**August 1, 2011 REMAC Protocol revisions in effect**

**From the Editor**

August 1, 2011 REMAC Protocol revisions in effect

Although normally scheduled for April 1, this year’s NYC REMAC protocol update were changed to August 1 implementation in the field and on certification exams.

Only the August 1, 2011 protocols are in effect.

Always see nycremsco.org for the current approved protocols.

REMEMBER: the protocols on the street are the protocols on the exam!

Mandatory REMAC Credentialing Fee

A $25 fee has been instituted by NYC REMAC for all new or recertifying paramedic credentials. On successfully completing a REMAC exam, candidates will receive a temporary letter verifying certification. They will soon after be mailed a memo directly from NYC REMSCO requiring a completed application, proof of NY State paramedic certification, and credentialing fee by money order only. On receipt, a permanent NYC REMAC certification card will be issued.

Please direct inquires on this process to NYC REMSCO at 212-870-2301
Outline of August 2011 NYC REMAC protocol changes

see REMAC Advisories 2011-02, 2011-03, 2011-04 at nycremsco.org

General Operating Procedures

- **CPR**: clarifies that REMAC follows AHA except as specified
- **Advanced Airway Management**: adds section making use of ETI and alternative airways equal except in non-cardiac arrest situations, limiting ETI to 2 total attempts
- **Definition of Unstable Dysrhythmias**: removes chest pain, SOB, possible MI from definition

CFR Protocols

- 300 WMD, 301 Resp Distress/Failure, 320 Traumatic Arrest, 328 Burn: updated to match BLS protocols
- 304 Non-Traumatic Chest Pain: removes blood pressure assessment and assistance or patient with NTG admin

BLS Protocols

- 403 Non-Traumatic Arrest: mandates AED availability & use; moves transport order to step 8
- 407 Wheezing: removes wheezing from list of assessment criteria; mandates OLMC contact for epinephrine to patients over 33 years-old
- 410 Anaphylaxis: mandates OLMC contact for patients over 33 years-old
- 413 Seizures: removes list of signs/symptoms
- 414 Poisoning or Drug OD: removes OLMC contact, information list, & order for dilution
- 426 Soft Tissue Injuries: adds tourniquet option
- 430 EDP: removes GCS from assessment

ALS Protocols

**“ETI” changed to “Advanced Airway Management”**

- 500-A Smoke Inhalation**: changes dopamine admin to Standing Order
- 500-B Cyanide Exposure**: removes note on indications; changes dopamine admin to Standing Order
- 501 Resp Arrest: protocol deleted
- 503 Non-traumatic Arrest: limits switching from AED to ALS monitor only at the end of CPR cycle
- 503-B PEA/Asystole**: removes atropine

- 504-A Suspected MI: moves aspirin to step1; makes total doses of NTG unlimited under Standing Orders; removes morphine & Medical Control Options
- 504-B Cardiogenic Shock: moves fluid bolus and dopamine to Standing Order
- 505-A, B & C Dysrhythmias: adds note: if defibrillator’s maximum joule setting is less than 360, use equivalent cardioversion energies
- 506 APE: makes total doses of NTG unlimited under Standing Orders
- 507 Asthma & 508 COPD: makes total doses of albuterol unlimited under Standing Orders; mandates mixing of albuterol & ipratropium, limited to 3 doses
- 510 Anaphylaxis: changes methylprednisolone and dexamethasone to Standing Orders
- 515 Non-Cardiogenic Shock & 520 Traumatic Arrest: removes repeat of fluids under Medical Control Options
- 521 Head Injuries**: clarifies indication for advanced airway management & moves it to step 2
- 528 Burns & 529 Pain Management: adds fentanyl to Medical Control Options
- 531 Severe Nausea/Vomiting: new protocol
- 543 Neonate Resus: removes meconium aspiration; moved IV/IO access, epi and fluid bolus admin to Standing Orders; removes Medical Control Options
- 550 Peds Resp Arrest: adds note referring to Peds AMS protocol; changes naloxone to weight-base dosing with titration; removes ET admin of naloxone
- 551 Peds Obstructed Airway: clarifies procedure with cuffed ET tube
- 553 Peds Non-Traumatic Arrest**: increases joule settings
- 559 Peds Traumatic Arrest**

Appendices

- **Appendix B Patient Assessment**: clarifies transport decision; removes CUPS
- **Appendix D AED Guidelines**: appendix deleted
- **Appendix I Hospital Listing**: adds pediatric ages
- **Appendix T Use of Tourniquets**: appendix added
REMAC Exam Study Tips

REMAC candidates have difficulty with:

* Epinephrine use for peds patients
* 12-lead EKG interpretation
* Ventilation rates for peds & neonates

REMAC Written exams are approximately:

* 15% Protocol GOP
* 10% BLS
* 10% Adult Arrest
* 40% Adult Med. Emerg.
* 10% Adult Trauma
* 10% Pediatrics

Certification & CME Information

- **Of the 36 hours of Physician Directed Call Review CME required for REMAC Refresher recertification, at least 18 hours must be ACR/PCR Review (which may include QA/QI Review). The remaining 18 hours may include ED Teaching Rounds and OLMC Rotation.**

- Failure to maintain a valid NYS EMT-P card will invalidate your REMAC certification.

- By the day of their refresher exam all candidates must present a letter from their Medical Director verifying fulfillment of CME requirements. Failure to do so will prevent recertification.

- FDNY paramedics, see your ALS coordinator or Division Medical Director for CME letters.

- CME letters must indicate the proper number of hours, per REMAC Advisory # 2000-03:
  - 36 hours - Physician Directed Call Review
    - ACR Review, QA/I Session (<strong>minimum 18 hours of ACR/QA review</strong>)
    - Emergency Department Teaching Rounds, OLMC Rotation
  - 36 hours - Alternative Source CME - **Maximum of 12 hours per venue**
    - Online CME - Clinical rotations
    - Lectures / Symposiums / Conferences - Associated Certifications:
    - Journal CME BCLS / ACLS / PALS / NALS / PHTLS

REMAC Refresher Written examinations are held monthly, and may be attended up to 6 months before your expiration date. See the exam calendar at the end of this Journal. To register, call the Registration Hotline @ 718-999-7074 by the last day of the month prior to your exam.

REMAC Quarterly Written and Oral examinations are held every January, April, July & October. Registration is limited to the first 50 applicants. See the exam calendar at the end of this journal.

REMAC CME and Protocol information is available, and suggestions or questions about the newsletter are welcome. Call 718-999-2671 or email swansoc@fdny.nyc.gov

www.EMINET.com
FDNY ALS Division Coordinators

Citywide ALS 718-999-1738  Division 4 718-281-3392
Capt. Joseph Pataky  Mike Romps
Division 1 212-964-4518  Division 5 718-979-7175
Joseph Farrell  Joseph D’Agosto
Division 2 718-829-6069  Bureau of Training 718-281-8325
Edwin Martinez  Hector Arroyo
Division 3 718-968-9750  EMS Pharmacy 718-571-7620
Gary Simmonds  Cindy Corcoran

FDNY EMS Medical Directors

Dr. Glenn Asaeda 718-999-2666  Dr. Dario Gonzalez 718-281-8473
Field Response Division 5  Field Response Division 2
OLMC Director, REMAC Coordinator  USAR/FEMA Director, OEM Liaison
Dr. David Ben-Eli 718-999-0404  Dr. Doug Isaacs 718-281-8428
Field Response Division 3  Field Response Division 1
Haz-Tac, PASU & EMS Resident Director  EMS Training & Rescue Medic Director
Dr. John Freese 718-999-2790  Dr. Bradley Kaufman 718-999-1872
Chief Medical Director  Field Response Division 4
Prehospital Research Director  QA, EMD & EMS Fellowship Director
EMS Fellows
Dr. Pamela Lai 718-999-0364  Dr. Michael Redlener 718-999-0351

FDNY OLMC Physicians and ID Numbers

Alexandrou, Nikolaos 80282  Isaacs, Doug 80299
Asaeda, Glenn 80276  Jacobowitz, Susan 80297
Barbara, Paul 80306  Jameson, Angus 80309
Ben-Eli, David 80298  Kaufman, Bradley 80289
Cox, Lincoln 80305  Munjal, Kevin 80308
Freese, John 80293  Schenker, Josef 80296
Giordano, Lorraine 80243  Schneitzer, Leila 80241
Gonzalez, Dario 80256  Schoenwetter, David 80304
Hansard, Paul 80226  Silverman, Lewis 80249
Hegde, Hradaya 80262  Soloff, Lewis 80302
Hew, Phillip 80267  Van Voorhees, Jessica 80310
Huie, Frederick 80300
**DIABETES MELLITUS & DIABETIC EMERGENCIES**

You respond to a call in the subway, where you find a confused and disheveled 35 year-old patient with warm / dry skin, heavy breathing, tachycardia, and sweet / fruity breath. On exam, you find that the patient is wearing a medical alert bracelet stating “Diabetes Mellitus.” What do you believe is the condition that he is experiencing? How would you manage this patient?

You respond to another call where you find a confused, elderly patient at home. The patient’s skin is pale, cool, and clammy, and he has a weak, rapid pulse. On the dining-table you notice the patient’s uneaten dinner. On exam, you find that the patient is wearing a medical alert bracelet stating “Diabetes Mellitus.”

What do you believe is this patient’s primary medical issue? How would you manage this patient? Would it differ from the first patient and why?

Many of us have treated such patients. Both have similar presentations: confusion, tachycardia, and bracelets stating “Diabetes Mellitus.” The causes of their conditions, however, are on the opposite ends of the diabetic spectrum.

As prehospital healthcare providers, we commonly encounter diabetic emergencies. These problems include complications from a blood sugar level that is too low (hypoglycemia), which can result in seizures, coma, or even death. At the other extreme, a blood sugar level that is too high (hyperglycemia) can manifest as one of two emergencies: diabetic ketoacidosis (DKA) or hyperosmolar hyperglycemic nonketotic coma (HHNC).

In this month’s article, we will review the basic principles of diabetes, describe the types of diabetes mellitus and the differences between the types, review the medications commonly used by diabetics, and discuss the medical emergencies that can result from this chronic, life-long disease.

**DIABETES MELLITUS OVERVIEW**

When translated from Latin, diabetes mellitus (DM) means sweet (mellitus) urine (diabetes), perhaps alluding to how this condition was once diagnosed. (Makes you thankful for glucometers, no?) DM is a multi-system disease that has significant physiologic and anatomical/structural complications. It is a chronic disease of carbohydrate, fat, and protein metabolism caused by either a decrease in or lack of insulin, or the decrease in the response to insulin.

Insulin, produced in the pancreas, is responsible for decreasing blood sugar by facilitating its movement into cells throughout the body. In addition to promoting the uptake of glucose into cells, it also helps to regulate fatty acids, amino acids, and potassium, and it stimulates enzymes that are responsible for storing glucose by generating glycogen in the liver.
The pancreas also produces another hormone called glucagon which works in a way opposite of insulin, increasing blood sugar by encouraging the conversion of glycogen back into glucose.

The morbidity and mortality associated with diabetes are related to both the short- and long-term complications. And although the pathophysiology of the disease differs between the types of diabetes (see below), most of the complications are similar regardless of the type of diabetes. As a result of these complications, people with DM have an increased risk of developing coronary artery disease (2-4 times greater in patients with DM than in the rest of the population), increased risk of infections, cerebral vascular disease (including CVA), peripheral vascular disease (PVD) that can lead to ischemia and even gangrene of lower limbs, chronic kidney disease, reduced visual acuity to the point of blindness, and autonomic and peripheral neuropathy.

And the statistics surrounding these complications are not inconsequential. Approximately two thirds of people with DM die of heart disease or stroke. DM is the major cause of blindness in adults aged 20-74 years, as well as the leading cause of heart disease, nontraumatic lower-extremity amputations, and end-stage renal disease (ESRD) requiring dialysis. Add to that the fact that people with DM are at an increased risk for many types of cancer, and it becomes clear why very strict control of this disease is needed if patients are to live normal, productive lives.

Patients with DM face a lifelong challenge to achieve and maintain blood glucose levels as close to the normal range as possible. With appropriate glucose control, the risk of both microvascular and neuropathic complications is decreased markedly. In addition, if high blood pressure (hypertension) and high cholesterol (hyperlipidemia) are treated aggressively, the risk of macrovascular complications (stroke, myocardial infarction) decreases as well. For this reason, daily self-monitoring of blood glucose with a glucometer is important for patients treated with insulin or oral diabetic medications (see below) to monitor for and prevent both hyper- and hypoglycemia and optimize the patient’s chronic treatment regimen.

**Type 1 DM**

Type 1 DM, formerly called insulin-dependent diabetes mellitus (IDDM) or juvenile diabetes, results from a lack of adequate insulin following autoimmune (the patient’s immune system turning on the patient’s body) destruction of the portion of the pancreas responsible for insulin production, in particular the beta cells (see Figure 1). One possible reason for this autoimmune reaction is thought to be a virus that triggers the immune system to develop antibodies which then target pancreatic beta cells or molecules in the beta cells that resemble the virus.
Type 1 DM is usually diagnosed early in life. However, it can occur at any age including adults, especially in those in their late 30s and early 40s. It accounts for 10% to 20% of all DM cases and affects men slightly more often than women. Approximately 1 million Americans have Type 1 DM, and it is the most common metabolic disease of childhood, affecting about one in every 400-600 children and adolescents.

No matter what the patient’s age may be, the failure of their pancreas to produce adequate insulin means that these patients are dependent upon regular treatment with supplemental (injected) insulin. Without it, their body will be unable to use the glucose available to it and will result to other processes to generate the energy needed by the body’s cells, including breaking down fatty acids and proteins (see below).

**Type 2 DM**

Type 2 DM, formerly known as non-insulin-dependent diabetes mellitus (NIDDM) or adult-onset diabetes, results from a combination of the inability of the body’s cells to use the available insulin (i.e. insulin resistance) and a decrease in insulin production. For Type 2 DM to occur, both must exist. This type of DM accounts for 80% to 90% of diabetes.

A distinguishing feature of Type 2 DM is that almost all patients have some natural insulin production. This means that the condition can be controlled with dietary changes and/or oral medications designed to aid the body in using the available insulin (overcoming the insulin resistance), though some patients do still require insulin therapy. This is because, with prolonged disease, patients with poorly controlled Type 2 DM eventually develop a lack of insulin production as well, and will require insulin supplementation.

**Gestational DM**

Gestational diabetes mellitus (GDM) is defined as any degree of glucose intolerance with onset or first recognition during pregnancy. GDM is a complication of approximately 4% of all pregnancies in the United States.

During the first trimester the presence of increased levels of progesterone and estrogen causes an increased sensitivity to insulin. This, along with the glucose demands of the placenta and fetus, can lead to episodes of hypoglycemia, especially in the patient with preexisting DM. During the second trimester resistance to insulin occurs and peaks in the third trimester. This can lead to significant elevations in blood glucose levels, or GDM. If untreated, GDM can lead to fetal complications including increased fetal size, hypoglycemia, hypocalcemia, and an increased risk of fetal death. However, if discovered early and appropriate treatment (typically insulin) is initiated, the impact is of the disease for the mother and fetus can be minimal.

**Diabetes Medications**

A comprehensive knowledge of diabetic medication is not required to care for a diabetic patient, but an understanding of these medications and general familiarity can help to make decisions for these patients (including whether or not to have a high index of suspicion for certain patients, particularly when they are requesting an RMA) and to help recognize a diabetic history from a patient’s medication list. Figures 2 and 3 provide an overview of the medications typically used to chronically manage diabetes.
<table>
<thead>
<tr>
<th>Type</th>
<th>Trade Name</th>
<th>Onset (hrs)</th>
<th>Peak (hrs)</th>
<th>Duration (Hrs)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aspart.glulisine.Lispro</td>
<td>Novalog, Aprida, Humalog</td>
<td>0.2-0.5</td>
<td>0.5-2</td>
<td>3-4</td>
</tr>
<tr>
<td>Regular</td>
<td>Humulin R, Novolin R</td>
<td>0.5-1</td>
<td>2-3</td>
<td>6-8</td>
</tr>
<tr>
<td>NPH</td>
<td>Humulin N, Novolin N</td>
<td>1.5</td>
<td>4-10</td>
<td>16-24</td>
</tr>
<tr>
<td>Lente</td>
<td>Humulin L, Novolin L</td>
<td>1.5-3</td>
<td>7-15</td>
<td>16-24</td>
</tr>
<tr>
<td>Ultralente</td>
<td>Humulin U</td>
<td>3-4</td>
<td>9-15</td>
<td>22-28</td>
</tr>
<tr>
<td>Glargine</td>
<td>Lantus</td>
<td>?</td>
<td>no peak</td>
<td>24-36</td>
</tr>
</tbody>
</table>

**Figure 2:** Insulin types, trade names, and timing of action.

<table>
<thead>
<tr>
<th>CLASS</th>
<th>MECHANISM OF ACTION</th>
<th>EXAMPLES</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sulfonylureas</td>
<td>Long acting medication that stimulates insulin release from the pancreas. This group probably has the greatest effect on lowering blood sugar of any of the oral agents.</td>
<td>glyburide, glipizide (Glucotrol), glimepiride (Amaryl)</td>
</tr>
<tr>
<td>Meglitinides</td>
<td>Shorter-acting medications that, taken before meals, help to stimulate natural insulin release with less risk for hypoglycemia.</td>
<td>Repaglinide (Prandin), Nateglinide (Starlix)</td>
</tr>
<tr>
<td>Biguanides</td>
<td>Older medication that reduces hyperglycemia by decreasing the production of new glucose via the liver (primary effect) and increasing peripheral insulin sensitivity (secondary effect). It does not increase insulin levels.</td>
<td>metformin (Glucophage)</td>
</tr>
<tr>
<td>Alpha-glucosidase</td>
<td>Delay sugar absorption from the GI tract and thereby help prevent glucose surges after meals.</td>
<td>Glyset, Precose</td>
</tr>
<tr>
<td>inhibitors</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Thiazolidinediones (glitazones)</td>
<td>Act as insulin sensitizers that reduce insulin resistance in the body (i.e., sensitize muscle and fat to the actions of insulin) and perhaps to a small degree in the liver.</td>
<td>Avandia, Actos, Rezulin</td>
</tr>
<tr>
<td>Incretin mimetics</td>
<td>Stimulates glucose-dependent insulin release from the pancreas; it also reduces glucagon production and slows gastric emptying.</td>
<td>Byetta, Victoza</td>
</tr>
<tr>
<td>dipeptidyl peptidase IV (DPP-4) inhibitors</td>
<td>Newer class of medications that prolong the action of naturally-occurring hormones that stimulate insulin release from the pancreas.</td>
<td>Januvia, Onglyza, Tradjenta</td>
</tr>
<tr>
<td>Amylin analogs</td>
<td>Mimics the action of naturally occurring hormones that delay gastric emptying and modulating appetite.</td>
<td>Symlin (pramlintide)</td>
</tr>
</tbody>
</table>

**Figure 3:** Oral Hypoglycemic Medications: Through the mechanisms described above, these medications are designed to lower blood sugar by taking advantage of natural insulin in the patient’s body or via the other mechanisms listed.

Perhaps the most important thing to recognize about diabetic medications that will alter the care that you provide in the prehospital setting is the mechanism of action. As discussed below, hypoglycemia is a common medical emergency, particular among diabetics. When a patient takes too much of their insulin, unless it is one of the short-acting forms, there is a risk for
further episodes of hypoglycemia. Similarly, when a patient is taking any of the oral hypoglycemic agents, there is a risk for recurrent hypoglycemia. For this reason, with any hypoglycemic event (even if treatment was not required), the patient should be viewed as having a high index of suspicion and, if an RMA is requested, OLMC contact should be made.

DM EMERGENCIES

HYPOGLYCEMIA

Hypoglycemia is a common problem experienced by both patients with Type 1 DM and Type 2 DM. As mentioned above, the diabetic patient must intensively control his or her DM to prevent short and long-term complications and, in trying to do so, reducing the blood sugar too much is a very real possibility.

Mild to moderate hypoglycemia is common and, although not completely preventable, it is easily treated. It can be detected with close blood glucose monitoring, and rarely results in complications to the patient.

Serious hypoglycemia requires immediate intervention and treatment. If undetected or untreated, serious hypoglycemia can lead to seizures, coma, or death.

The body's first line of defense against low blood sugar is to reduce insulin production by the pancreas and to increase glucagon production. The body's second line of defense is the secretion of catecholamines by the adrenal gland, which include epinephrine (“adrenaline”) and norepinephrine. The effects of this catecholamine release can be seen in the hypoglycemic patient as tachycardia and diaphoresis. Lastly, stimulation of the autonomic nervous system contributes to signals that trigger the release of various hormones and symptoms telling the body that sugar is low, which should lead a person to consume sugar.

Patients with Type 1 DM do not make sufficient insulin. As a result, the body's first line of defense against hypoglycemia is lost and a decrease of insulin levels is not possible. Often a low blood sugar in these patients is caused by excessive insulin action that results from:

- inaccurate dosing (“taking too much insulin” or “an extra dose”)
- mismatch with carbohydrate intake and exogenous insulin intake (“took insulin but forgot to eat or didn’t eat enough”)
- increased use of glucose, as in exercise, causing a sharp drop in blood glucose
- intentional overdose

Hypoglycemia is also common among patients with Type 2 diabetes. Medications given to treat Type 2 DM act by either stimulating the body's ability to secrete insulin, by improving insulin action, or by the other mechanisms described in Figure 3. These medications also have a tendency to contribute to hypoglycemia, especially in the elderly and those with liver or kidney disease, who cannot metabolize these medications properly.

If the hypoglycemia is caused by excessive insulin dosing or the prolonged or exaggerated effect of oral DM medication, the low blood sugar effect will be prolonged and a more long-term treatment (i.e. more than we can provide in the prehospital setting – oral glucose or an amp of D50) may be needed.

Some patients who have had Type 1 DM for many years, and to a lesser degree, patients who have had Type 2 DM for many years, have been shown to have a lack of glucagon release from the alpha cells of the islets of Langerhans in the
pancreas in response to hypoglycemia (thus preventing the liver from breaking down glycogen to glucose, termed *glycogenolysis*). Lack of glucagon response makes the body more dependent on epinephrine to overcome the effects of hypoglycemia, yet there may be some lack of responsiveness to epinephrine in DM as well.

Prolonged disease can also decrease a patient's ability to recognize a low blood sugar, preventing him / her from taking the necessary measures of self-treatment. This is called *hypoglycemic unawareness*.

**History and physical findings**: Hypoglycemia is defined as a blood glucose level of 60 mg/dL or less. Significant signs and symptoms often are seen near 50 mg/dL. (Keep in mind that higher levels are listed as the treatment threshold in the REMAC protocols because of concerns regarding glucose monitor accuracy and the potential for falsely high readings.)

The clinical presentation of hypoglycemia can take many forms. If not treated, all eventually lead to unconsciousness, seizures, and possibly death. Although most of the body's cells can withstand a drop in blood glucose level, the brain is dependent on - and very sensitive to - glucose levels. This explains why many of the signs and symptoms of hypoglycemia reflect neurologic changes and deficiencies. Whether you are a BLS or ALS provider, recognition of the signs and symptoms of hypoglycemia is important to ensure proper evaluation (including the use of a glucometer by ALS providers) and proper, timely treatment.

The signs and symptoms of hypoglycemia may include any of the following:

- Hunger
- Nausea
- Weakness
- Tachycardia, weak, rapid pulse
- Pale, cool, clammy skin
- Seizures
- Altered mental status:
  - Agitated
  - Confused
  - Combative
  - Lethargic
  - Unresponsive

In the field, a standard practice for advanced life support providers in verifying suspected hypoglycemia is the determination of blood glucose. Whenever possible, determination of a patient's blood glucose level will confirm that the clinical findings are consistent with hypoglycemia.

1. Take appropriate infection control precautions.
2. Verify patient condition warrants use of device.
3. Verify that device has been calibrated and is within normal testing limits.
4. Select a finger that will result in proper sampling.
5. Turn on unit and follow the manufacturer's instructions.
6. Clean fingertip with alcohol pad and allow to dry.
7. Use sterile lancet or lancet pen on the side of selected finger.
8. Penetrate skin to obtain a hanging drop of blood.
9. Apply blood to pad according to the manufacturer's instructions.
10. Properly position testing strip into device according to the manufacturer's instructions.
11. Properly dispose of lancet.
12. Correctly read and interpret findings.
13. Properly dispose of testing strip.
14. Place sterile dressing on penetrated finger as necessary.
15. Record blood glucose test result.
Treatment: We treat hypoglycemia in accordance with the NYC REMAC Protocols. Treatment of the patient with hypoglycemia always involves measures designed to increase the blood glucose level. Three basic methods are used to accomplish this.

The least invasive and most available method is to have the patient ingest glucose in the form of a glucose paste applied intra-orally (buccally) or in the form of tablets that dissolve in the mouth. Food sources, such as orange juice or hard candy, can be used if other sources are unavailable, however, these food sources often are not glucose (e.g., orange juice contains fructose) and therefore do not work as well or as fast. In addition, they may pose an aspiration risk if the patient cannot protect his / her airway or loses consciousness. Therefore, if a source of glucose is to be administered orally, the patient must be able to manage his / her own airway (i.e., has a gag reflex, is able to swallow, and controls secretions).

The patient's level of consciousness must be monitored at all times. If the patient cannot manage his / her airway, or if the level of consciousness deteriorates, intravenous (IV) dextrose is the treatment of choice. IV dextrose works almost immediately by increasing the patient's blood glucose level, and, as it is given IV, it can be administered regardless of the patient's airway status or level of consciousness. Attention should be given to ensure that the IV line is patent because dextrose 50% is hypertonic and can cause tissue necrosis if extravasation occurs.

If the patient is not a candidate for oral glucose and patent vascular access is not possible, glucagon is the last option. As previously mentioned, glucagon increases blood glucose levels by initiating the breakdown of glycogen (stored glucose) in the liver. However, patients who do not have adequate glycogen stores in the liver will not benefit from glucagon administration (as you cannot release what you do not have in the first place). Common reasons for inadequate liver glycogen stores are chronic alcoholism or malnutrition, young age, chronic illness, history of seizures, or severe trauma.

In all cases, when the patient's blood glucose level increases, an immediate decrease or disappearance of signs and symptoms should occur.

All patients need monitoring after treatment because the longevity of treatment is directly related to the level of imbalance of insulin in the patient's system, and a return of hypoglycemia is common.

Simply correcting the signs and symptoms is not enough, and attempts should be made to determine the cause of the hypoglycemic episode. Causes other than failure to eat after DM medication administration or administration of excess insulin should be considered and explored (e.g. infection, especially in the young and elderly, myocardial infarction, or medication interactions).

Despite the prehospital treatment and resolution of signs and symptoms, patients taking long-acting oral hypoglycemic medications (e.g., sulfonylureas) or long-acting insulins (e.g., Lantus) may experience recurrent hypoglycemia and must be transported to the hospital for further monitoring and evaluation. If the patient refuses transport and the RMA is approved by OLMC, the patient should be encouraged to ingest a more complex source of carbohydrates, such as peanut butter and crackers. This will help prevent the recurrence of hypoglycemia, since one ampule of dextrose 50% contains only 100 calories (25 grams of dextrose x 4 calories per one gram of dextrose) which will be rapidly consumed.

Hyperglycemia
Hyperglycemia is almost always a result of diabetes mellitus. By itself, and at a level lower than approximately 250 mg/dL, it represents no immediate life threat. In fact, many of the Type 2 DM patients may actually have the disease (and the hyperglycemia that it causes) for years prior to being diagnosed, and yet the majority do not develop any complications during that time. It does, however, cause several physiologic changes that have detrimental long-term effects. Hyperglycemia puts undue strain on the cardiovascular system, kidneys, and other end organs. The result over time is seen as increased incidence of disorders such as renal failure, congestive heart failure, retinopathy, coronary artery disease, and neuropathy.

The signs and symptoms of simple hyperglycemia are usually mild, if present at all. They can include blurred vision, polyuria, polydipsia, polyphagia, orthostatic syncope, frequent infections, prolonged wound healing, and skin ulcerations. Treatment of patients with simple hyperglycemia that has not progressed to the more serious syndromes covered in the following paragraphs should include supportive care and transport.

When serum glucose levels rise above tolerable levels, other physiologic changes occur. These changes represent actual pathologic conditions called diabetic ketoacidosis (blood glucose above 350 mg/dL, approximately, and usually in Type 1 DM patients) and hyperosmolar hyperglycemic nonketotic coma (blood glucose levels greater than 600 mg/dL, approximately, and usually in Type 2 DM patients).

**Diabetic Ketoacidosis**

Diabetic ketoacidosis (DKA) is a life-threatening emergency that results from persistent hyperglycemia caused by complete-lack-of or too-little insulin in the body. This results in elevated blood sugar level, excessive breakdown of the body's energy stores, and the resulting accumulation of acids in the body that cause dehydration, electrolyte abnormalities, and metabolic acidosis. It can occur in the following DM patients:

- Patients who are newly diagnosed with Type 1 DM (this may be their first presentation).
- Patients who have an increased release of glycogen in response to stress or illness and do not adjust their insulin intake accordingly.
- Patients with poorly controlled DM.
- Patients with dietary indiscretions.
- Patients who fail to take their insulin.

DKA is a complex metabolic problem that can be boiled down to three simultaneous processes: 1) A lack of insulin leads to an inability of the cells to obtain the glucose that they need to survive. 2) Despite the fact that there is adequate glucose in the blood (in fact, there is too much), the cells are not able to use this glucose in the absence of adequate insulin, and so they act as though the body is hypoglycemic (because the cells are) and stimulate the release of more glucose into the bloodstream. 3) Low insulin levels trigger the breakdown of fats and proteins in the body as an alternative to glucose. The breakdown of fat results in the production of ketones, and the breakdown of protein creates lactate. 4) Both ketones and lactate cause a drop in the pH of the blood (making it more acidic). The result is a high blood sugar level, ketones in the blood, and acidosis – diabetic ketoacidosis.

In patients with Type 2 DM, DKA is rare because insulin is still present, at least early in the disease, and, despite elevated blood sugar, the level of insulin is enough to prevent uncontrolled breakdown of glycogen, fat, and proteins. However, with
increased duration of Type 2 DM, a loss of pancreatic insulin production may occur and patients may develop DKA, similar to those patients with Type 1 DM.

**History and Physical Findings:** Patients have symptoms of dry mucous membranes, orthostatic hypotension, supine hypotension, fatigue, tachycardia and increased thirst (polydipsia) due to dehydration. The hyperglycemia results in excess glucose entering the urine, taking more fluid with it, and resulting in increased urination (polyuria). The patients will hunger will be stimulated (polyphagia) by the cells desire for glucose, and the patient will experience abdominal pain and vomiting (from the acidosis). In time, the dehydration will become so severe that the patient will develop altered mental status and, with time, weight loss from the hypermetabolic state. The patient’s respiratory rate is usually elevated and the tidal volume is increased (*Kussmaul's respirations – deep, sighing respirations*) because of ketonemia, acidosis, and the body's attempt to relieve itself of acid in the form of CO2. A fruity odor to the patient's breath is consistent with ketones as well.

**Therapeutic Interventions:** Treatment of DKA includes fluids and insulin. The replacement of losses must be slow. The majority of patients with DKA become ill and dehydrated over a prolonged period - days (and sometimes up to weeks). The replacement of fluids also should be gradual to prevent complications of overaggressive treatment. Therefore carefully consider bolus administration with fluids based on the patient’s cardiovascular status. If the patient is hypotensive, rapidly administer isotonic fluids until the systolic pressure is 90 mmHg, and then slow the infusion. As with any patient in whom fluid boluses are being administered, closely monitor the patient for the development of dyspnea and pulmonary edema.

Unlike patients with acute hypovolemia, there is reason for caution with IV fluids in DKA, where the dehydration developed more slowly. The elevated osmolality of the serum from hyperglycemia results in a prolonged and progressive shrinking (*crenation*) of the body's cells as water is pulled into the hypertonic intravascular compartment. The rapid administration of intravascular fluids promotes the rapid movement of fluid into the cells of the body and increases the risk for edema. Of particular concern is cerebral edema, which can lead to changes in mental status, coma, and death. Therefore, neurologic status monitoring is important. This is particularly true in pediatric patients, where IV fluid boluses should be limited to 20 mL/kg at a time with fluids such as normal saline or Ringer's lactate solution. These IV fluid boluses should not be repeated unless significant conditions exist. In adults, 1 to 1.5 L is typically given over the first hour.

The use of NaHCO3 (sodium bicarbonate) to treat the metabolic acidosis of DKA is controversial and generally reserved for when the blood pH is less than 7.0. In general, its use has no role in the prehospital management of DKA. This is because the serum pH must be closely monitored. Arbitrary administration of sodium bicarbonate without exact knowledge of the serum pH can cause a paradoxical acidosis in the cerebrospinal fluid as well as several other complications that may worsen the patient's condition and outcome16.

Hypermagnesemia is commonly associated with DKA as a result of the loss of magnesium through the urine. This can worsen vomiting and cause alterations in mental status, as well as induce other electrolyte abnormalities, and lead to a number of cardiac dysrhythmias (atrial fibrillation, supraventricular tachycardia, PVCs), including several which may be fatal (ventricular tachycardia, Torsades de Pointes, ventricular fibrillation). Magnesium deficiency from DKA is generally not
addressed in the prehospital setting; however, if severe dysrhythmias occur in the setting of suspected DKA, the use of magnesium should be considered in consultation with OLMC.

As the body attempts to buffer the acidosis by shifting hydrogen ions into the cells, potassium shifts out of the cells in exchange resulting in an associated increase of relative serum potassium levels, emphasizing the need to monitor the patient's electrocardiogram. The monitoring of the potassium level can be assisted by evaluation of T waves, P waves, and width of the QRS complex on the electrocardiographic pattern. Children are less likely than adults to have cardiovascular complications as potassium rises. However, this does not preclude the need for closely monitoring the cardiovascular status of all patients with DKA.

<table>
<thead>
<tr>
<th>Serum potassium</th>
<th>Typical ECG appearance</th>
<th>Possible ECG abnormalities</th>
</tr>
</thead>
</table>
| Mild (5.5-6.5 mEq/L) | ![Mild ECG](image) | Peaked T waves  
Prolonged PR segment                                         |
| Moderate (6.5-8.0 mEq/L) | ![Moderate ECG](image) | Loss of P wave  
Prolonged QRS complex  
ST-segment elevation  
Ectopic beats and escape rhythms                                  |
| Severe (>8.0 mEq/L) | ![Severe ECG](image) | Progressive widening of QRS complex  
Sinus wave  
Ventricular fibrillation  
Asystole  
Axis deviations  
Bundle branch blocks  
Fascicular blocks                                                         |

**Figure 4: Electrocardiographic (ECG) Manifestations of Hyperkalemia**

The treatment of DKA in the prehospital setting begins with simple IV fluid management and transport to the hospital, and this is typically enough to establish significant improvement of the hyperglycemia as well as help correct the metabolic acidosis. Additionally, close monitoring of the patient’s vital signs (including blood pressure, heart rate, respiratory rate, pulse oximetry) and recurrent assessments of the patient’s respiratory, cardiovascular, and neurologic status are imperative. Finally, utilizing the newest ALS protocol, severe nausea and vomiting can be treated with ondansetron (Zofran).

**Hyperosmolar Hyperglycemic Nonketotic Coma**

Hyperosmolar hyperglycemic nonketotic coma (HHNC) is the result of elevated glucose from poor or little insulin action. It is typically described in a patient with Type 2 DM; however, it has been reported in children with Type 1 DM.

Similar to DKA, secretion of hormones is exaggerated. This results in an increase of the blood glucose level because of mobilization of glucose stores. However, patients with hyperosmolar hyperglycemia have some insulin action remaining compared to those with DKA. Therefore the breakdown of fatty acids and excessive formation of ketone bodies is less
dramatic. These patients have smaller amounts of ketones present in the urine and serum than do patients with DKA, but they have higher blood sugar levels (often much higher) because of excessive hormone action and insulin resistance. Glucose is present in the urine and dehydration occurs because of elevated serum osmolality and free water losses in an effort to rid the body of sugar.

HHNC generally occurs in elderly patients, who may have decreased renal function and a decrease in the ability to eliminate glucose. This, along with insulin resistance, leads to the extremely elevated glucose levels (more than 600 mg/dL) associated with the condition. Because of the insulin resistance associated with HHNC, insulin therapy often is not effective, leading to a mortality rate as high as 50%-70%.

**History and Physical Findings:** The clinical presentation of these patients is similar to the dehydration component of DKA. The acidotic signs and symptoms, such as fruity breath and Kussmaul's respirations, are absent. The time of onset also differs between HHNC and DKA: DKA may occur in a matter of hours to days; whereas HHNC has a more insidious onset and can take days to weeks. Signs and symptoms manifest themselves through severe volume depletion and the central nervous system. These include warm, dry skin, dry mucous membranes, poor skin turgor, tachycardia, weakness, polyuria, polydipsia, polyphagia, orthostatic hypotension, supine hypotension, altered mental status, lethargy, coma, and possibly seizures.

**Therapeutic Interventions:** The treatment is as described for DKA, and includes fluid therapy and insulin, with close monitoring of vital signs and respiratory, cardiovascular, and neurologic status.

**CONCLUSION**

Thorough understanding of the spectrum and mechanism-of-disease of diabetes mellitus will ensure proper identification and prompt treatment of diabetic emergencies in the prehospital setting. Hypoglycemia, when suspected, should be verified with a glucometer (if ALS is on scene) and aggressively managed in order to avoid its complications. Hyperglycemia, when it manifests as either DKA or HHNC should be managed with fluid resuscitation, antiemetics, and (in the case of suspected DKA) cardiac monitoring. Familiarity with these conditions and their signs and symptoms will allow for accurate detection, timely management, and appropriate disposition (including recommended OLMC contact for all RMAs when these conditions are suspected).

Written by: Dr. David Ben-Eli  
*Office of Medical Affairs*
1. The term diabetes mellitus is Latin for:
   a. sweet blood
   b. sweet urine
   c. sweet sweat
   d. sweetbreads
   e. high sugars

2. Which of the following is not a long-term complication of DM?
   a. heart disease
   b. stroke
   c. hypoglycemia
   d. renal failure
   e. blindness

3. Mild to moderate hypoglycemia is:
   a. rare
   b. completely preventable
   c. easily treated
   d. frequently results in complications
   e. unable to be detected with blood glucose monitoring
4. Common signs and symptoms of hypoglycemia include all of the following **except**:
   a. hunger
   b. nausea
   c. weakness
   d. bradycardia
   e. pale, cool, clammy skin

5. Signs and symptoms of hyperglycemia include:
   a. polyuria
   b. polydipsia
   c. polyphagia
   d. frequent infections
   e. all of the listed answers are correct

6. Though it differs from the threshold used in the REMAC protocols, hypoglycemia is typically defined as a blood glucose level of:
   a. 120 mg/dL or less
   b. 100 mg/dL or less
   c. 80 mg/dL or less
   d. 60 mg/dL or less
   e. 50 mg/dL or less

7. The following may be found in patients with DKA:
   a. hypoglycemia
   b. hypermagnesemia
   c. acidosis
   d. hypokalemia
   e. fluid overload

8. EKG findings of hyperkalemia in DKA include all of the following **except**:
   a. peaked T waves
   b. flattening of P waves
   c. U waves
   d. prolongation (widening) of QRS complex
   e. sine wave

9. The mainstay of prehospital treatment of DKA is:
   a. insulin IV
   b. sodium bicarbonate IV
   c. IV fluid hydration
   d. Metformin
   e. chewable aspirin

10. In hyperosmolar hyperglycemic nonketotic coma (HHNC):
    a. onset is faster than DKA
    b. patients are not dehydrated
    c. patients have fruity breath
    d. patients have Kussmaul's respirations
    e. patients may experience altered mental status
Journal CME Credit Answer Sheet

Based on the CME article, place your answers to the quiz on this answer sheet. Respondents with a minimum grade of 80% will receive 1 hour of Online/Journal CME.

Please submit this page only once, by one of the following methods:

• FAX to 718-999-0119 or
• MAIL to FDNY OMA, 9 MetroTech Center 4th flr, Brooklyn, NY 11201

Contact the Journal CME Coordinator at 718-999-2790:

• three months before REMAC expiration for a report of your CME hours.
• for all other inquiries.

Monthly receipts are not issued. You are strongly advised to keep a copy for your records.

Note: if your information is illegible, incorrect or omitted you will not receive CME credit.

check one: □ EMT □ Paramedic □ other

Name

NY State / REMAC # or “n/a” (not applicable)

Work Location

Phone number

Email address

Submit answer sheet by the last day of this month.

October 2011 CME Quiz

<table>
<thead>
<tr>
<th></th>
<th></th>
<th>Required for BLS &amp; ALS providers</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>2.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>3.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>4.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>5.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>6.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>7.</td>
<td></td>
<td>Required for ALS providers only</td>
</tr>
<tr>
<td>8.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>9.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>10.</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
### Citywide CME – October 2011

*Sessions are subject to change without notice. Please confirm through the listed contact.*

<table>
<thead>
<tr>
<th>Boro</th>
<th>Facility</th>
<th>Date</th>
<th>Time</th>
<th>Topic</th>
<th>Location</th>
<th>Host</th>
<th>Contact</th>
</tr>
</thead>
<tbody>
<tr>
<td>BK</td>
<td>Kingsbrook</td>
<td>TBA</td>
<td>TBA</td>
<td>TBA: call to inquire →</td>
<td>ED Conference Room</td>
<td>Dr Hew</td>
<td>Manny Delgado 718-363-6644</td>
</tr>
<tr>
<td></td>
<td>LICH</td>
<td>TBA</td>
<td>1200-1500</td>
<td>TBA: call to inquire →</td>
<td>Avram Conference Room &quot;G&quot;</td>
<td>Dr Vlasica</td>
<td>Aaron Scharf 718-780-1859</td>
</tr>
<tr>
<td></td>
<td></td>
<td>TBA</td>
<td>0900-1200</td>
<td>TBA: call to inquire →</td>
<td>Avram Conference Room &quot;A&quot;</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Lutheran</td>
<td>4th Wed</td>
<td>1730-1930</td>
<td>Call Review RSVP →</td>
<td>Call for location →</td>
<td>Dr Chitnis</td>
<td>Dale Garcia 718-630-7230</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td><a href="mailto:dgarcia@lmcmc.com">dgarcia@lmcmc.com</a></td>
</tr>
<tr>
<td>MN</td>
<td>NY Presbyterian</td>
<td>TBA</td>
<td>TBA</td>
<td>TBA: call to inquire →</td>
<td>Weill Cornell Campus A-950</td>
<td>Dr Ewy</td>
<td>RSVP: <a href="mailto:ssamuels@nyp.org">ssamuels@nyp.org</a> Ana Doulis 212-746-0885 x2</td>
</tr>
<tr>
<td></td>
<td>NYU School of Medicine</td>
<td>TBA</td>
<td>TBA</td>
<td>TBA: call to inquire →</td>
<td>Schwartz Lecture Hall 401 E 30 Street</td>
<td>TBA</td>
<td>Jessica Kovac 212-263-3293</td>
</tr>
<tr>
<td>QN</td>
<td>FDNY-BOT</td>
<td>Cancelled until further notice</td>
<td>---</td>
<td>---</td>
<td>---</td>
<td>---</td>
<td>---</td>
</tr>
<tr>
<td></td>
<td>NYH Queens</td>
<td>Thursdays</td>
<td>0800-0900</td>
<td>Call Review/Trauma Rounds</td>
<td>East bldg, courtyard flr</td>
<td>Dr Sample</td>
<td>Mary Ellen Zimmermann RN 718-670-2929</td>
</tr>
<tr>
<td></td>
<td>Mt Sinai Qns</td>
<td>last Tues</td>
<td>1800-2100</td>
<td>Lecture or Call Review</td>
<td>25-10 30 Ave, conf room</td>
<td>Dr Dean</td>
<td>Donna Smith-Jordan 718-267-4390</td>
</tr>
<tr>
<td></td>
<td>Parkway Hosp</td>
<td>3rd Wed</td>
<td>1830-2130</td>
<td>Call Review</td>
<td>Board Room, 1st flr</td>
<td></td>
<td><a href="mailto:pabruzzino@capitolhealthmgmt.com">pabruzzino@capitolhealthmgmt.com</a></td>
</tr>
<tr>
<td></td>
<td>Queens Hosp</td>
<td>2nd Thurs</td>
<td>1615-1815</td>
<td>Call Review</td>
<td>Emergency Dept</td>
<td></td>
<td>718-883-3070</td>
</tr>
<tr>
<td></td>
<td></td>
<td>4th Thurs</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SI</td>
<td>RUMC</td>
<td>TBA</td>
<td>1400</td>
<td>TBA: call to inquire →</td>
<td>MLB conf room</td>
<td>TBA</td>
<td>William Amaniera 718-818-1364</td>
</tr>
</tbody>
</table>
## 2011-2012 NYC REMAC Examination Schedule

<table>
<thead>
<tr>
<th>Month</th>
<th>REMAC Refresher Exam (Written only - CME letter required)</th>
<th>REMAC Basic Exam (Written &amp; 3 Orals Scenarios)</th>
<th>NYS/DOH Written Exam</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Registration Deadline</td>
<td>Exam Date (on Wednesdays)</td>
<td>Registration Deadline</td>
</tr>
<tr>
<td>October 2011</td>
<td>9/30/11</td>
<td>10/26/11</td>
<td>Thursday 10/6/11</td>
</tr>
<tr>
<td>November</td>
<td>10/31/11</td>
<td>11/16/11</td>
<td>Wednesday 11/17/11</td>
</tr>
<tr>
<td>December</td>
<td>11/30/11</td>
<td>12/21/11</td>
<td>Wednesday 11/28/11</td>
</tr>
<tr>
<td>January 2012</td>
<td>12/31/11</td>
<td>1/25/12</td>
<td>Wednesday 1/4/12</td>
</tr>
<tr>
<td>February</td>
<td>1/31/12</td>
<td>2/22/12</td>
<td>Wednesday 2/22/12</td>
</tr>
<tr>
<td>March</td>
<td>2/29/12</td>
<td>3/21/12</td>
<td>Wednesday 2/29/12</td>
</tr>
<tr>
<td>April</td>
<td>3/31/12</td>
<td>4/25/12</td>
<td>Wednesday 3/31/12</td>
</tr>
<tr>
<td>May</td>
<td>4/30/12</td>
<td>5/16/12</td>
<td>Wednesday 5/9/12</td>
</tr>
<tr>
<td>June</td>
<td>5/31/12</td>
<td>6/20/12</td>
<td>Wednesday 5/9/12</td>
</tr>
<tr>
<td>July</td>
<td>6/30/12</td>
<td>7/25/12</td>
<td>Wednesday 7/4/12</td>
</tr>
<tr>
<td>August</td>
<td>7/31/12</td>
<td>8/22/12</td>
<td>Wednesday 7/24/12</td>
</tr>
<tr>
<td>September</td>
<td>8/31/12</td>
<td>9/19/12</td>
<td>Wednesday 8/29/12</td>
</tr>
</tbody>
</table>

The **REMAC Refresher Written examination** is offered monthly for paramedics who meet CME requirements and whose REMAC certifications are either current or expired less than 30 days. To enroll, call 718-999-7074 before the register registration deadline above. Candidates may attend an exam no more than 6 months prior to expiration. Refresher exams are held at 07:00 or 18:00 hours at FDNY-EMS Bureau of Training, Fort Totten, Queens.

The **REMAC Basic Written & Orals examination** is for initial certification, or for inadequate CME, or for certifications expired more than 30 days. Registrations must be postmarked by the deadline above. Email swansoc@fdny.nyc.gov instructions. You are encouraged to register at least 30 days prior to the exam as seating is limited. A $100 exam fee by money order is required.