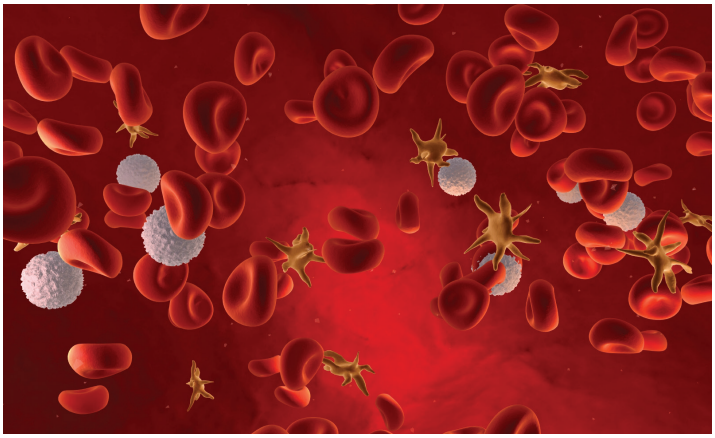
"Anticoagulants: Review"

October 2014

Anticoagulation is an ever increasing therapy consideration. Our goals in this lesson are to consider the clotting process & therapeutic options. The objectives of this lesson are such that upon completion:

Pharmacists will be able to:

1. Define "platelets" & state their role in hemostasis.
2. Describe the coagulation process.
3. Differentiate between thrombus, embolus, ischemia, & infarct.
4. List patients who are candidates for anticoagulants.
5. Describe the mechanisms of action of specific anticoagulants.



Technicians will be able to:

1. Define "platelets."
2. Define "hemostasis."
3. Define "thrombus, embolus, ischemia, & infarct."
4. Describe the rationale for using anticoagulant therapy.

This is a subscription program. To get continuing education credit, you must subscribe to the program, or pay fee for individual lessons.

OTHER 2014 TOPICS

Parenteral Nutrition

Pharmacy Law

CE PRN

PHARMACY CONTINUING EDUCATION FROM WF PROFESSIONAL ASSOCIATES

RENEWALS HAVE BEEN MAILED.
RESPOND NOW.

TO DOWNLOAD LESSONS!!!

- **Go to website www.wfprofessional.com**
- **Click on "COURSES."**
- **Click on "YEAR."**
- **Click on the ID #"707-000-……." for your lesson of interest.**

WHEN YOU SEND IN QUIZZES, ALWAYS KEEP A COPY. YOU MAY EMAIL OR FAX THEM. FAX # IS 847-945-5037. OR SEND A CONVENTIONAL EMAIL WITH YOUR ANSWERS TO CEINFO@WFPROFESSIONAL.COM

This lesson provides 1.25 hours (0.125 CEUs) of credit, and is intended for pharmacists and/or technicians in all practice settings. **Program ID #s 707-000-14-010-H01-P (for Pharmacists); 707-000-14-010-H01-T (for Technicians). Participants completing this lesson by September 30, 2017 may receive full credit.**

To obtain continuing education credit for this lesson, you must answer the questions on the quiz (70% correct required), and return the quiz. Should you score less than 70%, you will be asked to repeat the quiz. Computerized records are maintained for each participant.

If you have any comments, suggestions or questions, contact us at the above address, or call 1-847-945-8050. **Please write your ID Number (the number that is on the top of the mailing label) in the indicated space on the quiz page (for continuous participants only).**

CE PRN® (ISSN 0199-5006) is owned and published by W-F Professional Associates, Inc. 400 Lake Cook Road, Suite 207, Deerfield, Illinois 60015. William J. Feinberg, President. CE PRN® is published eleven times per year, monthly, January through November. Subscription rate is $110.00 per year. POSTMASTER: Send all address changes to W-F Professional Associates, Inc., 400 Lake Cook Road, Suite 207, Deerfield, IL 60015.

Anticoagulants, or blood thinners, are medications that are capable of preventing or reducing the risk of blood clotting. Blood thinning is the opposite of blood coagulation which is the process of causing the blood to clot. Blood clotting is vital in stopping bleeding, thereby enhancing wound healing. However, under certain conditions, blood in the circulation may form a life threatening clot that may lodge in the heart, brain, veins or arteries.

Blood is a constantly circulating body fluid that provides cells with nutrients and oxygen. Plasma is a liquid component of blood and makes up over half of total blood volume. About 5% of plasma consists of water and numerous blood cells and proteins such as albumin, globulin and fibrinogen, as well as glucose, clotting factors, electrolytes and hormones. Blood cells consist mainly of: red cells which transport oxygen to body tissue; white blood cells which protect the body from infection; and platelets which play a major role in blood clotting.

Platelets or thrombocytes are the smallest of the three blood cells. They are colorless, have no nuclei, but have a sticky surface. Each is about 2-3 µm in diameter, which is about 20% of the size of red blood cells. They are found in the bone marrow as a result of fragmentation of large nucleus cells or megakaryocytes. Once in the blood, these plates-shaped cells remain in the blood for about 5-10 days after which they enter the spleen where they are destroyed by phagocytes. Platelets are the source of growth factors such as hormones and cytokinesis. The main function of platelets is to contribute to blood clotting by sticking to the lining of the damaged blood vessels. When a blood vessel is injured, a signal is transmitted to the platelets which responds by transforming to the active form and rush to the site of injury. Once there, the platelets develop elongated hair-like structures. A normal adult platelet count is usually from 150,000 to 400,000 per microliter (mcL). A lower than normal count is called thrombocytopenia, and results in bleeding. Thrombocytopenia may be due to cancer, chemotherapy, hemolytic anemia, leukemia, and vitamin deficiency. Conversely, a higher than normal count is called thrombocytosis, and may occur as a result of anemia, polycythemia vera, chronic myelogenous leukemia and inflammation. Thrombocytosis increases the risk of blood clot formation in blood vessels, and may lead to strokes, infarcts, pulmonary emboli and blockage of blood vessels especially in the legs.

HEMOSTASIS (STOPPAGE OF BLEEDING)

When blood is lost following injury to blood vessels or from an unexplained reason, the body reacts to protect itself by initiating a pharmacologic process to stop the bleeding. This process is known as hemostasis. The endothelial cells of intact blood vessels do not allow blood clot formation due to the release of tissue plasminogen activator and to inactivating thrombin and adenosine diphosphate (ADP). Moreover, they prevent platelet aggregation by secreting nitric oxide and prostacycline. When injury occurs to blood vessels, the body reacts to prevent blood loss. In a healthy person, hemostasis or blood coagulation begins immediately after the injury to blood vessels and the endothelium. **There are three mechanisms that occur simultaneously to cause stoppage of bleeding.**

The first mechanism occurs as a result of the release of vasoconstrictors such as thromboxane A_2, which is produced by activated platelets, and by increased activity of new platelets and their ability to aggregate. Thromboxane also tends to constrict blood vessels during tissue damage and inflammation.

The second mechanism is platelet plug formation. This step follows vasoconstriction. Injury causes the exposure of blood to collagen fiber in the blood vessels. This results in gathering of platelets at the wound or damage sites to block blood flow by forming a plug at the site of

injury. The platelets become active and release ADP and thromboxane causing aggregation of more platelets around the wound.

The third mechanism is blood clotting or coagulation cascade which is a series of processes by the exposed collagen and clotting factors. The blood contains clotting factors that consist of a protein – phospholipid mixture present in the inactive form but are activated when blood vessels are injured. The activation process occurs in a step-wise manner. The completion of each step will activate another clotting factor until the conversion of fibrinogen to fibrin is complete. The first factor activates the second and the second activates the third until the process is complete. This sequence of reactions is known as clotting or coagulation cascade. It ultimately transforms blood from liquid to a semisolid state (clot).

A clot begins to form when blood with its platelets are exposed to air. The platelets begin to break apart and react with fibrinogen, an inactive precursor of fibrin, to form fibrin a gristly protein that resembles thin threads which polymerizes to form a mesh, a hemostatic plug or clot at the damaged site. This process requires the presence of the enzymes thrombin, calcium, vitamin K and clotting factors. If these elements are missing or deficient, a person with a serious wound could bleed to death. The function of calcium is to assist fibrin to from the web-like mesh that is stabilized by factor XII. The resultant fibrin net traps blood cells within it forming the clot that inhibits the flow of blood. The clot becomes hard as it dries up to form a scab. In the presence of vitamin K and calcium deficiencies, clotting time becomes prolonged. Internal blood clots may occur as bruises. A bruise or black-and-blue mark indicates internal superficial blood clot.

IMPAIRMENT OF CIRCULATING BLOOD

Under normal circumstances, blood should flow properly through arteries, veins and capillaries. However, any impediment in circulation could result in a number of pathologic conditions.

Thrombosis

This is the formation of a blood clot inside a vein or artery resulting in partial or total blockage of circulation through the particular blood vessel. The blood clot is known as a thrombus.

Embolus

An embolus is a blood clot that is dislodged from the formation site of a thrombus and travels to another narrower blood vessel in another part of the body where it blocks circulation. A fat embolus is formed from atherosclerotic plague or from the release of endogenous tissue into the blood stream.

Ischemia

When blood supply is restricted in an affected area of the body as a result of a thrombus, embolus or atherosclerotic plaque, oxygen and nutrients are prevented from reaching the tissues of the part of the body involved resulting in a build-up of metabolic substances and damage to the tissue. Irreversible damage may occur in tissues such as those of the heart and brain if oxygenated blood does not reach those areas within 3-4 minutes.

Myocardial Infarction (MI)

Commonly known as a heart attack, it occurs as a result of blockage of a coronary artery that supplies oxygen and nutrients to a part of the heart muscle (myocardium). If left untreated, deprivation of these substances can cause cells of the heart muscle to die and become permanently damaged. The damaged tissue is normally replaced by scar tissue within a few weeks. Blockage of the coronary artery is usually due to the formation of a blood clot that may form due to plaque build-up along the walls of the atherosclerotic artery. Blood clots usually do not form in normal healthy arteries. Most cases of myocardial infarcts occur when a crack develops inside an atherosclerotic plaque. The process exposes the inner portion of the plaque to the blood and then triggers coagulation cascade. Blood clots can be formed elsewhere in the body (embolus) and can travel to a coronary artery. Risk factors for MI occurrence include smoking, lack of exercise, stress, excessive intake of alcohol, obesity, presence of diseases such as high blood pressure, diabetes, and history of ischemic heart diseases.

Cerebral Infarction (Ischemic Stroke)

This event occurs as a result of irregularities in blood supply (ischemia) to the brain. Formation of a blood clot results in ischemic stroke which is different than a stroke due to cerebral hemorrhage. It has been estimated that one third of cerebral infractions are fatal. Symptoms of cerebral infarction depend for the most part on location of the infarct and the resultant ischemia. If an infarction takes place in the motor cortex, a contralateral hemiparesis (weakness in muscles and loss or reduced sensation of one side of the body), and not hemiplegia (total paralysis of a limb or trunk) may result. Risk factors of cerebral infarction are similar to those of a cardiac infarction.

Deep vein thrombosis

This condition occurs as a result of formation of thrombosis, arterial embolism, and an arteriosclerotic fragment in a deep vein especially in the legs. Such obstruction leads to necrosis of the nerve and muscle. Symptoms include absence of pulse in the affected area, pain which could be severe, numbness and muscle weakness.

ANTICOAGULANTS

These medications are used to prevent or reduce blood clotting and to manage thrombotic and thromboembolic disease. Certain blood clots readily form due to variations in blood clotting factors, health status of blood vessels, and the presence of cardiac arrhythmias. Candidates for the intake of anticoagulants are those who:

a) Have mechanical heart valves and are at risk of clot formation. Patients with valves made of animal tissue have reduced risk.

b) Have had myocardial and cerebral infarctions.

c) Have experienced or are at risk of deep thrombosis or pulmonary embolism.

d) Complain of arterial fibrillation.

e) Have undergone surgery, especially orthopedic (hip and knee replacements). Following surgery the blood becomes more prone to clotting.

f) Suffer from angina pectoris, a condition caused by clotting of coronary arteries.

g) Have stents inserted in coronary arteries.

Anticoagulants act by affecting coagulation factors. The mechanism of action of coagulation varies depending on the drug. They are available in the oral and parenteral forms.

ANTIPLATELET AGENTS

These agents act as anticoagulants through decreasing platelet aggregation, thereby inhibiting clot formation. The following antiplatelet drugs are taken orally: clopidogrel, parasugrel, ticagrelor, and triclopidine. Parenteral antiplatelet drugs include: argatroban, bivalirudin, delteparin, enoxaparin, fondaparinux, tinzaparin, and heparin.

Clopidogrel

This drug is a derivative of thienopyridine, which is a class inhibitor of P_2Y_{12} adenosine diphosphate (ADP). The drug was approved by the FDA in 1997 as an antiplatelet agent for the prevention of vascular ischemic events in patients with coronary artery disease, peripheral vascular disorders, cerebrovascular disease, and thrombosis after placement of coronary stent. In order to give a therapeutic effect, clopidogrel must be converted to its active metabolite. The activation is due to cytochrome P450. It may be used concurrently with aspirin and as a replacement in cases where aspirin is contraindicated. It exerts its anticoagulant activity by irreversibly inhibiting the binding of ADP on its receptor, known as P_2Y_{12}, and on platelets. The P_2Y_{12} is a protein found mainly on the surface of blood platelet cells and plays an important role in blood coagulation. Clopidogrel may be ineffective in persons whose body is incapable of converting the drug to its active metabolite. In 2010, the FDA issued a warning that patients whose system is poor in CYP_2C_{19} metabolites may not obtain the anticoagulant activity of clopidogrel due to lack of active metabolite of the drug in the blood. The adverse effects of clopidogrel include fatigue, headache, dizziness, nausea, nosebleed, tarry stool, bruising, and allergy.

Parasugrel

This drug also belongs to the thienopyridine group of ADP receptor inhibitors. It has identical action as clopidogrel and exerts its effect by preventing aggregation or clumping of platelets. It differs from clopidogrel in that it has a quicker and more consistent action. While clopidogrel is not effective in individuals with low level of CY_2C_{19} liver enzyme; parasugrel is effective in these patients.

Ticagrelor

This antiplatelet drug was approved by the FDA in 2011. It is a nucleoside analogue and has similar mechanism of action as clopidogrel and ticlopidine. It can be taken with aspirin, but the dose of aspirin should not exceed 100 mg.

Ticlopidine

Ticlopidine has similar chemical structure and action as ticagrelor. It is used mainly for patients who cannot tolerate aspirin. It may increase the risk of the development of serious hematologic adverse effects. It is also recommended in patients who cannot take parasugrel or clopidogrel.

Parental Antiplatelets Drugs

Parenterally administered antiplatelet medications are known as direct thrombin inhibitors (DTI) due to their inhibition of the enzyme thrombin. Some members of this class may replace heparin and warfarin. Depending on how these drugs interact with the thrombin molecule, DTI's exist in 3 types: bivalent, univalent and allosteric inhibitors. Bivalent DTI's bind to two distinct sites on thrombin, namely its active site and fibrinogen-binding exosite. Such properties are responsible for high affinity and specificity to thrombin.

Bivalirudin (R-hirudin), desirudin and lepiridin, which are derivatives of hirudin, are bivalent DTI's. Hirudin, a peptide which acts as an anticoagulant, is found in the salivary glands of medicinal leeches. Once a leech punctures the skin of its host, hirudin found in the saliva can prevent the blood from clotting at the incision site, thereby allowing the blood to keep flowing for the leech to feed. An adult leech can suck up to 10 times its body weight in one single meal (average 5-15 ml of blood). The anticoagulant activity of hirudin is due to its inhibition of the procoagulant activity of thrombin. In addition, it has a thrombolytic activity, namely it is capable of dissolving blood clots. Since extracting hirudin from leeches' saliva is tedious and costly, recombinant technology has been employed to provide hirudin derivatives. Hirudin and its derivatives have advantages over heparin in that they do not adversely affect the activity of serum proteins such as albumin and globulin.

Bivalirudin is used as an anticoagulant during angioplasty, coronary stent placement, and percutaneous coronary intervention (PCID), for both stable and unstable angina. The drug is analogous to hirudin, and is administered intravenously, and can be used with aspirin. It inhibits thrombin which is present in the circulation. Moreover, it inhibits platelet clumping which is usually mediated by thrombin. Other than binding to thrombin, it does not bind to albumin, globulin or red blood cells. Unlike heparin, it does not produce thrombocytopenia and does not activate platelets. Following binding with thrombin, biralurudin slowing begins to unbind, resulting in recovery of thrombin activity.

Desirudin

Desirudin is used to prevent formation of deep venous thrombosis, mainly in the legs, which may occur following hip replacement. The drug is given for several days after surgery while the patient is unable to walk. This period of immobility may trigger formation of blood clots, and hence the use of desirudin.

Argatroban

This anticoagulant is used in patients with heparin-induced thrombocytopenia and percutaneous coronary innervation. Furthermore, it is used in patients with bleeding cases caused by heparin. It can be used in patients with renal impairment, since it is metabolized in the liver and is not renally cleared.

Heparin

Heparin is an anticoagulant that acts both in vitro and in vivo but has no fibrolytic activity to dissolve blood clots. It acts at different stages of the blood coagulation process. It is capable of inactivating the Activated Factor X which is necessary for clotting. Heparin and its Low Molecular Weight Heparin (LMWH) are used in the prophylaxis of pulmonary embolism and

DVT, but have similar results in reducing mortality. Because it has a short biological half-life of one hour, heparin is given either frequently or as a continuous infusion. Main side effects are reversible and include: heparin-induced thrombocytopenia (HIT). Chromic use could cause osteoporosis. The antidote for heparin is protamine sulfate.

Delteparin Sodium

This LMWH is used with aspirin to prevent blood clotting during and after surgery as well as in the prevention of DVT, pulmonary embolism, and clots in patients with unstable angina.

Enoxaparin Sodium

This is a LMWH and has uses similar to other LMWH drugs.

Fondaparinux

This is chemically related to LMWHs and acts as a Factor X_a inhibitor. It is administered subcutaneously and used following orthopedic surgery and to prevent DVT. It has an advantage over heparin in that its risk of causing heparin-induced thrombocytopenia is low. However, it is not recommended for use in patients with renal impairment.

Tinzaparin sodium

This is an LMWH which can be given subcutaneously and intravenously. It has an advantage over the LMWHs in that it can be given during pregnancy and in patients who have renal dysfunction.

ORAL ANTICOAGULANTS

Anagrelide is used to treat thrombocythemia which may occur as a result of bone marrow disorders. It acts by reducing the production of platelets.

Apixaban is a direct Factor X inhibitor and is used in the prophylaxis of systemic venous thromboembolism and strokes especially those that occur as a result of atrial nonvalvular fibrillation. Moreover, it is used to prevent DVT following hip and knee replacement.

Dipyridamole is used with other drugs particularly aspirin to prevent blood clot formation after heart valve replacement and to reduce the risk of death after heart attacks and strokes.

Cilostazol inhibits platelet aggregation and acts as an arterial vasodilator.

Dabigatran is a DTI anticoagulant used for the prevention of blood clot formation in patients with arterial fibrillation. It reduces the risk of strokes and heart attacks in such patients. It is used as an alternative to warfarin.

Rivaroxaban is used to prevent blood clot formation which may occur after surgery. It also lowers the risk of strokes in the presence of atrial fibrillation and certain heart rhythm disorders. It is a DTI anticoagulant.

Warfarin is a vitamin K antagonist that exerts its activity by inhibiting the enzyme vitamin K epoxide reductase. It is given to patients who require long-term anticoagulant therapy. The drug has a narrow therapeutic index, and, as a result, it requires constant monitoring in order

to achieve optimal results and to reduce the risk of overdose and bleeding.

SUMMARY

Cerebro-cardio-vascular disorders such as infarcts and strokes are common worldwide and are responsible for thousands of deaths annually. There are risk factors that lead to narrowing of blood vessels which may result in formation of blood clots in veins and arteries. Anticoagulant medications are employed in the prevention of the developments of these blood clots.

REFERENCES

1. Hylek EM, Evans-Molina C, Shea C, Hanault LE, Regan S, "Major hemorrhage and tolerability of warfarin in the first year of therapy among elderly patients with arterial fibrillation", Circulation 115 (21):2689 (2007).
2. Heneghan C, Ward A, Perera R, "Self-monitoring of oral anticoagulation: systematic review and meta-analysis of individual patient data", Lancet, 379 (9813):322 (2012).
3. Finegold JA, Asaria P, Francis DP, "Monitoring from ischemic heart disease by country, region, and age: Statistics from World Health Organization and United Nations" International journal of cardiology, 168 (2): 934 (2012).
4. Mozaffarian D, Katan MB, Ascherio A, Stampfer MJ, Willett WC, "Trans fatty acids and cardiovascular disease", N. Engl. J Med, 354 (15):1601 (2006).
5. Eiklboom JW, Hankey GJ, "Low molecular weight happens and heparinoids", Med. J. Australia 177 (7):379 (2002).
6. Hirsh J, Fuster V, Ausell J, Haperin JL, "American Heart Association/American College of Cardiology Foundation guide to warfarin therapy", J. Am. Coll Cardiology, 41 (9):1633 (2003).

Fill in the information below, answer questions and return **Quiz Only** for certification of participation to: CE PRN®, 400 Lake Cook Road, Suite 207, Deerfield, IL 60015.

WHEN YOU SEND IN QUIZZES.
ALWAYS KEEP A COPY. YOU MAY EMAIL OR FAX THEM. FAX # IS 847-945-5037. OR SEND A CONVENTIONAL EMAIL WITH YOUR ANSWERS TO CEINFO@WFPROFESSIONAL.COM

NAME_____**CE PRN I.D.#**(1st line on label)_____
ADDRESS _____CITY _____STATE _____ZIP _____
I am a Pharmacist ☐ **I am a Technician** ☐

CPEMonitor ID _____**Birthdate (MM/DD)** _____
ARE YOU LICENSED IN FLORIDA? IF YES, FL LIC # _____
EMAIL Address (REQUIRED) _____

LESSON EVALUATION
Please fill out this section as a means of evaluating this lesson. The information will aid us in improving future efforts. Either circle the appropriate evaluation answer, or rate the item from 1 to 7 (1 is the lowest rating; 7 is the highest).

1. Does the program meet the learning objectives?

Define the term "platelets"	YES NO
Describe the coagulation process	YES NO
Differentiate between thrombus & embolus	YES NO
List patients who are candidates for anticoagulants	YES NO
Describe mechanisms of action of specific anticoagulants	YES NO

2. Was the program independent & non-commercial YES NO

	Low Relevance						Very Relevant
3. Relevance of topic	1	2	3	4	5	6	7

4. What did you like most about this lesson?_____
5. What did you like least about this lesson?_____

Please Mark the Correct Answer(s)

1. **Thrombocytopenia describes:**
   A. Low platelet count B. High platelet count
   C. WBC deficiency D. Low RBC O_2 level
2. **Which statement is true about hemostasis?**
   A. Takes effect 10 hours after injury
   B. Thromboxane A_2 is a vasodilator
   C. Endothelial cells of intact blood do not allow blood clotting
   D. Another term for blood clot dissolution
3. **A blood clot that is dislodged from the formation site is known as:**
   A. Thrombus B. Embolus
   C. Ischemia D. Infarction
4. **The antidote for heparin is:**
   A. Protamine sulfate B. Magnesium sulfate
   C. Potassium chloride D. Universal antidote
5. **Rivaroxaban acts by:**
   A. Dissolving blood clots
   B. Inhibiting ADP receptors
   C. Deactivating vitamin K
   C. Direct inhibition of thrombin
6. **Which of these is the drug of choice for patients who cannot tolerate aspirin?**
   A. Heparin B. Tricagrelor
   C. Ticlopidine D. Warfarin
7. **The intake of anticoagulants is recommended to patients who:**
   A. Suffer from migraine
   B. Manifest with atrial fibrillation
   C. Are over 65 years of age
   D. Are obese
8. **Usually, clopidogrel is not taken concurrently with aspirin.**
   A. True B. False
9. **Which statement is true about warfarin?**
   A. It is a vitamin K antagonist
   B. Has wide therapeutic index & no adverse effects
   C. Usually administered for only one week
   D. Only dosed parenterally
10. **To achieve optimal results & reduce the risk of bleeding from the use of warfarin:**
    A. Reduce dose by 50%
    B. Take once a week
    C. Take after meals
    D. Constant monitoring is required

CE PRN®
400 Lake Cook Road Suite 207
Deerfield, IL 60015

(Fax) 847-945-5037
(Email) ceinfo@wfprofessional.com

ATTENTION!
RENEWALS HAVE BEEN MAILED
RESPOND NOW.

ALL PHARMACISTS---READ THIS!!!
Check your CE activity or print a statement from your CPE Monitor eProfile Account. To login, go to www.nabp.net. Enter your user name (your email address) & your password. Click on "CE Activity" to view your history & print a CE report.
WE NO LONGER MAIL OUT CREDIT STATEMENTS.

Contributing Author
Farid Sadik, Dean Emeritus
University of South Carolina
College of Pharmacy
Columbia, SC
College of Pharmacy

Executive Editor
William J. Feinberg,
BS Pharm, MBA

Program ID # s for this lesson:
707-000-14-010-H01-P (for Pharmacists).
707-000-14-010-H01-T (for Technicians).

CE Provider Tracking # with CEBroker.com is 50-3170.

CE PRN® is a publication of W-F Professional Associates, Inc.
W-F Professional Associates, Inc. is accredited by the Accreditation Council for Pharmacy Education (ACPE) as a provider of continuing pharmaceutical education. Providers who are accredited by ACPE are recognized by **All States** for fulfilling CE requirements. Participants completing this course by by September 30, 2017 may receive full credit. This lesson furnishes 1.25 hours (0.125 CEUs) of credit.

TO DOWNLOAD LESSONS!!!

- **Go to website www.wfprofessional**
- **Click on "COURSES."**
- **Click on "YEAR."**
- **Click on the ID #"707-000-……." for your lesson of interest.**

FLORIDA PHARMACISTS---READ THIS!
We don't know you're Florida licensed unless you tell us. Place your Florida license # on EVERY quiz.

WHEN YOU SEND IN QUIZZES.
ALWAYS KEEP A COPY. YOU MAY EMAIL OR FAX THEM. FAX # IS 847-945-5037. OR SEND A CONVENTIONAL EMAIL WITH YOUR ANSWERS TO CEINFO@WFPROFESSIONAL.COM