

HeartFax

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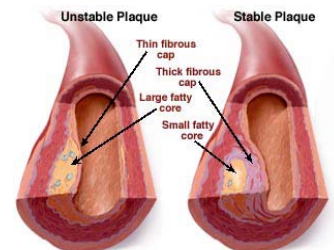
Acute Coronary Syndromes

William Heberden first described angina pectoris in 1772. His original paper describes chest discomfort provoked by exercise but he also mentions progression to chest discomfort at rest. Our current nomenclature for coronary syndromes retains this useful division. We now speak of non-acute and acute coronary syndromes. Depending on whom one reads, there are four or five syndromes in total.

The non-acute coronary syndromes consist of **stable angina pectoris** and **accelerated angina pectoris**. Stable angina pectoris is defined as chest discomfort provoked by exertion and relieved

by rest or nitroglycerin. It is associated with non-disrupted plaque. Accelerated angina pectoris seems to be a transitional syndrome in which angina is provoked by lesser and lesser amounts of exertion. The mechanism is not known for certain but may involve severe, highly obstructive plaque that is nearly occlusive.

The acute coronary syndromes include **unstable angina**, **non-ST elevation infarct**, and **ST elevation infarct**. A unifying feature is the occurrence of chest discomfort at rest. Moreover each of these syndromes is associated with disrupted plaque and a variable amount of superimposed



As plaque builds up, it can become either stable or unstable. Unstable plaque is more prone to sudden rupture, a potentially life-threatening event.

thrombus. In unstable angina the thrombus is partially obstructive whereas in non-ST elevation infarct the thrombus is often subtotally occlusive. In a few cases of non-ST elevation infarct the artery is totally occluded but there are abundant collaterals. With respect

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Save Lives, Restore Health, Instill Hope....

Southern California Cardiovascular Consultants was founded in 1980 by Lawrence R. O'Connor, M.D., F.A.C.C. Since its establishment, SCCC has provided its services to more than 20,000

patients in the greater Los Angeles community. Located in Glendale, California on the campus of Glendale Memorial Hospital and Heart Center, a nationally recognized top 100 heart hospital,

SCCC strongly believes its mission is to:

**Save Lives
Restore Health
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SCCC specializes in the following areas:

- Coronary Artery Disease
- Heart Failure
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- Congenital Heart Disease (Adult)
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- Valvular Heart Disease
- Vascular Disease

Acute Coronary Syndromes (continued)



Glendale Heart Center

"The Heart Center at Glendale Memorial Hospital has been selected as one of the top 100 heart hospitals in the U.S. two out of the last three years."

to ST elevation infarct, the coronary artery is almost always totally occluded. The mechanism of plaque disruption is currently the center of intense scientific scrutiny. Clearly, inflammation plays a central role in thinning and making vulnerable the endothelial/fibrous cap of the immature and usually soft plaque. Exposure of plaque elements to the blood stream provokes activation and cascade of the clotting proteins that result in thrombin formation and platelet adhesion.

Understanding the centrality of plaque rupture and clot propagation informs our approach to therapy for the acute coronary syndromes. We select drugs that antagonize thrombin formation and platelet adhesion. For both unstable angina and non-ST elevation infarct a useful mnemonic is **ALPS**. This stands for aspirin, Lovenox, plavix, and, if necessary, a small molecule 2b3a inhibitor. Usually patients stabilize with the first three of these drugs. Patients who have recurrent chest pain typically "cool off" on the addition of Integrilin or Aggrastat. When stable, the patient may be taken to the cath lab where stent implantation effectively terminates the syndrome. Thrombus is trapped between the stent and arterial wall and the stent re-apposes the disrupted endothelium and media.

The treatment of ST elevation infarct is more problematical. Roughly 80 percent of hospitals in the United States do not have catheterization laboratories and

thus rely on lytic drugs whose success rate in opening the artery is in the neighborhood of 60 percent. Long-term patency may be only half this number. A more effective approach is direct infarct coronary intervention. Presently the state-of-the-art involves passing a guide wire through the occlusive thrombus, delivering a stent into this region and deploying it without pre-dilation. An area of hot debate centers around whether a combination of half dose lytic and bolus dose ReoPro should be administered on the way to cath lab. Pilot studies suggest a patency rate of 80 percent on arrival in the catheterization suite. TIMI 2 or 3 flow is often present. Chest pain frequently abates in the ER or during transfer to the lab. Establishing even a small lumen at the culprit site facilitates the choice of an appropriate diameter stent and technical aspects (like length and positioning) that affect successful deployment. Small, elderly females and patients over the age of 70 may be at increased risk of intracerebral hemorrhage. This is especially so in the setting of marked hypertension. Mortality rates for direct coronary intervention have been reported as low as four percent. Mortality rates for thrombolytic therapy remain in the 9-11 percent range. Mortality rates for acute ST elevation infarct in the pre-thrombolytic era were approximately 30 percent.

The Heart Center at Glendale Memorial Hospital has been selected as one of the top 100 heart hospitals in the U.S. two out of

the last three years. In part this is due to a very low mortality rate for acute myocardial infarction. This in turn is due to a 24/7 availability of direct infarct intervention by an experienced and dedicated multidisciplinary team. •



Lawrence R. O'Connor, M.D.



Y. Joseph Wen, M.D.