50 syringe into the trash, then refilling the syringe with saline from the IV. This gives you a syringe of 25% dextrose.

According to the BioTel Treatment Guidelines, how much D25 should you administer to a child?
ANSWER:

For infants, D50 should be diluted to a D10 concentration. According to the BioTel Treatment Guidelines, what is the age range for infants to receive D10?
ANSWER:

D10 can be made by wasting 40 mL of the D50 and replacing it with 40 mL of saline.

According to the BioTel Treatment Guidelines, D10 can be administered to both infants and newborns (neonates)? What is the age range for a newborn (neonate)?
ANSWER:

According to the BioTel Treatment Guidelines, what is the correct dose for D10 administration for an infant and for a newborn?
ANSWER: NEWBORN (first month of life): **ANSWER: INFANT (after first month but before first year birthday)**:

If the patient has an altered mental status or is unconscious and IV access cannot be established, administer the hormone glucagon by subcutaneous or intramuscular injection. Glucagon stimulates the breakdown of glycogen in the liver to glucose which the body can use for energy production. Glucagon administration will temporarily raise the blood glucose levels of the body; however, the drug takes 10 to 20 minutes to take effect. A two milligram intranasal dose is as effective within the first thirty minutes as a one milligram intramuscular dose (Pontiroli, Calerara, Pajetta, Albertetto, & Pozza, 1989).

Glucagon is only effective in patients with a sufficient reserve of glycogen in the liver. If the glycogen reserves have been depleted, such as might be the case in chronic alcoholics or patients with liver disease, the effectiveness of glucagon will be compromised. In any case, the standard dose of glucagon for altered mental status hypoglycemia when no IV can be established is 1 mg IM, IN, or SubQ.

The Medical Directors are concerned about the number of patients found hypoglycemic in the field, given glucagon, and not brought to the hospital. Please remember that BioTel guidelines call for an IV attempt and the administration of IV dextrose in hypoglycemic patients who are not eligible for oral glucose administration. Glucagon is a second-line drug and should be used only if you cannot gain IV access. Here is why:

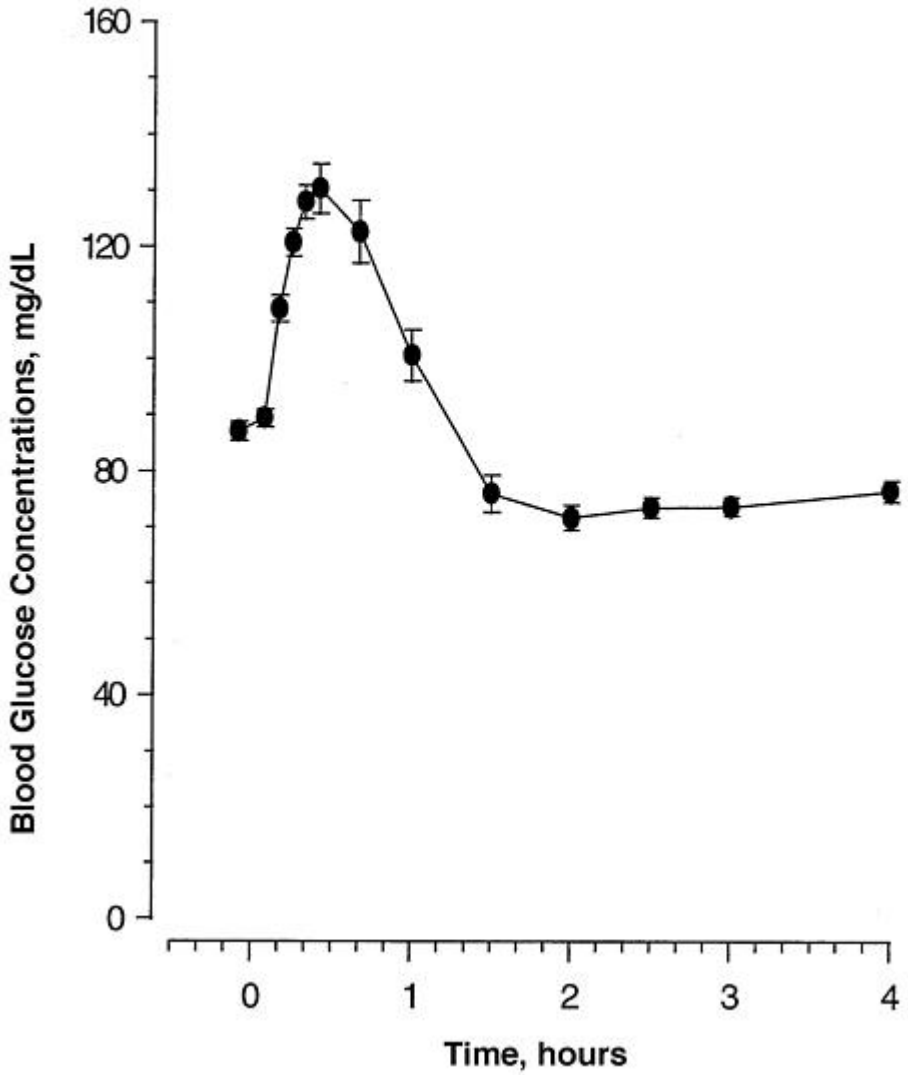

Figure 2 Eli Lilly and Company. (2009). Glucagon for injection. Retrieved from https://dailymed.nlm.nih.gov/dailymed/archives/fdaDrugInfo.cfm?archiveid=13362

In a study of 25 volunteers, a subcutaneous dose of 1 mg Glucagon resulted in a mean peak glucose concentration of 136 mg/dL 30 minutes after injection (Figure 2). Similarly, following intramuscular injection, the mean peak glucose level was 138 mg/dL, which occurred at 26 minutes after injection.

Notice that after the peak (at about 30 minutes), the blood glucose levels begin to rapidly decline. This is because the half-life of glucagon is about 30 minutes. In contrast, the half-life of intermediate acting insulin (Humulin N) is about 4.5 hours. Longer acting insulin has a half-life of about 7 hours. After the glucagon wears off, the insulin responsible for the episode of hypoglycemia will still be present and will remove the glucose from the blood stream, causing another episode of hypoglycemia.

Also notice that in just over an hour, the blood glucose levels are as low as they were before glucagon administration. And they continue to fall. At about two hours after glucagon administration,

blood glucose levels stabilize substantially lower than before the administration of glucagon.

The minimum standard for treatment of a patient with hypoglycemia is the documentation of both a "pre-" and "post-" treatment glucose check in the chart regardless of whether the patient is transported. Failure to do so is a violation of the guidelines.

Definitive treatment of ketoacidosis involves correcting dehydration, reversing the acidosis and ketosis, reducing plasma glucose concentration to normal, replenishing electrolyte and volume losses, and identifying the underlying cause. Most of this must occur under very close monitoring conditions generally not found in the prehospital environment.

The treatment of DKA in the field usually centers on fluid resuscitation. In addition to fluid replacement, IV administration dilutes both the glucose level in the blood and the counter regulatory hormones that are also present. Insulin will eventually have to be given, but that is too dangerous to be carried out under field conditions.

Start by giving the adult patient up to 1 liter of an isotonic saline solution, depending on the patient's vital signs and other indicators of hypovolemia. Further isotonic saline can be administered at a rate appropriate to maintain adequate blood pressure, pulse, and mental status.

Paramedics must be very careful when administering fluids to children suspected of having DKA. Although rare, life-threatening cerebral edema occurs early in the treatment of pediatric patients suffering from DKA. In about 20% of cases, cerebral edema is already occurring when the patient presents to the healthcare system (Lawrence, Cummings, Gaboury, & Daneman, 2005).

Summary

Diabetes is a significant health care problem in the United States and is the sixth leading cause of death. EMS personnel frequently encounter patients experiencing some type of diabetic event. Familiarity with the pathophysiology, signs, and symptoms can help the EMT differentiate the diabetic from other patients with similar presentations. Finally, proper field care may help to minimize the complication rate and duration of hospital stay for this deadly disease.

ALTERED LEVEL OF CONSCIOUSNESS

Inclusion criteria: Patients who are disoriented, weak, dizzy, confused, suffered a syncopal episode, or are unconscious. In these guidelines, hypoglycemia is defined as a POC glucose analysis of:
Adult: less than 80 mg/dL (non-diabetic), *OR* less than 110 mg/dL or symptomatic (diabetic);
Pediatric: less than 70 mg/dL (non-diabetic), *OR* less than 70 mg/dL or symptomatic (diabetic).
NOTE: Known diabetics may be symptomatic at a higher POC glucose level.

NOTE: Never administer dextrose to a patient who is NOT hypoglycemic. If the patient's level of consciousness is altered, and a POC glucose analysis is normal, search for alternative causes. Additional information is available at the bottom of these guidelines, on the next page. Refer to **NEONATAL** Guidelines for newborn care.

SPECIAL NOTE: The use of naloxone should be restricted to patients suspected of opioid narcotic overdose AND hypoventilation and/or hypoxia, AND pinpoint pupils. Its use outside of these indications may cause undesirable narcotic withdrawal.

Basic Level

1. Assess and support ABCs. If trauma is suspected, refer to the **SPINAL MOTION RESTRICTION** Policy to immobilize the spine & refer to the **TRAUMA** Guidelines.
2. If there is no evidence of trauma, place the patient in a position of comfort or in the left lateral position. If there is evidence of shock, place the patient supine with the feet elevated and closely monitor the airway.
3. Administer oxygen, as needed, to maintain SpO_2 of at least 94%.
4. If hyperthermia is suspected, monitor the patient's temperature frequently. Be prepared to cool the patient aggressively, but do not cause shivering.
5. Perform a POC glucose analysis.
   a. If the adult patient is hypoglycemic but responsive AND able to protect his or her airway, administer 1 tube (15 g) oral glucose SL. (Pediatric patient 1 to 13 years old: administer ¼ - ½ tube SL.)
   b. If symptoms persist after 10 minutes, administer a second tube (15 g) of oral glucose SL. (Pediatric patient 1 to 13 years old: administer ¼ - ½ tube SL.)

Advanced Level

6. Consider establishing IV access at a TKO rate or use a saline lock. If the patient is hypotensive, treat according to **SHOCK** Guidelines.
7. If the patient is hypoglycemic AND . . .

. . . the level of consciousness does not improve with oral glucose, or if oral glucose could not be given, administer:		
At least 14 years of age (or over 50 kg) • 50% dextrose, 50 mL (25 grams) IVP/IO. • If symptoms and/or hypoglycemia persist after 10 minutes, administer an additional 25 grams (50 mL).	**1 year to 13 years of age** • 25% dextrose 2 mL/kg IVP/IO (waste 25 mL of D50; replace with 25 mL Normal Saline). • If symptoms and/or hypoglycemia persist after 10 minutes, administer an additional 2 mL/kg IVP/IO.	**Less than 1 year of age** • 10% dextrose 5 mL/kg IVP/IO (waste 40 mL of D50; replace with 40 mL Normal Saline). • Contact BioTel. • Newborn under 1 month of age: administer only 2 mL/kg.

Continued on the next page...

. . . IV or IO access cannot be obtained, administer:		
At least 14 years of age • Glucagon 1 mg IM, IN or SQ. • May repeat once after 20 min.	**1 year to 13 years of age** • Glucagon 1 mg IM, IN, or SQ. • May repeat once after 20 min.	**Less than 1 year of age** • Glucagon 0.5 mg IM, IN, or SQ. • May repeat once after 20 min.

8. All patients treated under these guidelines must have continuous cardiac monitoring. If a dysrhythmia develops, treat accordingly under its specific guidelines. Patients with continued altered mentation should also have $ETCO_2$ monitoring.

9.

If there is evidence of opioid narcotic use, with altered mental status, hypoventilation and/or hypoxia, AND pinpoint pupils, administer:	
Adult • Naloxone 0.4 mg every 5 minutes via IN or SLOW IVP or IO until the respiratory rate improves and the patient can maintain a SpO_2 of at least 94%, OR until 2 mg have been given.	**Pediatric** • Naloxone 0.1 mg/kg via IN or SLOW IV Push or IO (maximum single dose 0.4 mg) until the respiratory rate improves and the patient can maintain a SpO_2 of at least 94%, OR until 2 mg have been given.
If unable to establish IV access or if IN administration is not possible, administer the naloxone IM.	

10. If the respiratory rate or oxygen saturation does not improve with a full naloxone dose, secure and monitor the patient's airway with an advanced airway, discontinue naloxone use, and proceed in the algorithm.

11.

If altered mental status with bradycardia is caused by beta-blocker toxicity, administer:	
Adult • Glucagon 1 mg – 5 mg IVP/IO over 2 to 5 min, OR 1 mg IM or IN. • May repeat once after 20 minutes.	**Pediatric** • Glucagon 0.5 mg (under age 1 yr) or 1 mg (at least one year of age) IV/IO, IM, or IN. • May repeat once after 20 minutes.

12.

If altered mental status with bradycardia is caused by calcium-channel blocker toxicity, administer:	
Adult • Calcium chloride, 10 – 15 mg/kg slow IVP/IO. (optional medication)	**Pediatric** • Contact BioTel for authorization and dosing (risk of phlebitis). (optional medication)

13. For patients with excited delirium, refer to **EXCITED DELIRIUM** Guidelines.

14. Monitor vital signs and attempt to transport.

15. For additional patient care considerations (e.g. other drug toxicities) not covered under standing orders, consult BioTel.

Notes:

- If the patient becomes alert and oriented after glucose/glucagon administration, do NOT administer naloxone.
- If the patient does not respond to glucose/glucagon & naloxone, consider other causes of altered LOC.
- Do not attempt to restore full consciousness in patients with evidence of narcotic use. Titrate naloxone administration to restore adequate ventilatory status, or to a SpO_2 of at least 94%.
- Transport any patient taking any medication combination that includes glipizide (Glucotrol®) or other sulfonylureas (Dymelor® [acetohexamide], Diabinese® [chlorpropamide], Orinase® [tolbutamide], or Tolinase® [tolazamide]) if hypoglycemia is present in the field, as these agents are cleared very slowly from the bloodstream and necessitate physician evaluation.

Dextrose 50%

Indications:	• Altered mental status or seizure caused by hypoglycemia – hypoglycemia is defined as:
	  • Adult:
	  o Diabetics = POC glucose analysis less than 110 mg/dL or symptomatic
	  o Non-diabetics = POC glucose analysis less than 80 mg/dL
	  • Pediatric:
	  o Term and Preterm Newborn = POC glucose analysis less than 45 mg/dL
	  o Diabetics = POC glucose analysis less than 70 mg/dL or symptomatic
	  o Non-diabetics = POC glucose analysis less than 70 mg/dL
	  • Coma of unknown cause

Contraindications:	None
Precautions:	• For IV administration, use the antecubital fossa, if possible, to reduce the risk of infiltration • During administration, continuously monitor IV/IO site for patency and signs/symptoms of infiltration • Contact BioTel (prior to drug administration) for hypoglycemia in the patient with head trauma or suspected increased intracranial pressure • Recheck POC glucose analysis 10 – 15 minutes after administration
Side Effects:	Tissue necrosis with infiltration
Adult Dose:	25 grams to 50 grams - standing order
Pediatric Dose:	Newly born infant up to 30 days of age (0.2 g/kg) – standing order, as D10: • Newly born infant under 30 days of age: Discard 40 mL from one 50 mL pre-filled syringe & replace with 40 mL of Normal Saline: administer 2 mL/kg of D10 solution Infant 31 days up to 1 year of age (0.5 g/kg) – standing order, as D10: • 31 days to 1 year of age: Discard 40 mL from one 50 mL pre-filled syringe & replace with 40 mL of Normal Saline: administer 5 mL/kg of D10 solution Child 1 to 13 years of age (0.5 g/kg) – standing order, as D25: • 1 year to 13 years of age: Discard 25 mL from one 50 mL pre-filled syringe & replace with 25 mL of Normal Saline: administer 2 mL/kg of D25 solution
Route:	IV or IO, slow push (to prevent infiltration)
Drug Action:	Increases blood glucose level
Class:	Carbohydrate
Onset:	1 minute
Duration:	Depends on the degree of hypoglycemia

Glucagon

Indications:	• Hypoglycemia when no IV is obtainable and gag reflex is absent (NOT a first-line choice for hypoglycemia); hypoglycemia defined as POC glucose analysis:
	  • Adult: less than 80 mg/dL (non-diabetic), or less than 110 mg/dL or symptomatic (diabetic)
	  • Pediatric: less than 70 mg/dL (non-diabetic), or less than 70 mg/dL or symptomatic (diabetic)
	• Beta blocker and calcium channel blocker toxicity

Contraindications:	Hypersensitivity to proteins
Precautions:	Administer cautiously to: • Patients with cardiovascular disease • Patients with kidney or liver dysfunction
Side Effects:	• Hypotension • Nausea and vomiting • Tachycardia
Adult Dose:	Hypoglycemia: • 1 mg IV/IO/IM/IN/SQ - standing order • If no response, BioTel may authorize additional doses at 20-minute intervals, if necessary Bradycardia (beta-blocker toxicity): • 1 mg – 5 mg IV/IO - standing order • 1 mg IM/IN if IV or IO access not available Cardiac arrest (beta-blocker toxicity): • 1 mg – 5 mg IV/IO push - standing order, may repeat once.
Pediatric Dose 1 to 13 years of age	Standing order: Hypoglycemia: • 1 mg IV/IO/IM/IN/SQ - standing order; may repeat once after 20 minutes. • If no response, BioTel may authorize additional doses. Beta-blocker toxicity: • 1mg IV/IO – BioTel may authorize additional doses at 20-minute intervals.
Pediatric Dose Under 1 year of age	Standing order: Hypoglycemia: • 0.5 mg IV/IO/IM/IN/SQ - standing order; may repeat once after 20 minutes. • If no response, BioTel may authorize additional doses. Beta-blocker toxicity: • 0.5 mg IV/IO – BioTel may authorize additional doses at 20-minute intervals.
Route:	• IM or SQ for hypoglycemia • IM or slow IV push (over 2-5 minutes): bradycardia due to beta-blocker or calcium-channel blocker toxicity • Rapid IV/IO push: cardiac arrest due to beta-blocker or calcium-channel toxicity • Intranasal (IN) as an alternative route when other routes are inaccessible
Drug Action:	• Converts stored glycogen to glucose, increasing blood glucose level • Improves cardiac contractility and increases heart rate
Class:	• Pancreatic Hormone • Insulin Antagonist
Onset:	Within 1 minute - however it may be 15 minutes before any response is observed
Duration:	60 - 90 minutes

Bleeding Control

Although large scale multiple casualty incidents (MCI) such as the Boston Bombing and the San Bernardino shooting capture the nation's attention, smaller scale MCIs are much more common. The National Association of State EMS Officials provides one definition of an MCI as any incident "which generates more patients at one time than locally available resources can manage using routine procedures" (National Association of State EMS Officials, 2012). Using that definition, researchers estimate the yearly incident rate in the United States is about 13.0 MCIs per 100,000 population (Schenk et al., 2014).

Hemorrhage is the second leading cause of death for patients injured in the prehospital environment, accounting for 30-40 percent of all mortality (Kauvar, Lefering, & Wade, 2006). Many of the patients who hemorrhage do so after suffering vascular injuries in one or more extremities. The annual incidence of extremity vascular injuries in the U.S. ranges from a low of 12.4 injuries at a rural trauma center in Missouri (Humphrey, Nichols, & Silver, 1994) to a high of 55 lower extremity injuries at a high-volume urban trauma center in Houston (Feliciano et al., 1988). In a study of isolated penetrating injuries to the extremities, 57 percent of the patients who died had injuries that might have been amenable to tourniquet application (Dorlac et al., 2005).

There is little debate about the value of rapid hemorrhage control for improving outcomes in critically injured trauma patients. The American College of Surgeons Committee on Trauma (ACS-COT) has stated that bleeding must be controlled by prehospital providers as quickly as possible. For maximum efficiency, healthcare providers must apply tourniquets before the patient has developed shock (Department of Homeland Security, 2015). During Operation Iraqi Freedom, tourniquets applied in the field and before the onset of shock were strongly associated with survival (Kragh et al., 2009).

Unfortunately, in cases of severe bleeding, trained professionals may not always arrive in time to prevent exsanguination. Researchers in Austria and Germany found that when traumatic injury occurs, bystanders with varying levels of first aid training are often present on scene before EMS arrives (Pelinka, Thierbach, Reuter, & Mauritz, 2004). In addition, these bystanders often attempt to provide hemorrhage control for patients suffering from an exsanguinating injury. Although prior first aid training increased the probability of successful hemorrhage control by the bystander, the lack of first aid training did not prevent bystanders from attempting to control bleeding and a significant percentage were successful.

Can the Public Help

A central question is whether these bystanders who are present on the scene and willing to help control severe bleeding can become part of a trauma chain of survival. There is very little data in support of this position. However, studies involving a cardiac arrest chain of survival demonstrate that trained bystanders can safely and effectively use defibrillators to resuscitate victims of out-of-hospital cardiac arrest (MacDonald, Mottley, & Weinstein, 2002; O'Rourke, Donaldson, & Geddes, 1997; Page et al., 2000; Valenzuela et al., 2000; Wassertheil, Keane, Fisher, & Leditschke, 2000). Even sixth graders with no previous medical training can achieve performance goals similar to those achieved by trained medical responders (Gundry, Comess, DeRook, Jorgenson, & Bardy, 1999).

Similarly, is it reasonable to think ordinary citizens would be able to safely and effectively apply tourniquets when indicated before the arrival of EMS personnel. Limited available evidence suggests it is. During a simulated explosion, one in five people with no medical training were able to correctly apply a commercially available tourniquet to a manikin's leg in less than 60 seconds (Goolsby, Branting, Chen, Mack, & Olsen, 2015). Providing instructions on a notecard more than doubled the rate of successful placements.

During the Boston Marathon bombing, 29 patients with life-threatening limb exsanguination had 27 improvised tourniquets applied in the field (King, Larentzakis, & Ramly, 2015). EMS personnel applied one-third of those tourniquets and non-EMS personnel or an unknown person applied the remainder.

In a ten-year evaluation of isolated penetrating or blunt extremity injury requiring either arterial revascularization or limb amputation at Boston Medical Center, only 2 percent of patients had a tourniquet applied before arriving at the trauma center and all were improvised tourniquets applied by police officers or bystanders (Kalish et al., 2008). An additional 2 percent of patients had a tourniquet applied by emergency department staff within one hour of arrival. While a very small number of patients without a tourniquet exsanguinated, no patient with a tourniquet died.

During a seven-year period, researchers at Boston Medical Center identified 11 patients who had an improvised tourniquet applied in the field by EMS (Bulger et al., 2014). Only one patient died, however, that patient was in cardiac arrest when EMS arrived on the scene. Of the 10 patients who survived, all had complete neurologic function in the affected extremity despite having the tourniquet in place for as long as 167 minutes (mean 75 +\- 38 minutes).

One concern about bystander application of a tourniquet is whether the bystander will be able to apply the device tightly enough to be effective. Indeed, a manikin study involving non-medical trained bystanders found that 70% of the incorrectly placed tourniquets were judged to be too loose (Goolsby, Branting, Chen, Mack, & Olsen, 2015). However, a battlefield evaluation found that

although morbidity remained high with partially ineffective tourniquet application (persistent distal pulses), mortality actually improved when compared to totally ineffective tourniquets (continued bleeding) (Kragh et al., 2008). This suggests that even when tourniquets are not tight enough to be totally effective, they may still be better than no tourniquet at all.

Hemorrhage-control Training Courses for the Lay Rescuer

The American College of Surgeons convened a special committee to identify changes necessary to improve survival following active shooter and MCIs (Jacobs et al., 2014). One of the major themes to emerge from these series of meetings, known as the Hartford Consensus, is that the public will act as responders to provide aid before the arrival of professional rescuers.

Another major theme was the value of a comprehensive educational program for all members of this trauma chain of survival. Critical to this concept is educational campaigns targeting members of the general public, which should include training on how to apply direct pressure, how to use hemostatic dressings, and application of tourniquets (Jacobs & the Joint Committee to Create a National Policy to Enhance Survivability from Intentional Mass-Casualty and Active Shooter Events, 2016).

In response to the Hartford consensus, the EMS Education Department of the Denver Paramedic Division, in cooperation with the Prehospital Trauma Life Support (PHTLS) committee of the National Association of EMTs (NAEMT) developed training program targeting ordinary citizens (Pons et al., 2015). The 2 ½-hour Bleeding Control for the Injured course combines didactic lectures with hands-on training to teach the lay rescuer important life-saving skills such as hemorrhage control and how to open an airway (National Association of EMTs, 2016).

Also in response to the Hartford Consensus, the White House launched the “Stop the Bleed” campaign (The White House, Office of the Press Secretary, 2015). This campaign hopes to provide public awareness to the simple steps that anyone can take to slow life-threatening bleeding. The campaign also promotes the placement of Bleeding Control Kits in public spaces that would allow members of the general public access to life-saving supplies, similar to public access defibrillation programs.

In 2015, the Harvard School of Public Education and the Harvard School of Government began a bleeding control pilot program at Charlotte Douglas International Airport (National Preparedness Leadership Initiative, 2015). The team placed bleeding control kits inside of each AED cabinet in the airport. Each kit contained pressure dressings, hemostatic dressings, tourniquets, and personal protective gloves. After training the airport emergency staff on the contents, use and location of the kits, the pilot team in conjunction with airport police, conducted three active shooter

scenarios. After-action reporting indicated the responders were able to locate and appropriately use the kits in a simulated incident.

Tourniquets for EMS

A panel of experts in prehospital trauma care convened by the American College of Surgeons recently recommended the prehospital personnel, from emergency medical responders to paramedics incorporate the early application of tourniquets into clinical practice for controlling extremity hemorrhage when direct pressure is ineffective or not practical (Passos et al., 2014). The panel further recommends tourniquets selected for use at a local level be a commercially produced windlass, pneumatic, or ratcheting type device with demonstrated efficacy at arterial flow occlusion.

Three commonly cited reasons for failure to implement TCCC recommendations were the differences in injury patterns between combat and civilian casualties, a perception of no proven benefit in the civilian arena and the perception of harm from prehospital application. However, prehospital application of tourniquets appear safe even when the tourniquet remains in place for one or two hours (Inaba et al., 2015), with a reported complication rate of about 2% (Kue et al., 2015).

For maximum efficacy, tourniquets must be applied before the patient has developed shock (Department of Homeland Security, 2015). During Operation Iraqi Freedom, tourniquets applied in the prehospital environment and before the onset of shock were strongly associated with survival (Kragh et al., 2009). In this study, when field personnel applied the tourniquet before the onset of shock, rather than waiting for shock symptoms to develop mortality virtually disappeared (4% vs. 96%, respectively).

Background on Tourniquet Use

- Several factors led to the historical prehospital prohibition on tourniquet use
    - Tourniquets were sometimes placed when not indicated, or improperly placed
    - Fears of tissue damage and limb loss appear to have discouraged civilian use
- Tourniquets work properly when compression of limb tissue stops arterial blood flow and no distal pulse is present (Kragh, 2010).

Indications (both MUST be present)

- Potentially life-threatening extremity hemorrhage **AND**
- Hemorrhage cannot be controlled by direct pressure

Contraindications

- Non-extremity hemorrhage OR
- Site of extremity hemorrhage precludes the ability to appropriately apply a tourniquet

Procedures for Tourniquet Application (written for the CAT tourniquet)

1. Place the tourniquet snuggly around the affected extremity at least 2 inches, or 4 fingers, above the wound.
2. Twist the windlass until bleeding has been stopped. This should take approximately three, 180 degree rotations. Additional rotations may be needed.
3. Insert the windless end into the Delta Clip. It is only necessary to use one clip.
4. Tighten the safety set screw to prevent accidental release of tension.
5. Once in place, do not remove the tourniquet.
6. Notify BioTel or the receiving hospital en route. Communicate whether bleeding has been controlled or not, as well as time of tourniquet application.
7. Continue to monitor the patient's vital signs and the wound for recurrent bleeding.
    a. The tourniquet and bleeding site should be left uncovered or with minimal bandaging to facilitate frequent wound site re-evaluation.
    b. If the application of a tourniquet fails to control bleeding apply a second tourniquet.
    c. Document the time that a tourniquet was applied.
        i. Time of application should be written directly on the tourniquet
        ii. Time of application should be documented in the patient care report

Special Circumstances

- Improvised tourniquets applied by bystanders and non-medical personnel prior to EMS arrival are not a substitute for a commercial device properly applied by UTSW/BioTel paramedics.
- In such cases, apply (but do not secure) a BioTel agency-approved commercial tourniquet proximal to the improvised device.
- Apply direct pressure to the wound and remove the improvised tourniquet.
- If the wound begins bleeding again and direct pressure does not control the hemorrhage apply the commercial tourniquet as described above.

Complications

- Insufficient compression will stop only venous flow (essentially creating a venous tourniquet) trapping blood in the limb with potentially life threatening consequences (Kragh, 2010; Lee, Porter, & Hodgetts, 2007).
    - The trapped blood causes limb edema and loss of blood to the general circulation, which can hasten the onset of shock.
    - Bleeding may actually increase with development of venous hypertension.
    - Venous tourniquets have been associated with increased mortality (Kragh et al., 2008).
- Other complications include ischemia, compression, and reperfusion injury (Fitzgibbons, Digiovanni, Hares, & Akelman, 2012; Kragh, 2010).
    - Muscle cells, in particular, may be more susceptible to ischemia and reperfusion effects after prolonged tourniquet use.
    - Nerve compression may result in neuropathy and weakness; however, evidence suggests this nerve damage is typically minor and reversible (Kragh, 2010).

Hemostatic Agents and Dressings

Increasingly, EMS systems across the country are adding topical hemostatic agents to their prehospital treatment of hemorrhage (Kerby & Cusick, 2012). Topical hemostatic agents are available as powders, granules, or dressings composed of traditional gauze or dressings impregnated with the active agent. The ideal agent (Granville-Chapman, Jacobs, & Midwinter, 2011) should be

- able to stop hemorrhage from large arteries and veins within two minutes of application,
- capable of effective application through pools of blood, packaged as a ready to use agent,
- simple to use even by non-medically trained responders,
- lightweight and durable,
- capable of extended storage under a wide range of temperatures,
- inexpensive, and
- free from bacterial or viral risk.

To date, no topical hemostatic agent meets all these criteria. However, there are a number of agents available on the market. Physicians categorize these agents into one of three groups based on the mechanism of action: mucoadhesive agents, factor concentrators, or procoagulant supplements (Granville-Chapman, Jacobs, & Midwinter, 2011).

Mucoadhesive agents react with blood to create a seal over the wound, which arrests continuing blood flow. Both HemCon® and Celox™ utilize a granular chitosan salt derived from the shells of marine arthropods (Granville-Chapman, Jacobs, & Midwinter, 2011). These salts, which are positively-charged, react with and bind to negatively-charged red blood cells rapidly forming a cross-linked barrier clot which seals the injured vessel (Burkatovskaya et al., 2006; Kozen, Kircher, Henao, Godinez, & Johnson, 2008).

Researchers found HemCon® to be clinically superior to standard gauze in a low-pressure, high-flow model of venous bleeding (Pusateri et al., 2003), although this type of injury might not represent the injury patterns encountered in the prehospital environment (Lawton, Granville-Chapman, & Parker, 2009). In a high-pressure model of uncontrolled arterial hemorrhage, HemCon® was initially effective at controlling but could not sustain hemostasis (Kheirabadi, Acheson, Sondeen, Ryan, & Holcomb, 2004). Despite this failure, a retrospective review of 34 cases of hemorrhage treated with HemCon® by Portland, Oregon firefighters revealed bleeding control in 79% of the cases (Brown, Daya, & Worley, 2009). On the other hand, in a side by side comparison of commonly used topical hemostatic agents in a swine model of uncontrolled hemorrhage, Celox™ was the only agent that improved short term survival (Kozen, Kircher, Henao, Godinez, & Johnson, 2008).

Factor concentrators, such as QuikClot® rapidly absorb water from the blood at the injury site, which concentrates platelets and other intrinsic clotting factors resulting in faster clot formation. The active ingredient in QuikClot® is zeolite, an inert volcanic mineral that rapidly absorbs water in an exothermic (heat-producing) reaction. In addition to its water absorbing properties, an in vitro examination revealed zeolite also rapidly increases calcium ion concentration of blood which promotes rapid clot formation (Li et al., 2013). In the first generation of QuikClot®, healthcare providers poured the zeolite granules directly into the wound. However, physicians soon found the exothermic reaction was significant enough to cause burns and tissue necrosis (McManus, Hurtado, Pusateri, & Knoop, 2007; Rhee, et al., 2008; Wright et al., 2004). As a result, the granular form of QuikClot® is no longer available.

The second generation of QuikClot® replaced the granules with larger zeolite beads and packed them into a small mesh bag (QuikClot® ACS+™) that was inserted into the bleeding wound. The bag facilitates removal of the product during surgery. Changes in the second generation of the product reduced the temperatures created by the reaction and produced a safer topical agent (Ahuja et al., 2006).

One factor concentrator that does not produce an exothermic reaction is WoundStat™, which is a biodegradable powder composed of smectite clay mineral and a cross-linked poly-acrylic acid (Lawton, Granville-Chapman, & Parker, 2009). Smectite particles have a negative charge which

activates coagulation pathways and promotes clotting (Kheirabadi et al., 2010). Although early animal studies demonstrated the effectiveness of WoundStat™ in controlling hemorrhage and improving survival (Clay, Grayson, & Zierold, 2010; Kheirabadi et al., 2009; Ward et al., 2007), a subsequent study demonstrated the active particles in WoundStat™ damaged blood vessel linings, caused occlusive thrombus in injured vessels and migrated to the pulmonary vasculature (Kheirabadi et al., 2010). As a result, this product was removed from the market (Kerby & Cusick, 2012).

Other factor concentrators use microporous polysaccharide hemospheres derived from potato starch (TraumaDex™). When compared to QuikClot® in a swine groin wound model, this product proved less effective and in fact, was no better than standard gauze dressings (Alam et al., 2003).

Procoagulant supplements deliver additional clotting factors to the wound which then combine with clotting factors already present. Together, these clotting factors increase the rate of blood clot formation. Some of the products deliver human clotting factors while other deliver factors derived from bovine blood (Granville-Chapman, Jacobs, & Midwinter, 2011).

The only procoagulant supplement approved by the Food and Drug Administration is Combat Gauze™ (Littlejohn, Bennett, & Drew, 2015). This product is actually the third generation of QuikClot® products in which the manufacturer replaced the zeolite with kaolin, a clay containing the active ingredient aluminum silicate. Combat Gauze™ uses gauze dressings impregnated with kaolin. An animal model determined Combat Gauze™ to be as effective as the second generation QuikClot® at controlling hemorrhage without producing excessive heat (Baker, Sawvel, Zheng, & Stucky, 2007). Researchers conducting an evidence-based review in an attempt to determine if Combat Gauze™ was safe for controlling hemorrhage in the prehospital setting determined that although not conclusive, the results were promising in support of the product (Gegel, Austin, & Johnson, 2013).

A side-by side comparison of four hemostatic dressings in an animal model of arterial hemorrhage demonstrated survival superiority associated with the use of Combat Gauze™ (Kheirabadi, Scherer, Estep, Dubick, & Holcomb, 2009). In this study, researchers planned to test each of the products in 10 animals. However, two of the chitosan-based products (HemCon® and Celox™-D) failed to achieve hemostasis in the first six tests and all of the animals died. As a result, the researchers did not test those products in the final four animals.

In a similar study, researchers found rebleeding after initial hemostasis in 33% of the animals treated with Celox™ gauze compared to no rebleeding seen in animals treated with Combat Gauze™(Rall et al., 2012).

In an animal model of uncontrolled hemorrhage, researchers tested whether Combat Gauze™ produced a more stable clot compared to standard wound packing practices (Gegel et al., 2012). After achieving hemostasis, the researchers moved the animals affected leg to simulate movement

that might occur during evacuation and transportation to more definitive care. The number of movements required to produce rebleeding after using Combat Gauze™ was significantly higher compared to standard wound packing therapy.

Clinicians have even reported success in using Combat Gauze™ to control bleeding related to percutaneous catheter insertion sites for patients undergoing extracorporeal membrane oxygenation (ECMO) support (Lamb, Pitcher, Cavarocchi, & Hirose, 2012). Use of safe and effective hemostatic dressings for patients undergoing ECMO has the potential to reduce the need for blood transfusions, surgical exploration, overall healthcare costs, and promote faster patient recovery.

Tactical Combat Casualty Care guidelines developed by the United States Special Operations Command recommend Combat Gauze™ as the hemostatic dressing of choice (Bennett et al., 2014). However, the guidelines allow for alternative use of Celox™ gauze and ChitoGauze® in the event Combat Gauze™ is not available.

A panel of experts in prehospital trauma care convened by the American College of Surgeons recently recommended the prehospital use of topical hemostatic agents in conjunction with direct pressure for controlling hemorrhage in injuries where direct pressure alone is ineffective or not practical and in cases where tourniquet application is not possible due to anatomic limitations (Bulger et al., 2014). Although not endorsing the use of a specific product, the panel recommended that EMS systems choose a product with demonstrated efficacy and available in gauze format, which permits wound packing.

Special Note

- There are no current plans for placing hemostatic agents or dressings on the MICUs.
- However, every Dallas Police officer was issued a tourniquet and QuickClot® LE Combat Gauze, which they may use on themselves, other officers, or civilians.
- It is important for each paramedic to be familiar with the agents because they may arrive on a scene where an officer is using the gauze.
- In addition, some of the products are available commercially (Cabella's, Walmart, etc.) and you see them being used before you arrive on the scene.

Background

- Topical hemostatic agents may be useful for injuries (such as junctional wounds) in which tourniquet use is not feasible (Kheirabadi, 2011; Sharpe, Barneby, & Russell, 2011; Smith, Laird, Porter, & Bloch, 2013).

- These agents have physical properties that allow the agent to adhere to damaged tissue and seal ruptured blood vessels or enhance natural blood clotting mechanisms to accelerate clot formation and produce a strengthened clot.

What is QuickClot® LE Combat Gauze?

- QuickClot® LE Combat Gauze is a soft, white, sterile, 2” x 2”, nonwoven gauze impregnated with kaolin, an inert mineral that does not contain animal or human proteins or botanicals.
- The gauze contains an X-ray detectable strip for easy identification
- Unlike the first and second generation of hemostatic agents, kaolin does not create an exothermic reaction when exposed to blood and therefore does not burn the patient.
    - The only procoagulant supplement approved by the Food and Drug Administration (Littlejohn, Bennett, & Drew, 2015)
    - Produces a more stable clot compared to standard wound packing practices (Gegel et al., 2012)
    - Demonstrated survival superiority when compared to other hemostatic dressings (Kheirabadi, Scherer, Estep, Dubick, & Holcomb, 2009)
    - Safe for controlling hemorrhage in the prehospital environment (Gegel, Austin, & Johnson, 2013)
    - Is the hemostatic agent of choice by the United States Special Operations Command (Bennett et al., 2014)

How is QuickClot® LE Combat Gauze used?

- The gauze is Z-folded
- Law enforcement officers will insert one end of the gauze into the wound and quickly pack the remaining gauze
- After wound packing, the officer should hold direct pressure until bleeding stops.
- If blood soaks through the first application of Combat Gauze, the officer may apply additional dressings over the first

What do paramedics need to know about QuickClot® LE Combat Gauze?

- If bleeding continues, do not remove the QuickClot® LE Combat Gauze
- Instead, apply additional dressings over the first
- Do not disturb the clot that is forming under the first dressing

TOURNIQUET APPLICATION

Purpose: The purpose of this policy is to assist paramedics with the indications for, and application and management of a prehospital tourniquet.

UTSW/BioTel paramedics may apply any EMS agency-approved tourniquet to control blood loss under certain conditions. Once applied, these patients require transport to a Trauma Center.

1) **Indications for tourniquet application**
   a. Potentially life-threatening extremity hemorrhage **AND**
   b. Hemorrhage cannot be controlled by direct pressure.

2) **Contraindications for tourniquet application**
   a. Non-extremity hemorrhage OR
   b. Site of extremity hemorrhage precludes the ability to appropriately apply a tourniquet.

3) Procedures for tourniquet application:
   a. UTSW/BioTel paramedics shall be familiar with safe tourniquet application technique.
   b. The tourniquet and bleeding site should be left uncovered or with minimal bandaging to facilitate frequent wound site re-evaluation.
   c. If the application of a tourniquet fails to control bleeding a second tourniquet may be applied.
   d. It is essential to document the time that a tourniquet was applied.
      i. Time of application should be written directly on the tourniquet
      ii. Time of application should be documented in the patient care report
   e. Once placed, the tourniquet should not be removed until the patient is transferred to a higher level of care (see special circumstance below)
   f. Notify BioTel or the receiving hospital en route that the patient has had a tourniquet applied. Communicate whether bleeding has been controlled or not, as well as time of tourniquet application.
   g. Continue to monitor the patient's vital signs and the wound for recurrent bleeding.

Special circumstances:

Improvised tourniquets applied by bystanders and non-medical personnel prior to EMS arrival are not a substitute for a commercial device properly applied by UTSW/BioTel EMS providers.

a. In such cases, a BioTel agency-approved commercial tourniquet should be applied (but not secured) proximal to the improvised device prior to its removal, if possible. If hemorrhage uncontrolled by direct pressure reoccurs after removal of the improvised device, the commercial tourniquet shall be deployed using the procedure described in this Policy.

Rarely, a patient who has had a tourniquet placed by a first-responding law enforcement officer or citizen prior to EMS arrival may decline an offer of transport to the hospital by ambulance. This should be strongly discouraged. However, should paramedics be unsuccessful in convincing the patient to accept ambulance transport, the following steps shall be taken:

a. Explain to the patient that the tourniquet cannot remain in place if the patient is not being transported by ambulance, and that removal of the tourniquet may result in uncontrolled bleeding and possibly death.
b. Contact BioTel requesting that the Medical Command Physician speak directly with the patient to try to convince the patient to accept transport.
c. If the Medical Command Physician fails to convince the patient to accept ambulance transport, and upon acknowledgment of the warnings, the tourniquet should be slowly released over 3 to 5 minutes.
d. If bleeding recurs, apply direct pressure/pressure bandaging and observe the patient for 10 minutes. If bleeding remains uncontrolled, re-apply the tourniquet and contact BioTel for further assistance.
e. If bleeding is controlled with direct pressure/pressure bandage, document this, as well as the presence of distal pulses and capillary refill. Then, have the patient sign the refusal, and encourage them to seek immediate emergency medical care by whatever means they choose.