New feature: Ask the Academy  The Journal CME Newsletter is pleased to announce a new feature in upcoming issues. FDNY paramedic instructor James “Bubba” Fallar will research and respond to your questions regarding prehospital care. You can write to Bubba directly at fallarj@fdny.nyc.gov Please keep your correspondence professional. We look forward to reading your questions.

Effective August 1, 2006, NYC REMAC has issued protocol revisions that may be implemented after medics are updated by their ambulance service. All EMS personnel must be updated by January 1, 2007. Per REMAC, ambulance services in NYC are responsible to provide copies of the protocols to their personnel. The REMAC Advisories are available to all at www.nycremsco.org

The REMAC Certification exams will not include questions reflecting the protocol changes until after January 1, 2007.

Questions may be referred to the REMAC Liaison at 718-999-2671 or swansoc@fdny.nyc.gov

Outline of August 2006 protocol changes:

- General Operating Procedures: Stroke Patient Criteria has been added
- 403 Non Traumatic Cardiac Arrest: updated to meet AHA Guidelines
- 412 Stroke (CVA): GCS replaced by Stroke Patient Criteria
- 414 Poisoning or Drug Overdose: deletion of Syrup of Ipecac
- 453 Pediatric Non Traumatic Cardiac Arrest: updated to meet AHA Guidelines
- Series 503 Non Traumatic Cardiac Arrest:
  - Asystole and PEA protocols are combined
  - updated position of Defibrillator pads – one (1) inch from permanent pacemaker
  - deletion of High Dose Epinephrine, Lidocaine, Dextrose, Narcan, Dopamine, TCP
  - addition of Calcium Chloride
- Series 505 Cardiac Dysrhythmias:
  - updated joule settings for Cardioversion, and doses for Atropine, Epinephrine and Dopamine
  - deletion of Lidocaine
  - addition of Calcium Chloride
- 511 AMS: Naloxone may be administered via intra nasal (IN) route using an atomizer
- 521 Head Injuries: changed sequence of Standing Orders; Prehospital Sedation included for ET
- Appendix D AED Guidelines: updated to meet AHA Guidelines
- Appendix R Stroke Patient Criteria: has been created
REMAC Refresher (written) examinations are held monthly, and may be attended up to 6 months before your expiration date. Call the Registration Hotline @ 718-999-7074.

REMAC Challenge (written and orals) examinations are held every January, April, July & October. Registration is limited to the first 50 applicants. The next available Challenge examination is scheduled for October 17 & 24. By September 17, call 718-999-2671 to register.

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

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**Important Certification & CME Info**

- **Effective January 1, 2007:** 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, Emergency Department Teaching Rounds, OLMC Rotation
  - **36 hours – Alternative Source CME – Maximum of 12 hours per Venue**
    - Online CME
    - Lectures/Symposiums/ Conferences
    - BCLS / ACLS / PALS / NALS / PHTLS

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**ePCR TIPS OF THE MONTH**

- Only use the medical codes 97 (“Not listed”) or 98 (“Unknown Medical”) when there is no other way to describe the condition.

- Always use the Body Matrix section when appropriate for either medical or injury.

- Use of secondary confirmation devices for every intubated patient is mandated by the state. Remember to darken the "ETT secondary confirmation" box on the ePCR.
FDNY ALS Division Coordinators

- **Citywide ALS:** 718-999-1738
  Lt. Patrick Dillon
- **Division 1:** 212-964-4518
  Paramedic Andrea Katsanakos
- **Division 2:** 718-829-6069
  Paramedics Steve Pilla & Cesar Escobar
- **Division 3:** 718-968-9750
  Paramedics Gary Simmonds & Al Navarro
- **Division 4:** 718-883-2150
  Paramedic Lisa DeSena
- **Division 5:** 718-979-7175
  Paramedic Russell Shewchuk
- **Bureau of Training:** 718-281-8325
  Paramedic James "Bubba" Fallar
- **EMS Pharmacy:** 718-571-7620
  Paramedic Anthony Esposito

FDNY Division Medical Directors

- **Division 1 and OLMC:** 718-999-2665
  Dr. John Freese
- **Division 2 and BOT:** 718-281-8473
  Dr. Dario Gonzalez
- **Division 3 and 5:** 718-999-2666
  Dr. Glenn Asaeda
- **Division 4:** 718-999-1872
  Dr. Bradley Kaufman
- **EMS Fellows:**
  - Dr. David Ben-Eli 718-999-2670
  - Dr. Doug Isaacs 718-999-0749

FDNY OLMC Physicians and ID Numbers

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REMSCO website: [www.NYCREMESCO.org](http://www.NYCREMESCO.org)


News & Information from the EMS Bureau of Training

EMS Training Supervision

Chief of EMS Training: Division Chief James P. Martin
Executive Officer EMS Training: Deputy Chief Scott C. Holliday
Commanding Officer EMS Academy: Captain John Quigley
ALS Program Coordinator: Lieutenant Andres Rodriguez
Tour 2 BLS Program Coordinator: Lieutenant Lillian Bonsignore
Tour 2 Program Coordinator: Lieutenant John Scotch
Tour 3 BLS Program Coordinator: Lieutenant Arthur Lester
Tour 3 ALS Program Coordinator: Lieutenant David Russell
EVOC Program Coordinator: Lieutenant Robert Raheb
Haz Tac Training Coordinator (detailed): Lieutenant John Ryan

Important Contact Numbers for the Bureau of Training

EMS Academy Main Number: 718-281-8325
EMS Academy Fax: 718-352-3954
CFR Training: 718-281-8405
EVOC Training: 718-281-8317

Haz Tac Training: 718-281-8310
EMS Training Administration: 718-281-8460
Training Schedule Coordinator: 718-281-8466
Registrar: 718-281-8467

Academic Schedule – Summer 2006

Paramedic Basic Program: Paramedic Basic Program 5 has 45 EMTs and 5 officers in class. This program finishes in November. Medic Basic 6 will start in September of 2006 and finish in May 2007.

Trainee Orientation Program: Paramedic TOP class has 5 medics in it and the two TOP classes currently have 75 students in them. All TOP classes will be finished on September 15.

BLS Programs: The EMT refresher programs will resume on September 18.

ALS Programs: Paramedic refresher programs will begin again on September 5.

Haz Tac Programs: Haz Mat Operations for all non-HazTac personnel will be starting in September.

CFR Programs: The staff of the CFR Unit continues to train and refresh FDNY firefighters in the CFR-D program. Original, refresher and probationary firefighter training is on going.

EVOC: Due to the construction of the new EVOC field there is limited parking available at the Academy as the staff and student parking areas is being utilized for EVOC training. This is scheduled to continue until December 1, 2006. Members attending training at the Academy this fall should seek alternate means of transport or the use of car pools.
New Protocols, New Questions – Part 2: Vasopressin

Last month, we began a series of articles designed to discuss the most recent changes to the New York City REMAC protocols. This month, we continue that series with a look at the drug vasopressin and include a brief discussion of some of the other changes to the cardiac arrest protocols. Coming articles will discuss amiodarone, calcium chloride, continuous positive airway pressure, and carbon monoxide monitoring.

Vasopressin – Pharmacology

Vasopressin (Pitressin), otherwise known as arginine vasopressin or antidiuretic hormone (ADH), was first described in 1895 and is a peptide that exists naturally in the body - produced by the hypothalamus and released by the pituitary. The physiologic action of vasopressin occurs via its activation of V1 receptors distributed throughout the vasculature.

As shown in the adjacent diagram, this V1 stimulation produces an increase in the levels of calcium within the vascular smooth muscle, leading to muscle contraction / vasoconstriction. However, within the brain this vasoconstriction is overridden by simultaneous release of nitrous oxide and its potent vasodilator effects. This combination of increased systemic vasoconstriction and cerebral vasodilation would result in increased cerebral blood flow.

When administered via the intravenous route, the onset of vasopressin’s action occurs within 30 seconds. The duration of action, which is somewhat longer than epinephrine, is estimated to be four minutes.

Vasopressin versus Epinephrine

A theoretical comparison of these two drugs would seem to suggest an advantage to the administration of epinephrine. While vasopressin results in direct vasoconstriction via the V1 receptor, epinephrine stimulates multiple components of the vasculature. Epinephrine’s action on the alpha, beta-1, and beta-2 receptors may all produce physiologic effects beneficial to the cardiac arrest patient.

In the intravenous doses used in the setting of a cardiac arrest, stimulation of alpha-1 receptors comprises the majority of epinephrine’s action and produces significant vasoconstriction. In addition, beta-1 stimulation results in inotropic, chronotropic, and dromotropic effects, resulting in an increase in both heart rate and contractility. Finally, and particularly in those patients whose arrest is secondary to some form of reactive airway disease or bronchospastic process, the beta-2 effects of epinephrine result in significant bronchodilation.
The potential detriment to epinephrine’s mechanisms lies particularly with the beta-1 effects. Given that a significant portion of cardiac arrests result from myocardial ischemia, increases in heart rate and myocardial contractility are certain to result in increased myocardial oxygen demand and worsening myocardial ischemia. Following the return of spontaneous circulation (ROSC), this effect may contribute to post-resuscitation myocardial dysfunction. Vasopressin, on the other hand, does not cause beta-1 stimulation and therefore avoids these associated adverse effects.

This is not to say that vasopressin is without adverse effects. Because of its potent vasoconstricting properties, the drug results in constriction of the coronary vessels as well. While blood flow may change minimally or even increase in the largest of the coronary vessels, one study found that smaller branch vessels within the coronary circulation experienced a nearly three-fourths (72%) increase in resistance following the administration of intravenous vasopressin. In addition, V1 receptors on the surface of platelets are also activated leading to increased platelet aggregation, an action that may worsen coronary perfusion in the setting of plaque rupture within a coronary artery.

To examine the role of vasopressin in the management of cardiac arrest, we will address each cardiac arrest scenario and the available, related literature for its use in the setting of refractory ventricular fibrillation, pulseless electrical activity, and asystole.

**Vasopressin in Ventricular Fibrillation**

The most effective treatment for ventricular fibrillation (VF) is the delivery of an appropriately timed defibrillatory shock. And yet in not every case will VF be abolished by the initial defibrillatory efforts. For those patients, the administration of vasopressin is appropriate.

Several animal studies have shown a benefit of vasopressin in the treatment of VF, including some studies that demonstrate an advantage over epinephrine. The administration of vasopressin leads to increased coronary perfusion pressure and improved myocardial blood flow. Cerebral blood flow is also increased, even when compared to epinephrine, both in the resuscitation phase and following return of spontaneous circulation. Specific to VF, the drug results in increased waveform amplitude with higher amplitude VF being associated with an increased likelihood of successful defibrillation.

Human studies of vasopressin in VF are limited, but seem to suggest some benefit. A group of 40 patients with persistent post-shock VF demonstrated improved survival when vasopressin was used instead of epinephrine. Yet the largest study of vasopressin, which is described in greater detail below (see Vasopressin in asystole), failed to demonstrate an advantage of vasopressin or epinephrine, though an overall survival advantage for all cardiac arrests was seen when patients received vasopressin followed by epinephrine. Finally, when epinephrine and vasopressin are given in combination, they have been shown to produce a more rapid and sustained increase in cerebral perfusion pressure than either drug given alone.
Data related to the use of vasopressin in pulseless electrical activity (PEA) is limited. While some studies contain a subgroup of patients or animals in PEA, most of these groups are so limited in number as to limit the conclusions that can be drawn.

The most relevant animal model of PEA compared the administration of vasopressin to vasopressin combined with epinephrine. The results found no difference in the rate of ROSC, cerebral or coronary perfusion pressures, or myocardial blood flow, but it did find that the administration of both drugs led to a decrease in cerebral blood flow. Another study by the same group, directly comparing epinephrine to vasopressin, found similar results but found that vasopressin (alone) resulted in increased cerebral flow as compared to epinephrine (alone).

Human data describing vasopressin’s use in PEA are limited to subgroup analyses. These less than perfect scientific approaches have revealed no significant benefit when vasopressin is used either alone or in combination with epinephrine. That said, no study has demonstrated any harm when vasopressin is used in place of epinephrine, and one study showed a “trend” toward increased survival for patients receiving vasopressin followed by epinephrine.

Recent attention has been paid to the potential benefit of vasopressin in patients presenting to prehospital providers in asystole. In a 2004 article in the New York Times, the opening line began, “People with a hard-to-treat type of cardiac arrest are three times as likely to survive if they are given a drug called vasopressin than if they receive the standard emergency treatment, adrenaline…”

In a pediatric animal model, Voelckel and colleagues found a trend toward improved cerebral blood flow with a combination of vasopressin and epinephrine versus epinephrine alone. An adult model comparing the combination of vasopressin and epinephrine to both drugs alone found that the combination resulted in improved coronary perfusion pressure, ROSC rate, and short-term survival.

The largest study of vasopressin in cardiac arrest, which prompted the Times article mentioned above as well as a number of other lay press reports about the drug, found no difference between those patients that received only epinephrine and those that received vasopressin in place of the first epinephrine dose. But a subgroup analysis found that, among asystolic patients, receiving vasopressin significantly increased the likelihood of survival to hospital discharge. And so came articles such as those in the Times.

The table below shows some of the results of that study. If you compare the first and second columns, it is clear that vasopressin resulted in a significant increase in the number of asystole survivors (12 patients vs. 4 patients, or 4.7% vs. 1.5%). But if you continue down the table you will also notice
one other thing: the use of vasopressin followed by epinephrine resulted in a 1000% increase in the number patients who were discharged in a neurologically devastated state (severe disability, coma, or persistent vegetative state).

But this is not the only study to find an advantage of vasopressin followed by epinephrine in patients presenting in asystole. Two other human trials found an increased rate of ROSC and survival among asystolic patients who are initially given vasopressin followed by atropine.

Overall, vasopressin plus epinephrine does not appear to cause any harm and may in fact provide some benefit to asystole patients. This is particularly true for patients with prolonged down times, longer resuscitations, and those with more severe acidosis or ischemia.

### Vasopressin – Putting It All Together

In reality, no study has ever shown that the administration of any vasoactive substances (epinephrine, isoproterenol, dopamine, vasopressin) results in increased survival as compared to the administration of no vasoactive substances. But when CPR and other basic life support maneuvers fail to restore circulation, the use of such medications in an attempt to improve perfusion is still considered appropriate.

The current REMAC recommendations allow for the use of vasopressin (40 units) in place of the first dose of epinephrine. Based on the above physiologic effects, the goal of this order is to maximize the physiologic effects while minimizing the potential adverse effects. The administration of vasopressin attempts to restore perfusion while minimizing the adrenergic effects on the myocardium. If this fails to restore circulation, the subsequent administration of epinephrine allows the patient to then benefit from the combined physiologic effects of the two drugs. In addition, vasopressin may result in changes in the VF waveform that will allow for more successful defibrillation.

That said, it is imperative to recognize that the addition of vasopressin to all of the cardiac arrest protocols and its use in combination with epinephrine will not supplant the delivery of adequate chest compressions, appropriately limited ventilations, or the recognition and prompt treatment of reversible causes of asystole or PEA. As with all resuscitative efforts, the key to success among ALS interventions is the effective performance of the supporting BLS skills. But when combined, these new changes may

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<th>Variable</th>
<th>Vasopressin Group (n=379)</th>
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<td>37 (9.9)</td>
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<td>Good cerebral performance</td>
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result in more successful coronary perfusion, improved cerebral blood flow, and increased survival among our most critical of patients.

One final note… A potentially deadly mistake could be made if one is not careful as to which medication is given. As shown below, the packaging for vasopressin is somewhat similar to the packaging of thiamine, and in the heat of the moment, the wrong vial might easily be obtained. Certainly the accidental administration of thiamine to a cardiac arrest patient is unlikely to either benefit or harm the patient. But the administration of vasopressin to a living patient… well, suffice it to say that there is a reason that “death” is one of the listed adverse effects found in the vasopressin insert. Be careful out there!

Written by:  John Freese, MD
EMS Division 1 & OLMC Medical Director
Fire Department of New York

References
1. Which of the following best describes the drug classification of vasopressin?
   a. Sympathomimetic c. Class I antiarrhythmic e. None of the above
   b. Direct vasopressor d. Class IV antiarrhythmic

2. Physiologic effects of vasopressin administration include:
   a. Increased platelet aggregation
   b. Nitrous oxide (NO) release within the systemic vasculature
   c. Alpha-1 stimulation within the central nervous system
   d. Decreased levels of calcium within the vascular smooth muscle
   e. None of the above

3. In the setting of ventricular fibrillation (VF), vasopressin has been shown to:
   a. Decrease VF waveform amplitude d. Decrease coronary perfusion pressures
   b. Increase VF waveform amplitude e. More than one of the above
   c. Decrease cerebral blood flow

4. Vasopressin acts through which of the following receptors within the systemic vasculature?
   a. None of the following
   b. Alpha-1
   c. Alpha-2
   d. Beta-1
   e. Beta-2

5. The dose of vasopressin to be administered under REMSCO protocols is:
   a. 20 units
   b. 60 units
   c. 40 units
   d. 80 units
   e. “Whatever telemetry orders”

6. True or false: Vasopressin administration, in combination with epinephrine, results in a larger number of patients being discharged from the hospital without neurologic deficits.
   a. True
   b. False

7. For patients presenting in pulseless electrical activity, vasopressin has been shown to result in:
   a. Increased survival c. Equivocal survival e. Trend away from survival
   b. Decreased survival d. Trend toward survival

8. True or false: An adverse effect of vasopressin, as listed in the product information, is “death.”
   a. True
   b. False

9. Which of the following is not an alternative name for vasopressin?
   a. Arginine vasopressin
   b. Diuretic hormone
   c. Pitressin
   d. All of the above are correct
   e. None of the above are correct

10. Which of the following are not considered to be vasoactive substances?
    a. Epinephrine
    b. Vasopressin
    c. Isoproterenol
    d. Dopamine
    e. All of the above are correct

** DO NOT RETURN THIS PAGE – SEND ANSWER SHEET ONLY (page 11) BY SEPT. 30**
Journal CME Credit Answer Sheet

After reading the article, place your answers to the following quiz on this answer sheet.

Paramedics receiving a minimum grade of 80% will receive 1 hour of Online/Journal CME.

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

INTER-OFFICE MAIL (pony express): FDNY Office of Medical Affairs

Or  FAX: 718-999-0119

Or  U.S. MAIL: FDNY OMA, 9 MetroTech Center 4\textsuperscript{th} flr, Brooklyn, NY 11201

You are strongly advised to keep a copy for your records

*Required information to receive your CME

*Name

NY State / REMAC #

*Work Location

*Email address

September 2006 Journal CME
New Protocols - Vasopressin Quiz Answers

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Please submit by September 30, 2006
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<td>9/5/06</td>
<td>1930-2230</td>
<td>Call Review</td>
<td>Cherkasky Auditorium</td>
<td>Dr. Wollowitz</td>
<td>Don Cardone 718-763-8888 x506</td>
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<td>1930-2230</td>
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<td>Don Cardone 718-763-8888 x506</td>
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<td>1600-1900</td>
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<td>1800-2100</td>
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<td>FDNY-Station 16</td>
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<td>9/5/06</td>
<td>0800-1100</td>
<td>Call Review</td>
<td>ED Conference Rm 1st fl</td>
<td>Dr. Hew</td>
<td>Donell Harvin 212-523-6532</td>
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<td>1400-1600</td>
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<td>Donell Harvin 212-523-6532</td>
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<td>John Bray</td>
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<td>1030-1400</td>
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<td>REMAC Liaison 718-999-2671</td>
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<td>9/14/06</td>
<td>1400-1600</td>
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<td>FDNY-Station 45</td>
<td>Dr. Ben-Eli</td>
<td>ALS Coordinator 718-883-2150</td>
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<td>FDNY-Station 47</td>
<td>9/22/06</td>
<td>0700-0900</td>
<td>Call Review</td>
<td>FDNY-Station 47</td>
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<td>ALS Coordinator 718-883-2150</td>
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<td>QN</td>
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<td>1600-1800</td>
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<td>FDNY-Station 50</td>
<td>Dr. Cherson</td>
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<td>QN</td>
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<td>0830-1030</td>
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<td>ALS Coordinator 718-883-2150</td>
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<td>QN</td>
<td>NS-LIJ</td>
<td>9/13/06</td>
<td>0800-1600</td>
<td>Critical Care Transport</td>
<td>15 Burke Lane, Syosset</td>
<td>Center for EMS</td>
<td>Anthony Conrardy 516-719-5065</td>
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<td>QN</td>
<td>Parkway Hosp.</td>
<td>9/20/06</td>
<td>1830-2130</td>
<td>Call Review/Acute G.I Bleeds</td>
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<td>SVCMC-St Anthony</td>
<td>9/13/06</td>
<td>1700-2000</td>
<td>Lecture/Protocol Update</td>
<td>Board Room</td>
<td>Dr. Politi</td>
<td>Robert Hou 718-357-0500 x112</td>
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<td>SI</td>
<td>SVCMC-S.I.</td>
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<td>---</td>
<td>Call Review</td>
<td>MLB Conf.Room</td>
<td>Ian Swords</td>
<td>347-219-9391</td>
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# 2006 REMAC Examination Schedule

<table>
<thead>
<tr>
<th>Month</th>
<th>REMAC Challenge Exam</th>
<th>REMAC Refresher Exam</th>
<th>NYS/DOH Exam</th>
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<tbody>
<tr>
<td></td>
<td>Part A - Written Tuesdays@18:00</td>
<td>Part B – Orals Tuesdays@09:00</td>
<td>Exam Date (on Wednesdays)</td>
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<tr>
<td>January</td>
<td>1/17/06</td>
<td>1/24/06</td>
<td>1/25/06</td>
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<tr>
<td>February</td>
<td></td>
<td></td>
<td>2/15/06</td>
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<tr>
<td>March</td>
<td></td>
<td></td>
<td>3/22/06</td>
</tr>
<tr>
<td>April</td>
<td>4/18/06</td>
<td>4/25/06</td>
<td>4/26/06</td>
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<tr>
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<tr>
<td>July</td>
<td>7/18/06</td>
<td>7/25/06</td>
<td>7/26/06</td>
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<tr>
<td>August</td>
<td></td>
<td></td>
<td>8/16/06</td>
</tr>
<tr>
<td>September</td>
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<td>October</td>
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<td>10/25/06</td>
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<tr>
<td>November</td>
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<td></td>
<td>11/15/06</td>
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<tr>
<td>December</td>
<td>12/13/06</td>
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<td>11/1/06</td>
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</table>

Paramedics with current REMAC certification must register for upcoming refresher examinations by calling 718-999-7074 during the registration periods. All refresher exams are held at 07:00 or 18:00 hours at FDNY-EMS Bureau of Training, Fort Totten, Queens.

The REMAC Challenge Examination is offered quarterly. **Register early as seating is limited, and at least 30 days prior to the exam.** To take a Challenge Exam for expired REMAC or inadequate CME, call 718-999-2671 to register. Part A (written) is held at 18:00 and Part B (orals) at 09:00 at FDNY-EMS Bureau of Training, Fort Totten, Queens.

**Send correspondence to:** FDNY Office of Medical Affairs, Attn: REMAC Liaison, 9 MetroTech Center - 4th Floor, Brooklyn, New York 11201-3857
# 2007 REMAC Examination Schedule

<table>
<thead>
<tr>
<th>Month</th>
<th>REMAC Challenge Exam</th>
<th>REMAC Refresher Exam</th>
<th>NYS/DOH Exam</th>
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</thead>
<tbody>
<tr>
<td></td>
<td>Part A - Written @18:00</td>
<td>Part B – Orals @09:00</td>
<td>Exam Date</td>
</tr>
<tr>
<td>January</td>
<td>Wednesday 1/24/07</td>
<td>Wednesday 1/31/07</td>
<td>1/17/07</td>
</tr>
<tr>
<td>February</td>
<td>2/21/07</td>
<td>1/1/07</td>
<td>1/31/07</td>
</tr>
<tr>
<td>March</td>
<td>3/21/07</td>
<td>2/1/07</td>
<td>2/28/07</td>
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<tr>
<td>April</td>
<td>Thursday 4/19/07</td>
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<td>May</td>
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<td>June</td>
<td>6/20/07</td>
<td>5/1/07</td>
<td>5/31/07</td>
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<tr>
<td>July</td>
<td>Tuesday 7/24/07</td>
<td>Tuesday 7/31/07</td>
<td>7/18/07</td>
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<tr>
<td>August</td>
<td>8/15/07</td>
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<tr>
<td>November</td>
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