



## Dental management of the anticoagulated patient

Lieutenant Commander Nicholas J. Toscano, DC, USN, Captain John Mumford, DC, USN and Captain Blake Turner, DC USN

### Introduction

With advances in medicine, dentists are faced with managing patients with complex medical histories. Frequently, patients on anticoagulation therapy present to the dental office. These patients can be of significant intra-operative and post operative bleeding risk when procedures such as dental extractions, periodontal surgery, biopsies, and even block local anesthesia, are required. Knowledge of the drugs involved, common lab values, and surgical management of the anticoagulated patient can help the dentist lower the risks of bleeding associated with these patients.

### Coagulation physiology

When the integrity of a vessel's endothelium is interrupted, underlying collagen fibers are exposed to the circulation. This collagen attracts platelets and a loose aggregate called the temporary hemostatic plug is formed on the damaged surface. Aggregated platelets release substances which cause local vasoconstriction and facilitate the formation of a definitive clot.

There are three phases of hemostasis. The first phase occurs immediately after injury and involves vasoconstriction of the blood vessel to limit blood loss. The second phase is mediated by the platelet whose functions include maintenance of vascular integrity, formation of the platelet plug, and stabilization of the plug. The final phase is mediated by the intrinsic and extrinsic coagulation cascade which involves the formation of fibrin.

The final phase is key to the formation of a definitive clot. It is dependant upon a complex series of biochemical reactions that ultimately leads to the formation of a fibrin clot. These reactions involve conversion of an inactive coagulation plasma protein into an inactive coagulation factor that activates the next coagulation factor in the cascade. The final step in this cascade is the conversion of the plasma protein fibrinogen to fibrin. Fibrinogen is a large, soluble protein produced by the liver and its conversion to fibrin is catalyzed by the enzyme thrombin. The generation of thrombin follows the same general principle as fibrin. An inactive precursor, prothrombin, is converted to the active enzyme thrombin. This conversion is mediated by activated factor X. Activated Factor X can be generated by one of two pathways: the intrinsic pathway mediated by Factor XII and the extrinsic pathway mediated by VII. The anticoagulation drugs affect different areas of this clotting cascade which effects the formation of the definitive clot.<sup>1</sup>

### Medications used in anticoagulant therapy

Aspirin is routinely used to lower the risk of heart attacks and strokes. It may be prescribed in as little as 81 mg a day. Aspirin

inhibits prostaglandin synthesis and prevents formation of the platelet-aggregating substance thromboxane A<sub>2</sub> thereby preventing formation of the platelet plug.<sup>2</sup>

Clopidogrel bisulfate (Plavix<sup>®</sup>) is used in the prevention of atherosclerotic events in patients with documented atherosclerosis, recent history of myocardial infarction (MI), strokes, or peripheral arterial disease. The usual dosage is 75 mg per day. Plavix works by blocking the adenosine diphosphate (ADP) receptor which prevents the binding of fibrinogen to the site of injury.<sup>2</sup>

Warfarin (Coumadin<sup>®</sup>, Jantoven<sup>®</sup>) is used in the prophylaxis and treatment of cardiovascular thromboembolic events. Warfarin works by interfering with the synthesis of vitamin K dependent coagulation factors produced by the liver, i.e. Factors II, VII, IX and X. These Factors are thus inactive and affect both the intrinsic and extrinsic pathway.

Heparin (Hep-lock<sup>®</sup>) is commonly used in hospital procedures such as hemodialysis or may be prescribed on an outpatient basis for patients with total hip, joint replacement, deep vein thrombosis or asymptomatic pulmonary embolism. Heparin works by forming a complex with antithrombin III and makes it one thousand times more effective at removing thrombin. It also inactivates prothrombin as well as Factors IX, X, XI and XII preventing the formation of the fibrin clot.<sup>2</sup>

### Lab evaluation of the anticoagulated patient

Bleeding problems can be screened by various lab tests which include the platelet count, bleeding time, prothrombin time, partial thromboplastin time, and International Normalized Ratio (INR).

The platelet count provides a quantitative evaluation of platelet function. A normal platelet count should be 100,000 to 400,000 cells/mm<sup>3</sup>. A platelet count of less than 100,000 cells/mm<sup>3</sup> is called thrombocytopenia and often can be associated with major postoperative bleeding. The average lifespan of a platelet ranges from 7-12 days.

The bleeding time provides an assessment of adequacy of platelet count and function. The test measures how long it takes a standardized skin incision to stop bleeding by the formation of a temporary hemostatic plug. The normal range of bleeding time depends on the way the test is performed, but is usually between 1 and 6 minutes. The bleeding time is prolonged in patients with platelet abnormalities or taking medications which affect platelet function. This test assesses platelet function.<sup>1</sup>

The prothrombin time (PT) measures the effectiveness of the extrinsic pathway to mediate fibrin clot formation. It is performed by measuring the time it takes to form a clot when calcium and tissue factor are added to plasma. A normal

prothrombin time indicates normal levels of Factor VII and those factors common to both the intrinsic and extrinsic pathways (V, X, prothrombin, and fibrinogen). A normal prothrombin time is usually between 10 and 15 seconds. Prothrombin time is most often used by physicians to monitor oral anticoagulant therapy such as warfarin.<sup>1</sup>

The partial thromboplastin time (PTT) measures the effectiveness of the intrinsic pathway to mediate fibrin clot formation. It tests for all factors except for Factor VII. The test is performed by measuring the time it takes to form a clot after the addition of kaolin, a surface activating factor, and cephalin, a substitute for platelet factor, to the patient's plasma. A normal partial thromboplastin time is usually 25 to 35 seconds. Partial thromboplastin time is most often used by physicians to monitor heparin therapy.<sup>1</sup>

The INR was designed for patients on chronic anticoagulant therapy. It allows comparisons from one hospital to another. A patient with normal coagulation parameters has an INR of 1.0. The therapeutic range for a patient on anticoagulant therapy is between 2.0 and 3.5.<sup>1</sup>

### Dental management

Management is dependent on the type of procedure being performed, lab test results and type of medication the patient is taking. Aspirin or Plavix<sup>®</sup> therapy can be discontinued 7 days prior to surgery which should result in better hemostasis. These drugs can then be restarted safely 48 to 72 hours post-operatively. For patients taking Coumadin<sup>®</sup>, proper lab tests should be done and a consult to the physician may be required depending on lab results, medical condition, type of surgery being performed and the possible need for drug dosage reduction. Studies have shown that extractions can be done in patients with an INR of 2.5 to 3.5 safely, however the higher the INR, the more the need for hemostatic measures.<sup>3,4,5</sup> Jeske found that the literature does not support the routine withdrawal of anticoagulation therapy. Dentists should be prepared for bleeding that exceeds normal and may have to provide hemostatic measures.<sup>6</sup> Giglio suggested that single tooth extractions or minimally invasive procedures such as crown lengthening where minimal bleeding is expected, are indicated if the INR is less than 4. In procedures where moderate bleeding is expected, such as block or gingival grafts, an INR of less than 3 is necessary.<sup>7</sup> Little and Falace's review of the literature, recommends that surgery may be performed with an INR of 2.0 to 3.0. For INR values of 3.0 to 3.5, it is recommended that the dosage of anticoagulant be altered depending on bleeding expected during the surgical procedure. Surgery should be delayed for values of 3.5 until the INR is within the therapeutic range of 2.0 to 3.5.

### Local hemostatic measures

1. Local pressure can be applied for 5 minutes to promote clot formation in the surgical area.
2. Sutures can help maintain primary wound closure and approximate the tissue margins during surgery.
3. Electrocautery enhances hemostasis during surgery; it cauterizes the wound as it cuts.

4. An absorbable gelatin sponge (Gelfoam<sup>®</sup>) provides a template for fibrin strands in which platelets become entrapped. It is placed within the surgical site.<sup>8</sup>
5. Oxidized cellulose (Surgicel<sup>®</sup>) increases hemostatic pressure in the surgical area. It is placed within the surgical site.
6. Microfibrillar collagen (Avitene<sup>®</sup>) when placed in the wound, and contacts blood, attracts platelets and initiates the platelet plug.
7. Topical thrombin converts fibrinogen into fibrin and results in hemostasis by enhancing the formation of a clot.
8. Antifibrinolytic therapy (tranexamic acid) works by neutralizing the fibrinolytic effects of saliva and stabilizes the fibrin clot.
9. Post-op use of ibuprofen, aspirin, steroids as well as certain antibiotics, should be used with caution as these drugs may enhance the effects of warfarin.

### Conclusion

Surgical dental procedures in the anticoagulated patient can be safely achieved provided the dentist has a knowledge and understanding of the limits and risks of the anticoagulated patient.

### References

1. Little J, Falace D. Dental Management of the medically compromised patient. 6<sup>th</sup> ed. St. Louis: Mosby; 2002.
2. Wynn R. Drug information handbook for dentistry. 10<sup>th</sup> ed. Hudson, OH: Lexi Comp; 2004.
3. Devani P, Lavery KM, Howell CJ. Dental extractions in patients on warfarin: is alteration of anti coagulation necessary? Br J Oral Maxillofac Surg. 1998 Apr;36(2):107-11.
4. Blinder D, Manor Y, Martinowitz U, Taicher S, Hashomer T. Dental Extraction in patients maintained on continued oral anticoagulant: comparison of local hemostatic modalities. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 1999 Aug;88(2):137-40.
5. Blinder D, Manor Y, Martinowitz U, Taicher S. Dental extraction in patients maintained on oral anticoagulant therapy: comparison of INR value with occurrence of postoperative bleeding. Int J Oral Maxillofac Surg. 2001 Dec;30(6):518-21.
6. Jeske A, Suchko G. Lack of scientific basis for routine discontinuation of oral anticoagulation therapy before dental treatment. J Am Dent Assoc. 2003 Nov;134(11):1492-7.
7. Giglio: Complications of dentoalveolar surgery. In Kwon P, Laskin D. Clinicians manual of oral and maxillofacial surgery London: Quintessence;1997.
8. Ball JH. Management of the anticoagulated dental patient. Compend Contin Educ Dent. 1996 Nov;17(11):1100-2, 1104, 1106 passim.

Lieutenant Commander Toscano is a recent graduate in the Periodontics Program at the Naval Postgraduate Dental School. Captain Mumford is staff periodontist at the Branch Health Clinic, Naval Academy, and Captain Turner is Department Head, Oral Maxillofacial Surgery Department, Naval Postgraduate Dental School, National Naval Medical Center.

The views expressed in this article are those of the authors and do not necessarily effect the official policy or position of the Department of the Navy, Department of Defense, nor the U.S. Government.

Note: The mention of any brand names in this *Clinical Update* does not to imply recommendation or endorsement by the Department of the Navy, Department of Defense, or the US Government.