Dual Antiplatelet Therapy for Heart Disease
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Within the body, the heart pumps blood through paths called vessels so that all of the body’s organs get the oxygen and nutrients they need to function properly. The blood itself contains many components in addition to oxygen and nutrients, including tiny plate-shaped cell fragments called platelets. These platelets are useful because when they are needed, for example, when someone cuts a finger, the platelets can change their shape and stick together to help form clots to stop the bleeding. Platelets also form clots inside the body in the vessels that carry blood. Sometimes these internal clots are helpful, but at other times, a blocked heart vessel results in decreased blood flow, reduced oxygen supply, and ultimately injury to the heart.

Antiplatelet therapy is a term used to describe the use of medications to block platelets and therefore to help prevent the formation of clots. Dual antiplatelet therapy (DAPT) refers to treatment regimens in which 2 different medications that block platelets are given at the same time. DAPT is usually prescribed after a heart attack or stent placement to keep the vessels open and to prevent future heart attacks. This Cardiology Patient Page explains how this works and why DAPT is useful.

How Does the Heart Function Normally?
The heart is responsible for pumping blood to the body, providing organs with the oxygen and nutrients necessary for normal function. The heart requires its own steady supply of oxygen and nutrients to pump effectively. To meet this need, there are vessels, called coronary arteries, that supply the heart with blood, ensuring that the heart always has a sufficient supply of oxygen. This task is easiest when one is inactive (ie, during rest or sleep) and becomes more challenging as bodily demands increase (ie, during exercise or stress, when the heart is asked to pump much harder and faster than usual).

What Can Go Wrong?
Over time, some people develop blockages within the coronary arteries, usually in the form of plaques filled with cholesterol and other cells. As plaques grow in size, blood flow is diminished, and the heart suffers a mismatch of the supply and demand (Figure 1). Sometimes, plaques can rupture. The body tries to contain these ruptures by forming blood clots on top of the plaques. Unfortunately, blood clots can do more harm than good, further obstructing the coronary arteries and diminishing blood flow (Figure 2). When the heart suffers an acutely decreased supply of blood, heart cells are starved of oxygen and cannot survive. This is called a myocardial infarction or, more commonly, a heart attack.

Why Are Stents Important?
There are 2 main reasons to place a stent inside a coronary artery. The first arises when a severe heart attack occurs and blood flow needs to be quickly restored to the heart to prevent further myocardial infarction. The second occurs when stable plaques grow very large and severely obstruct blood flow. In either case, the purpose of the stent is to open the blockage and to restore normal blood flow to the heart. However, blood cells can form clots on stents. Just as with ruptured plaques, blood clots inside coronary arteries can lead to heart attacks, so it is critical that these clots are prevented from forming after stents are placed.

The information contained in this Circulation Cardiology Patient Page is not a substitute for medical advice, and the American Heart Association recommends consultation with your doctor or healthcare professional.

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What Role Do Platelets Play?
One of many components of blood, platelets are small cell fragments that play an important role in clotting. When blood vessels are damaged, the body sends out signals that recruit platelets to the site of injury and cause them to become sticky and adhere to each other. When a cholesterol plaque inside a coronary artery ruptures, a similar reaction occurs, and these sticky platelets play a major role in forming the clots that lead to heart attacks. Thus, inhibiting platelets is a critical part of preventing blood clots on plaques and stents and thereby preventing heart attacks.

How Are Platelets Blocked?
The most basic medication used to block platelets is aspirin (acetylsalicylic acid). Platelets contain an enzyme that is activated during clot formation and releases chemicals that cause platelets to stick together. Aspirin inhibits this enzyme, thereby making platelets less sticky. However, certain people benefit from even more platelet inhibition than aspirin alone can provide. In those who suffer a heart attack or undergo stent placement, in whom it is most critical to prevent blood clots, doctors often prescribe 2 medications that block platelets, referred to as DAPT.

What Is DAPT?
There are multiple ways by which platelets stick together and contribute to blood clot formation. Whereas aspirin blocks an enzyme that leads to sticky platelets, there are also receptors on the platelet surface that, when activated, lead to platelet adherence and clot formation (Figure 3). One class of medications that block platelets does so by inhibiting a certain receptor (known as the P2Y12 receptor) on the surface of platelets. This class includes medications such as clopidogrel (Plavix), prasugrel (Effient), and ticagrelor (Brilinta). When DAPT is prescribed, it usually consists of aspirin plus 1 of these 3 drugs. DAPT leads to greater platelet inhibition than that achieved by aspirin alone. By decreasing the likelihood of blood clot formation inside coronary arteries, at the site of ruptured plaques, or on stents, DAPT decreases the likelihood of future heart attacks. Therefore, DAPT is an essential component of the medical regimen of certain patients with heart disease, specifically those with recent heart attacks or stents.
What Else Do I Need to Know?

- **Side effects.** It is important to be aware that DAPT increases the chances of bleeding. The exact risk depends on individual factors.
- **Duration.** DAPT is most often prescribed for a specific period of time after a heart attack or stent placement, ranging anywhere from several weeks to possibly lifelong.
- **Other medications.** The risk of bleeding with antiplatelet medicines can be influenced by other drugs. Medications of particular importance include blood thinners (such as warfarin) and nonsteroidal anti-inflammatory drugs (such as ibuprofen).
- **Every person is unique.** Although there are certain broad groups of people who generally benefit from DAPT, patients and physicians must ultimately weigh the risks and benefits when determining if DAPT is right. The specific concerns about side effects, duration, and interactions with other medications should always be discussed.

**Additional Resources**

For additional information, please consult one of the following resources:

- American Heart Association Web site (www.americanheart.org).
- To learn about ongoing clinical trials, see www.clinicaltrials.gov.

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**References**


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**Figure 3.** Cellular image of a platelet depicting 2 mechanisms of platelet activation. Adenosine diphosphate (ADP) attaches to the P2Y12 receptor, leading to platelet activation and further ADP release. Once a platelet is activated, it also releases arachidonic acid (AA), which is converted to thromboxane A2 (TXA2) and attaches to the TXA2 receptor, leading to further platelet activation and further release of AA and ADP. Clopidogrel, prasugrel, and ticagrelor inhibit the activation of the P2Y12 receptor by ADP. Aspirin inhibits the conversion of AA to TXA2.
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