THE KNOWLEDGE OF ACUTE CARE NURSES REGARDING ACUTE CORONARY SYNDROMES

By

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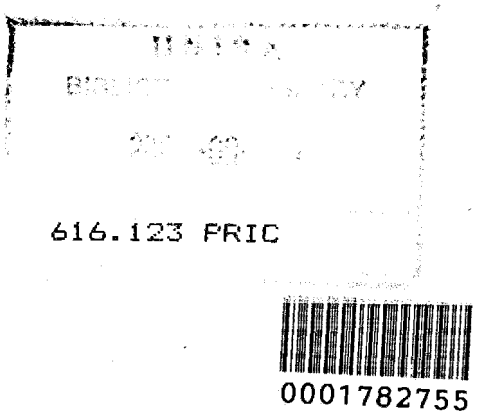
SUMMARY

The term *Acute Coronary Syndrome* (ACS) encompasses a spectrum of patients who present with chest discomfort or other symptoms caused by myocardial ischemia or infarction. Since critical or acute care nurses care for such patients, they should have a thorough knowledge of ACS pathophysiology and current treatments for ACS

The purpose of this research study is to explore and describe the knowledge level that the critical care nurses in a state hospital in East Texas feel they have regarding ACS. This study was quantitative, descriptive and contextual in design, in which a sample survey was performed, using a questionnaire based on a literature study.

The response of most of the critical care nurses tested was that they felt they had insufficient knowledge. An in-service training session has been proposed to help improve the nurses' knowledge and expertise on ACS.



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LIST OF ABBREVIATIONS

ACE	angiotension-converting enzyme
ACS	acute coronary syndromes
ADN	Associate Degree of Nursing (Registered Nurse)
BSN	Bachelor of Science in Nursing (Registered Nurse)
CAD	coronary artery disease
CBC	complete blood count
CPK	creatinine phosphokinase
CPK-MB	creatinine phosphokinase
ECG	electrocardiogram
FPCI	facilitated percutaneous coronary intervention
GP IIb/IIIa	glycoprotein IIb/IIIa inhibitor agents
ICU	intensive care unit
LDL	low density lipoprotein
LMWH	low molecular weight heparins
LVN	Licensed Vocational Nurse
MI	myocardial infarction
MS/MN	Masters of Science / Masters in Nursing
NSAID	nonsteroidal anti-inflammatory drug
NTG	nitroglycerine
PCI	percutaneous coronary intervention
RN	Registered Nurse
UFH	unfractioned heparin
VSMC	vascular smooth muscle cell

CHAPTER 1

OVERVIEW OF STUDY

1.1 INTRODUCTION

Acute coronary syndromes (ACS) – unstable angina, non-Q-wave myocardial infarction (MI), and Q-wave MI – all share the physiological feature of thrombosis due to disruption of atherosclerotic plaque. Advances in understanding of the pathophysiology of ACS have led to the development of new therapeutic agents and treatment strategies that hold promise for better clinical outcomes.

Patients with life-threatening acute myocardial infarctions have been the main focus of traditional chest pain protocols. That is because they require immediate treatment to stop the infarction, usually with thrombolytics and angioplasty, atherectomy, or intracoronary stenting. This emphasis on immediate care for the acute MI patient puts patients who might be at long-term risk for developing an MI in the background, which means they may receive less aggressive treatment, or no treatment at all. ACS encompasses not only acute MI patients, but also subsets of high-risk patients who need aggressive treatment to *prevent* an MI (Sieck 2000: 5).

An **acute MI** is a full-thickness injury to the myocardial tissue, demonstrated on ECG by ST segment elevation. A **non-Q wave MI** suggests that plaque or a thrombus is just beginning to reach critical form in the vessel wall, and is denoted by ST segment depression on ECG. In these patients, the ST segment could flip up as blockage develops into a full-thickness injury. **Unstable angina**, depicted by ST segment depression or T-wave inversion on ECG, can develop because of plaque, a small thrombus or arterial spasms.

In the past decade, considerable progress has been made in treatment of the ST segment elevation population, and we now have a number of established therapies for this

population, including reperfusion therapy, anti-platelet therapy, as well as the use of beta-blockers, ACE inhibitors, and lipid-lowering therapy. As critical care nurses have increased their knowledge and expertise of such therapies, they have become more comfortable in providing nursing care for ACS patients. In contrast, the non-ST segment elevation population is more diverse and the severity of ischemic heart disease is more difficult to establish at the time of presentation. New approaches in the treatment of unstable angina and non-Q-wave MI include the use of new cardiac markers for diagnosis, glycoprotein IIb/IIIa inhibitor agents as an adjunctive therapy with percutaneous coronary intervention (PCI) or alone as medical therapy, and low molecular weight heparins (LMWH). Because many of these new approaches to treating unstable angina and non-Q-wave MI are not yet firmly established, some critical or acute care nurses may find themselves in the middle of a steep learning curve. As they increase their knowledge and expertise in these areas, they will achieve a greater familiarity with the nursing care required for such patients.

In addition to being familiar with current approaches to treating ACS, a thorough knowledge of the pathophysiology of ACS is vital if the nurse is to understand the rationale behind approaches for treating ACS. This begins with the very beginnings of ACS – atherosclerosis, endothelial damage, the clotting cascade, and the formation and rupture of thrombi and plaque (Gylys & Gold 2000: 3-13; Sieck 2000: 5-6). Critical or acute care nurses must know the pathophysiology of coronary artery disease (CAD) and understand the rapidly evolving treatment options for ACS in order to provide highly skilled and knowledgeable critical nursing care and be able to answer questions and provide guidance to their patients and other members of the healthcare team.

1.2 RESEARCH PROBLEM

Acute Coronary Syndromes (ACS) is a term that has been coined over the past decade which encompasses unstable angina, non-Q-wave MI and Q-wave MI. Although the disease processes themselves are not new, advances have been made in understanding the causes and in treatment options.

Cardiovascular intensive care nursing has been of great interest to the researcher for many years, but what sparked interest in this study was comments made by some very experienced intensive care nurses during a conversation one day where it became clear that they did not know the difference between a non-Q-wave and a Q-wave MI. Likewise, the definition of ACS was not known. This surprised the researcher, because these nurses had extensive ICU nursing experience and regularly cared for cardiac patients with MI's and open-heart surgery.

The problem then that emerged is that it seems as if nurses working in the critical or acute care units lack knowledge about ACS.

1.3 SIGNIFICANCE OF THE STUDY

This research study is significant to the critical or acute care nurse population in general because it stresses the need for such nurses to be knowledgeable about the conditions of their patients. This study revealed a perceived knowledge deficit about ACS in a sample population of critical or acute care nurses and subsequently provided a comprehensible in-service training session on ACS. The researcher believes that in order to provide quality nursing care for patients who fall in the ACS continuum, the nurse should have a thorough knowledge of the disease process of ACS, treatments and medications currently available and their rationales for use. She should also be able to explain these in a clear and simple manner comprehensible to their patients. Understanding the pathophysiology of ACS begins with the beginnings of ACS – atherosclerosis, endothelial damage, the clotting cascade, and the formation and rupture of thrombi and plaque (Ryan 2000: 5-6; Gylys & Gold 2000: 3-13). A thorough knowledge of the disease process of coronary artery disease (CAD) provides a basis for comprehending the rapidly evolving treatment options for ACS. It helps the nurse provide highly skilled and knowledgeable critical nursing care and be able to answer questions and provide guidance to their patients and other members of the healthcare team. It also behooves the critical care nurse to have

some knowledge of current research about ACS and possible treatments available in the future.

The researcher noted during her literature study that the majority of information on ACS was found in medical journals for physicians, although a small number of articles were found in nursing publications. In addition to numerous medical journal publications and several nursing journal publications (see bibliography), the researcher found two books to be immensely comprehensive and informative on the subject of ACS: Acute Coronary Syndromes, edited by EJ Topal, is an excellent source which covers detailed pathophysiology of ACS: pre-, in- and post-hospital treatment of unstable angina, non-Q-wave MI and Q-wave MI; cardiac markers; chest pain control centers; includes an explanation of the GUSTO Trials; stenting and angioplasty; plaque stabilization; and future possible therapies for treating ACS. Cardiovascular Trials Review editors, RA Kloner and Y Birnbaum, give short one to two page reviews on cardiovascular trials, many of which deal with acute MI, unstable angina, coronary artery disease, atherosclerosis and the prevention of its progression, and other related topics.

1.4 OBJECTIVES

The objectives of this research study were:

- to explore and describe the critical care nurse's knowledge according to their own rating regarding the pathophysiology of ACS and current treatments available
- to use the information obtained to formulate a four hour in-service training session for critical care nurses about ACS and current treatments

1.5 ASSUMPTIONS OF THIS STUDY

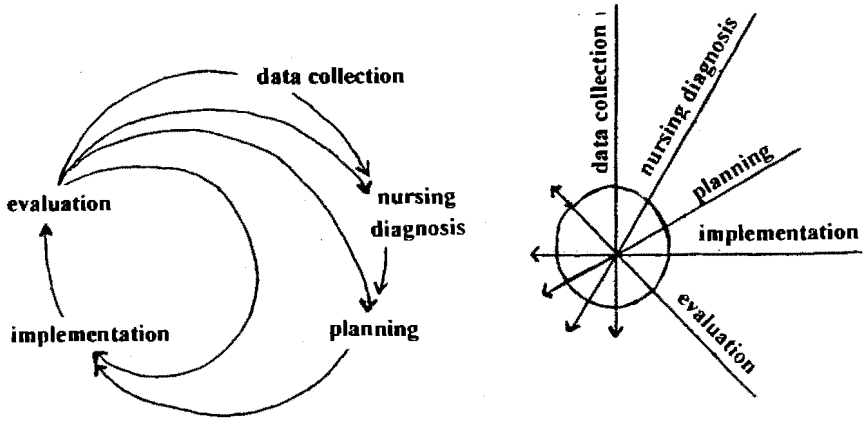
Critical thinking is imperative to the nursing process. This is the underlying philosophy driving this research. Smeltzer & Bare (2000:23) defines critical thinking as follows:

"Critical thinking is a cognitive or mental process that involves conscious, systematic, reflective, rational, and goal-oriented examination and analysis of all available information and ideas and the formation of conclusions and the most appropriate, often creative decisions." Independent judgments and decisions arise from a sound knowledge base and the ability to integrate information within the context in which it is gathered. Nursing practice today – especially critical care nursing – *must* involve the use of high-level critical thinking skills within the nursing process. Critical thinking is imperative to clinical decision making, helping to identify patient needs and to decide which nursing actions will help the patient in meeting those needs.

The nursing process is a method for making decisions. It is a way of thinking and acting in relation to the clinical phenomena of concern to nurses. Classically, it comprises five phases or dimensions: nursing assessment, nursing diagnosis, planning, implementation and evaluation. The nursing process is a way of solving clinical problems, but it is more than just a problem-solving method. It is a *way of thinking*, and unlike a problem-solving method, the nursing process is *continuous*, not episodic.

The five phases of the nursing process represent a continuous cycle throughout the nurse's moment-to-moment data interpretation and management of patient care. This process is also *interactive*, i.e. all the phases operate and influence each other and the patient simultaneously. Such a systematic method for approaching, analyzing and managing clinical problems is invaluable because it leads to sound decisions (Thelan, Davie, Urden & Lough 1994: 3-4). The two diagrams in Figure 1.1 illustrate the cyclical and interactive nature of the nursing process.

Figure 1.1 Cyclical and interactive nature of the nursing process



Critical or acute care nurses are highly specialized nurses, and the researcher assumes that they have a solid knowledge of critical care nursing in order to provide the skilled and competent intensive care for patients whose condition may change rapidly and who require both close monitoring and critical care intervention. The nurse is responsible for maintaining competence by continual learning, education and in-service training. The nurse is responsible for the patient to whom he/she has been assigned, and is accountable for his/her actions and omissions. It is therefore assumed that the critical or acute care nurse should have a thorough and relevant knowledge and competency regarding the patients he/she cares for, including ACS where ACS patients are cared for.

1.6 KEY TERMS AND CONCEPTS FOR THIS RESEARCH STUDY

1.6.1 ACUTE CORONARY SYNDROMES

ACS includes a continuum of patients presenting with chest pain or other symptoms caused by myocardial ischemia or infarction. ACS includes unstable angina, non-Q-wave MI and Q-wave MI.

1.6.2 ATHEROSCLEROSIS

A generalized disease of the arteries characterized by focal changes in the intima of the large and medium-sized arteries. These changes consist of the accumulation of lipids, calcium, blood components, carbohydrates, and fibrous tissue (atheroma or plaque) on the intimal layer of the artery. Because atherosclerosis is a generalized disease of the arteries, when it is found in the extremeties, it is usually present elsewhere in the body (Smeltzer & Bare 2000: 749).

1.6.3 CRITICAL OR ACUTE CARE NURSING

In 1984, the American Association of Critical Care Nurses (AACN) defined critical or acute care nursing in this way: " ... the American Nurses' Association defines nursing as

the 'diagnosis and treatment of human responses to actual or potential health problems.' Critical care nursing is that specialty within nursing that deals specifically with human responses to life-threatening problems." (Hartshorn, Lamborn & Noll 1993: 4) Critical or acute care nursing involves the care of highly unstable, highly at-risk patients whose health conditions may change minute by minute. Critical care or acute nurses are highly specialized nurses who have a solid understanding of critical care nursing and of relevant information from other sources. The term ***critical care nursing*** is exchangeable with the terms ***acute care nursing*** or ***intensive care nursing***. In this study the term *critical care nursing* is used more often than the term *acute care nursing,* as in the literature the term *critical care nursing* is used more often.

1.6.4 CRITICAL OR CARE ENVIRONMENT

The critical care environment as defined for this research study is the areas of the hospital with specialized facilities and technology needed to care for critical care patients, including pre- and post-operative patients. The facilities include monitoring devices and resuscitation equipment. Staff members are physicians, both general practice and specialized; anesthesiologists; respiratory technicians; and critical care nurses with post-registration qualifications or experience in critical care nursing.

The critical care environment at the hospital where this study was performed includes the Intensive Care Unit (ICU), the Operating Room (OR), the Emergency Room (ER), the Cardiac Catheterization Laboratory (cath lab) and Telemetry (5 East).

1.6.5 STABLE ANGINA

Stable angina is chest pain caused by intermittent myocardial ischemia brought on by vasospasms of coronary arteries that may or may not be narrowed by atheroscloerotic plaque. Symptoms of stable angina are exacerbated by activity and relieved by rest and sublingual nitroglygerine. Angina pain differs between patients and has a characteristic pattern for each patient. Common descriptions of pain include squeezing, heaviness, or

burning in the chest; a pressure-like sensation or a feeling of strangulation or constriction around the chest. Some patients report that the pain radiates to the neck, jaw, left arm or between the shoulder blades. Some complain of nausea, headaches or dizziness. Section **2.5.3** in Chapter 2 refers to what causes stable angina (McAvory 2000:34-36).

1.6.6. UNSTABLE ANGINA

Angina is considered "unstable" if symptoms occur unpredictably, or if previously stable symptoms become more intense, last longer, or radiate to a different location than usual. Unstable angina (UA) is not always caused by exertion – it can also occur during rest or sleep – and it is not always relieved by rest and NTG. It indicates severe coronary vessel narrowing from atherosclerotic plaque. Because atherosclerotic lesions are inflexible, they limit the vessels' ability to dilate in response to NTG. UA indicates severe coronary vessel narrowing from atherosclerotic plaque, a small thrombus or arterial spasms (McAvoy 2000: 34-39). Sections **2.5.3** and **2.8.1** refer to what causes unstable angina.

1.6.7 MYOCARDIAL ISCHEMIA

This occurs where there is an insufficient supply of oxygen to the heart, usually related to coronary insufficiency, or decreased blood flow to the heart. On ECG, ST segment depression of 1 mm or more in two or more contiguous leads or T-wave inversion indicates myocardial ischemia. Contiguous leads reflect electrical activity in the same area of the myocardium. It is usually a reversible, transient event that is corrected when the blood and oxygen supply to the myocardium is restored. The ST segment and T-wave changes occur during periods of ischemia and may be seen only when the patient is experiencing pain. If these changes occur despite treatment, continued ischemia can damage the myocardium, resulting in an injury pattern on the ECG.

1.6.8 MYOCARDIAL INJURY

Myocardial injury ranges from an acute myocardial infarction (AMI) to minor myocardial damage. On ECG, elevation of the ST segment of 1 mm or more above the isoelectric line in two or more contiguous leads indicates myocardial injury. This indicates impaired blood flow through the coronary artery from vasospasms and probably thrombus development. ST segment elevation indicates an acute injury to the heart muscle and requires immediate treatment to reestablish blood flow to the myocardium before infarction (death) occurs. ST segment elevation typically develops into myocardial infarction (MI), but with rapid treatment, injury may be reversible.

1.6.9 MYOCARDIAL INFARCTION (MI)

Myocardial infarction, or cell death, is caused by prolonged interruption of myocardial perfusion. It is the destruction of enough cardiac tissue in order to cause clinical, electrical and/or biochemical changes in the body. Classic symptoms are crushing pain, squeezing pressure in the sternum with radiation to the left arm; diaphoresis; pallor; dyspnea; and nausea lasting 30 minutes or longer. The patient may describe this chest pain as the worst he has ever had and experience a sense of impending doom. Rest and NTG do not relieve symptoms of MI. Sometimes the pain of MI is not always excruciating. Many patients complain of a nagging sensation of heaviness, pressure or tightness. Other possible signs and symptoms of MI include dyspnea as the main complaint, indigestion, epigastric discomfort, right-sided chest pain, fatigue, light-headedness, back pain, and nausea and vomiting. Patients more likely to experience these symptoms instead of severe chest pain include women of any age and race, people with diabetes, and the elderly (McAvoy 2000:34-39; Dennison 1996: 107-117).

1.6.10 Q-WAVE MI

Also called a "transmural infarction", a Q-wave MI affects all three layers of the myocardial wall: epicardium, myocardium, and endocardium. Q-wave MI is associated

with complete coronary occlusion and infrequent coronary collaterals (Dewood 1988: 36F-38F; Church 2000: 36-37). On ECG, there will be ST segment elevation and may also be a pathologic or significant Q-wave which is greater than 0.04 seconds in width and 25% the height of the R-wave, which indicates the MI has already occurred. The Q-wave may decrease in size over time following an MI, but it will always be present in a 12-lead ECG.

1.6.11 NON-Q-WAVE MI

This type of MI does not affect all three layers and is more common with diabetics or those who have developed good collateral circulation. This type of MI is more difficult to diagnose. A non-Q-wave MI suggests that plaque or a thrombus is just beginning to reach critical form in the vessel wall, denoted by ST depression on ECG. In these patients the ST segment could flip up as blockage develops into full thickness injury. Although non-Q-wave MI was originally considered to be less serious than Q-wave MI, subsequent investigations have shown that the incidence of death and recurrent MI is as great as that associated with Q-wave infarction (Dennison 1996:110; Statland 1996: 43-51; McAvoy 2000: 34-39; DeWood 1988: 36F-38F; Sieck 2000: 4-8).

1.7 RESEARCH DESIGN AND METHOD

1.7.1 DESIGN

The design of this research study was a quantitative, descriptive and contextual study in which a questionnaire was used as instrument. The knowledge of critical care nurses was explored and described according to their own rating, with the goal being to plan a four hour in-service training session on ACS for all critical care nurses in a state hospital in East Texas. The aim of this in-service training session is to increase nurses' knowledge about ACS, which will improve their nursing care for ACS patients.

1.7.2 METHOD

The research method is summarized in ***Table 1.1*** and will be discussed in detail in Chapter 3.

Table 1.1 Research Design and Method

AIM	DATA GATHERING	SAMPLING & POPULATION	VALIDITY & RELIABILITY	DATA ANALYSIS
Explore and describe the respondents' perceptions as to how they rate their knowledge regarding aspects of ACS such as its pathophysiology, current available treatments, rationales for such treatments and nursing care and to formulate an in-service training session on ACS.	Implementation of a questionnaire	Convenience sample of voluntary critical care nurses of all qualifications, regardless of staff appointment, in a state hospital in East Texas.	Literature review, professional experts' critique, Cronbach's alpha and factor analysis.	Descriptive and inferential statistics.

1.8 SUMMARY

Acute coronary syndromes: whether it is newly diagnosed unstable angina or an acute myocardial infarction, the goal of therapy is pain relief and the limitation of myocardial

ischemia and (when relevant) reinfarction. Treatment needs to be accurate, rapid and closely monitored.

For the critical care nurse, this rapidly evolving field regarding ACS presents an informational challenge. Challenging as it may be, nevertheless a sound knowledge base about ACS – its pathophysiology and current approaches to treatment – are necessary if the critical care nurse is to make judgments and decisions, identify patient needs and determine the best nursing care for that patient.

This research study explored and described the critical care nurses' knowledge according to their own rating regarding ACS, and the information obtained from the research study and the literature review will be used to formulate an in-service training session for the critical care nurses about ACS and current treatments.

CHAPTER 2

LITERATURE STUDY

2.1 INTRODUCTION

As stated in Chapter 1, nursing practice today – especially critical care nursing – must involve the use of high-level critical thinking skills within the nursing process. Independent judgments and decisions, which are one of the outcomes of the critical thinking process, arise from a sound knowledge base and the ability to integrate this knowledge within the context in which it is gathered. The following literature study serves as a background to this research study, but at the same time covers information that the researcher believes is important for critical care nurses to know in order to provide competent nursing care for acute coronary syndrome patients.

2.2 DEFINITION OF ACUTE CORONARY SYNDROMES

Acute Coronary Syndromes (ACS) encompasses a spectrum of patients who present with chest pain or other symptoms caused by myocardial ischemia or infarction. The unification of these symptoms of coronary artery disease under the term *Acute Coronary Syndromes* reflects the understanding that these symptoms are caused by a similar pathophysiology, characterized by erosion or rupture of atherosclerotic plaque, leading to intravascular thrombosis and impaired myocardial blood supply (Gylys & Gold 2000: 3; Simoons, Boersma, Vandeerzwaan, Deckers 1999: 1-4). ACS includes subsets of high-risk patients – unstable angina, non-Q-wave MI, and Q-wave MI – who need aggressive treatment to prevent a MI from occurring or prevent the reoccurrence of a MI if one has already occurred.

The outcome of these patients, especially whether the myocardial ischemia recovers fully or results in minor or major myocardial necrosis, depends on several factors -- the

presence or absence of mechanical obstruction by the plaque and its contents; the amount and extent of thrombus formation; the oxygen demand of the heart; the extent and duration of ischemia; vascular tone; and the degree of collateral circulation (Jesse 1999: 213-221).

2.2.1 UNSTABLE ANGINA, NON-Q-WAVE MI, Q-WAVE MI

The reader should refer to section 1.6, *Key terms and concepts for this research study* for definition of the subsets of ACS: unstable angina, non-Q-wave MI and Q-wave MI.

2.3 THE CLOTTING CASCADE

Understanding the processes of the clotting cascade is essential for comprehending the beginning stages of ACS and understanding when specific medications exert their effects.

2.3.1 FACTORS TRIGGERING THE INITIATION OF THE CLOTTING PROCESS

Clot formation occurs when the body initiates either the intrinsic or extrinsic pathway. When a blood vessel is lacerated or crushed from an outside source, the body initiates the **extrinsic pathway** to activate hemostasis. The vessel constricts to slow blood loss, and platelets adhere to the area and combine with **collagen**, the underlying connective tissue in the exposed **epithelium** (the layer of cells that forms a blood or lymphatic vessel's inmost lining, as well as that of the heart) of the damaged vessel, to form a temporary plug or clot over the area. When platelets combine with collagen, they stimulate platelet aggregation and the release of **thromboplastin** (also called "tissue factor", found in the interstitial compartments), which activates the clotting cascade.

In the **intrinsic pathway**, internal forces expose the blood to collagen fibers underlying the endothelium in the blood vessels. This exposure activates clotting factors, which likewise initiate the clotting cascade. Forces that initiate the intrinsic pathway include

venous stasis (blood pools in vessels, allowing clotting factors to reach levels high enough to initiate clotting), diseases associated with hypercoagulability, and internal vessel disruption or injury. Vessel disruption or injury can occur spontaneously, such as in the disruption or erosion of atherscleratic plaque, oxidation of low density lipoprotein (LDL) cholesterol which causes punctures in the endothelium, high shear stress resulting from hypertension, or sharp bends in the vessels, or it can occur as a result of **percutaneous cornary intervention,** known as **PCI** (Church 2000: 37; Fuster, Fayad & Badimon 1999: 5-9).

Whether intrinsic or extrinsic, the pathway results in **prothrombin activator**. In the presence of calcium, this activator converts prothrombin to **thrombin**. Thrombin then converts plasma **fibrinogen** to **fibrin threads** that surround the blood cells and platelets at the injury site, forming a **clot,** or **thrombus**. When thrombin is released, it also stimulates the production of **fibrin-stabilizing factor,** which increases the strength and effectiveness of the fibrin threads around the clot. ***Figure 2.1*** on page 16 is a schematic representation of these basic steps in clot formation, as drawn by this author.

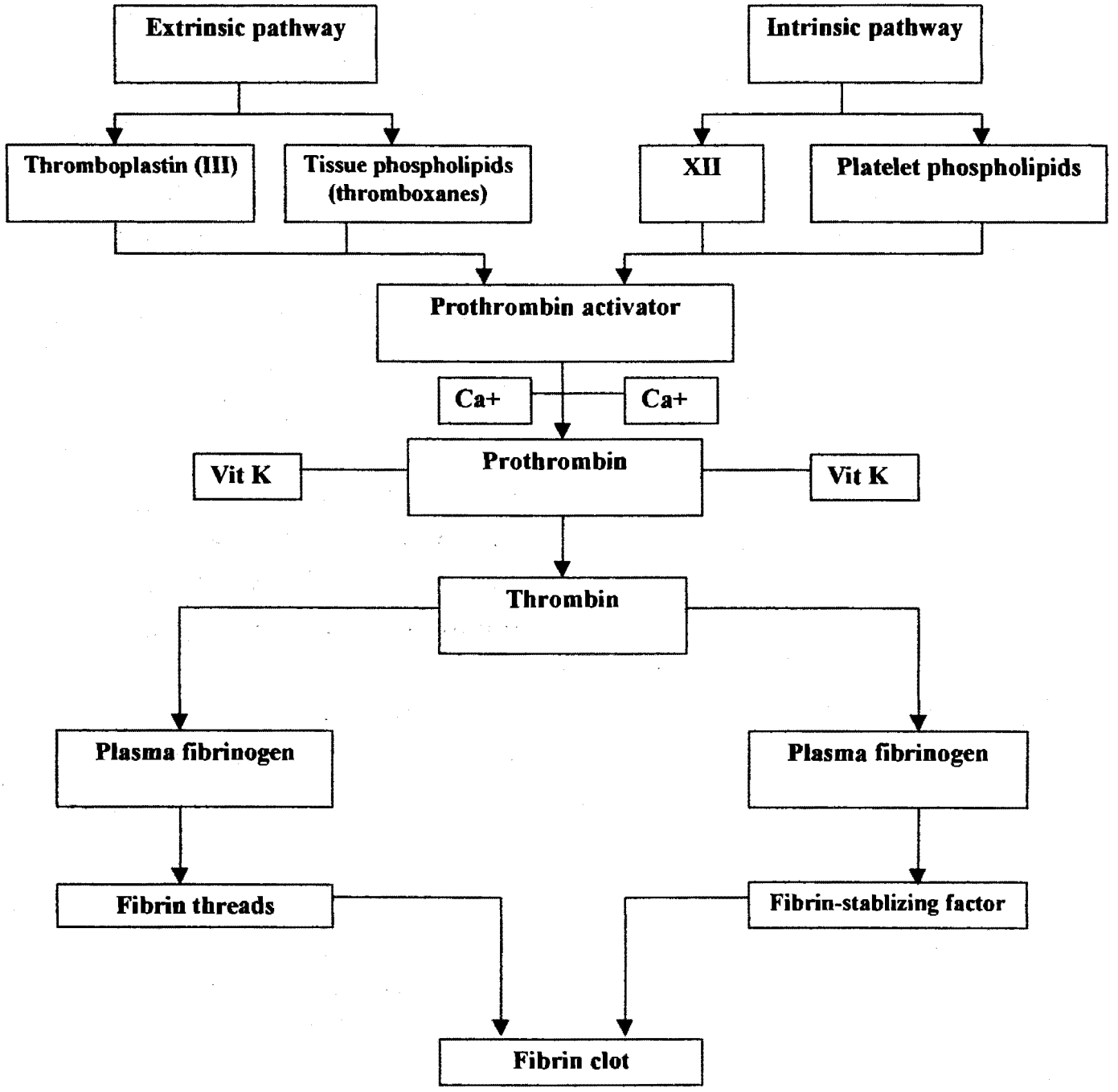
2.3.2 PLATELET AGGREGATION AND PLATELET PLUG FORMATION

Platelets have a propensity for adhering to surfaces, but they do not adhere to normal endothelial cells lining blood vessels. Injury to a vessel disrupts the endothelium and exposes the underlying connective tissue collagen molecules. An adhesive plasma protein called von Willebrand factor (vWF), secreted by the endothelial cells, binds to collagen, and then platelets bind to it, forming an initial hemostatic plug.

The initial platelet adherence to collagen triggers the platelets to release the contents of their secretory vesicles which contain a variety of chemical agents, one of which is **ADP** (adenosine diphosphate). When platelets are activated by agents such as collagen,

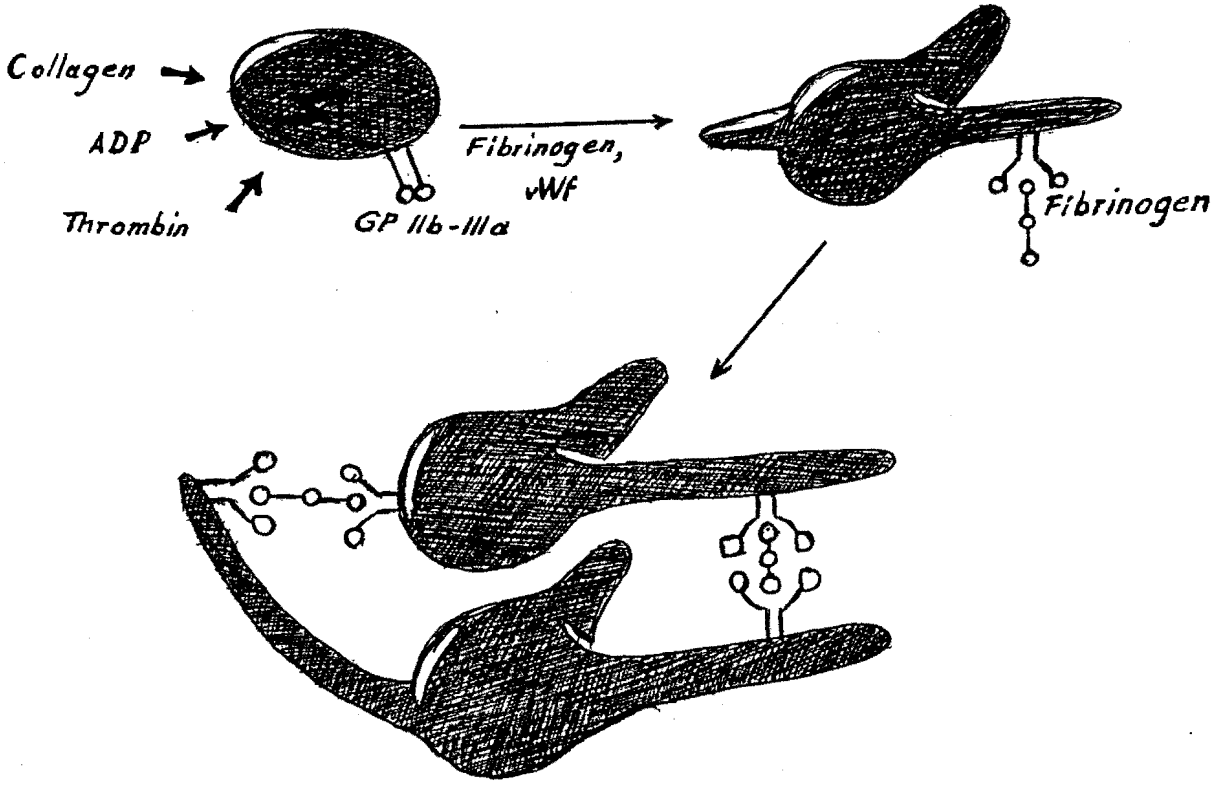
thrombin or ADP, they *change shape*, causing new platelets to adhere to old ones. When platelets are activated and change shape, **glycoprotein IIb/IIIa receptors** on the

Figure 2.1 Understanding clot formation and lysis



platelets' surfaces are activated to become receptors for adhesive plasma proteins, primarily soluble fibrinogen and vWF. Fibrinogen and vWF bind with these receptors, causing **platelet aggregation**. This cross-linking of activated GP IIb/IIIa receptors on adjacent platelets by adhesive plasma proteins is the final common pathway to platelet aggregation and vascular thrombosis. On unstimulated, normal shaped discoid platelets, GP IIb/IIIb receptors are unable to bind to fibrinogen. ***Figure 2.2***, redrawn by this author, diagrams this process (Harrington 1999: S277).

Figure 2.2 Cross-linking of activated GP IIb/IIIa receptors on platelets



platelet adherence to collagen causes the conversion of arachidonic acid in the platelet plasma membrane to **thromboxane A2**, which powerfully stimulates platelet aggregation and secretion of platelet granules, helping to further along thrombus formation.

In summary, the platelet plug is built up extremely rapidly and is the main sealer of breaks in the vessel wall. When the platelets are exposed to collagen and other

substances, they are activated, change shape, and their GP IIb/IIIa receptors bind with soluble fibrinogen causing platelet aggregation. Platelet aggregation is necessary for the next hemostatic event – blood coagulation and thrombus formation (Harrington 1999: S276-S285; MacCallum, Hanlon & Byrne 1999: 34-39; Vander, Sherman & Luciano: 1990: 689-695).

2.3.3 THE CLOTTING CASCADE AND THROMBUS FORMATION

Blood coagulation, or clotting, is a transformation of blood into a solid gel called a clot, or thrombus. Clotting is the dominant hemostatic defense. It occurs around the initial platelet plug, when the plasma protein fibrinogen is converted to fibrin. The function of the clot is to support and reinforce the platelet plug and to solidify the blood that remains in the wound channel.

Prothrombin, an inactive precursor of thrombin, circulates in the blood continuously. At the site of vessel damage, prothrombin is converted to **thrombin**. Thrombin catalyzes a reaction which causes **fibrinogen**, a large rod-shaped protein produced by the liver and always present in the plasma of a normal person, to be converted to **fibrin**. As mentioned above, soluble fibrinogen first binds with activated GP IIb/IIIa receptors on the platelets' surfaces. Initially fibrin is a loose mesh of interlacing strands, but rapidly becomes stabilized and strengthened. This stabilizing process of loose fibrin is catalyzed by an enzyme in plasma known as **factor XIII**, which is converted from inactive form to active form (**XIIIa**) by thrombin. Thus thrombin catalyzes the formation of fibrin *and* the active factor XIII, which will stabilize the fibrin network.

In the process of clotting, erythrocytes and other cells are caught in the fibrin meshwork, but the main components of the clot are fibrin and platelets, and clotting can occur in the absence of all cells except platelets.

The enzyme that catalyzes the conversion of prothrombin to thrombin is always present in plasma in its inactive form, and is converted to *its* active form by *another* enzyme. In

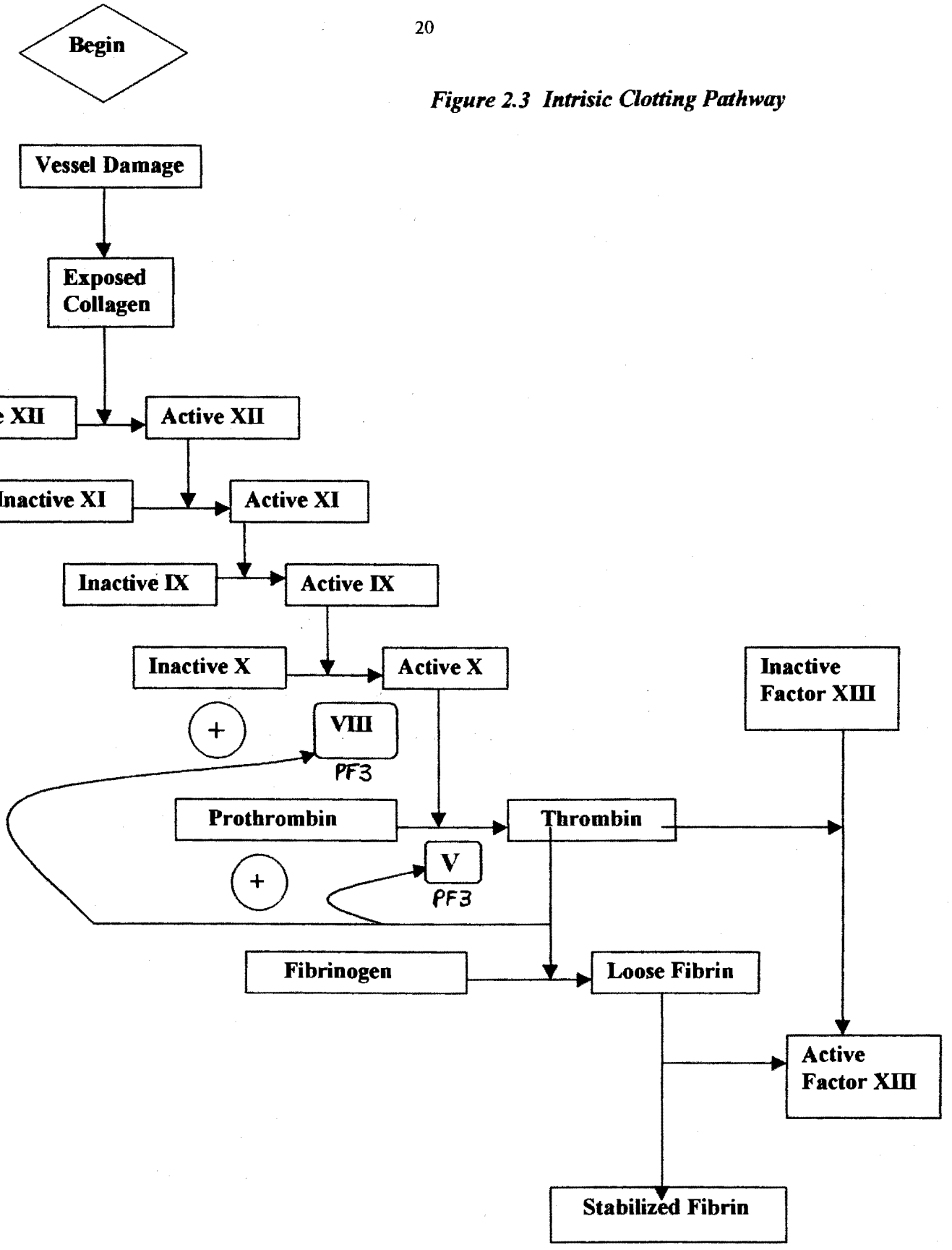
other words, there is a **cascade** of plasma proteins, each one normally an inactive proteolytic enzyme until it is activated by the previous one in the sequence. The first factor in the sequence is called **factor XII**. The question is -- what activates factor XII? When factor XII has contact with a damaged vessel surface, specifically with collagen fibers underlying damaged endothelium, then inactive XII is converted to active XII **(XIIa)** and thus the clotting cascade begins.

Two of the circulating clotting factors, **VIII** and **V**, do not form actual links in the clotting cascade. They work as cofactors in two of the cascade reactions along with **PF3**, and their activation is stimulated by thrombin. **PF3** is a phospholipid exposed in the plasma membranes of the aggregated platelets.

Figure 2.3 on page 20 is a diagram of the intrinsic clotting pathway, redrawn by the author (Vander et al 1990: 693). Thrombin profoundly stimulates platelet aggregation, in addition to its three other functions (stimulates the activation of factors V, VII and PF3: converts fibrinogen to fibrin; and converts inactive factor XIII to active factor XIII which then stabilizes the fibrin meshwork). Thus, once thrombin formation has begun, the overall reactions leading to clotting occur rapidly, due to the positive feedback effects of thrombin.

Thus we see that when the epithelial lining of a blood vessel is damaged, thereby exposing its underlying collagen layer, both platelet aggregation and the clotting cascade are activated, leading to the formation of a blood clot, or thrombus. The degree and duration of the thrombotic insult are major determinants of the clinical presentation of ACS. For example, persistent ST-segment elevation, usually accompanied by elevation of cardiac markers, is indicative of complete and prolonged ischemia distal to the occlusion. In patients without persistent ST-segment elevation but with other ECG (transient ST-segment elevation, ST-segment depression, T-wave inversion) or biochemical (elevation of cardiac markers) abnormalities suggestive of cardiac origin of chest pain, myocardial ischemia is of more limited duration and it is usually caused by

Figure 2.3 Intrisic Clotting Pathway



partially occlusive thrombi (Church 2000: 34-42; Harrington 1999: S276-S285; Gylys & Gold 2000: 3-13; Breen 2000: 59-62; Vander et al 1990: 690-694).

2.3.4 PHARMACOLOGIC AGENTS INTERFERING WITH THROMBUS FORMATION

It is important that the nurse have a thorough understanding of the pharmacologic agents used to treat ACS, including the drugs' actions, side effects, contraindications and rationales for use. Such knowledge enables the nurse to provide competent critical nursing care.

2.3.4.1 NURSING CARE

With all of the following antithrombotic agents, the nurse should check for signs of bleeding such as bleeding or oozing from a wound or venipuncture sites; nosebleeds; blood in urine, stools, sputum, or emesis; unexplained bruising; dizziness or confusion; a rapid or unusual heartrate; chest pain or shortness of breath; nausea or vomiting. The nurse should monitor the CBC, clotting times, renal and liver function tests and report any abnormalities to the doctor (Karch 2000: 68, 146, 330, 418-419, 1093, 1136, 1141).

2.3.4.2 ASPIRIN

Aspirin inhibits platelet production of **thromboxane A2**, a platelet-specific prostaglandin. As mentioned in section 2.3.3, when platelets adhere to collagen after initial vessel damage, this triggers the conversion of arachidonic acid in the platelet plasma membrane to thromboxane A2, which powerfully stimulates platelet aggregation. Thus, aspirin inhibits platelet aggreatation, and its effects last approximately eight days. There is no tolerance build-up to aspirin in the body and spontaneous, life-threatening bleeding is rare. It is inexpensive, thus cost-effective (Verheugt 1999: 20-23).

It is important that the nurse knows that aspirin should not be given if the patient has allergies to salicylates or nonsteroidal anti-inflammatory drugs (NSAIDs), bleeding and/or coagulation disorders, bleeding ulcers, chickenpox, influenza, surgery within one week, impaired renal function or is pregnant or lactating (Karch 2000: 144-145).

2.3.4.3 SECOND GENERATION PLATELET INHIBITORS

Second-generation platelet inhibitors include the adenosine diphosphate (ADP) inhibitors **clopidogrel (Plavix)** and **ticlopidine (Ticlid)**. The initial platelet adherence to collagen triggers the platelets to release the contents of their secretory vesicles, one of which is ADP. When platelets are activated by agents such as collagen, thrombin or ADP, they change shape, leading to platelet aggregation. Colpidogrel and ticlopidine block ADP receptors on platelets, thus preventing platelet aggregation. They are less potent than the GP IIb/IIIa inhibitors, and are often used along with aspirin for one month post-stent placement. Unlike aspirin, these agents provide a broader inhibition of platelet aggregation but do not have any effect on thomboxane A2 or prostaglandin synthesis as does aspirin.

Ticlopidine was originally used, but there has been a significant incidence of bone marrow suppression associated with its use, and consequently it is usually recommended only if the patient has an intolerance to aspirin and to the continued use of clopidogrel. Ticlopidine also prolongs bleeding time. It is used for maintenance, not acute, therapy.

Clopidogrel may be used with aspirin or as an alternative to aspirin to decrease platelet aggregation, and is indicated as part of maintenance, not acute, therapy. Unlike aspirin or ticlopidine, clopidogrel does not prolong bleeding time.

It is important that the nurse not give these agents if the patient is allergic to them, has neutropenia, thrombocytopenia, hemostatic disorders, bleeding ulcer, intracranial bleeding, severe liver disease, or is lactating. Although not as common as with aspirin,

these drugs may cause GI upset, so the nurse should give them with food (Futterman & Lemberg 2000:73; Verheught 1999: 20-23).

2.3.4.4 GLYCOPROTEIN IIb/IIIa RECEPTOR INHIBITORS

Third-generation platelet inhibitors are the glycoprotein IIb/IIIa receptor inhibitors (GP IIb/IIIa inhibitors) and currently include **abciximab (ReoPro), eptifibatide (Integrelin)** and **tirofiban (Aggrastat)**. These agents are indicated for unstable angina and non-Q-wave MI and to prevent thrombotic and ischemic complications in PCI. These drugs inhibit platelet aggregation by binding with the GP IIb/IIIa receptor site on the platelet instead of fibrinogen and other ligands, thereby preventing or stopping platelet aggregation. GP IIb/IIIa inhibitors are most effective because they completely block fibrinogen's ability to bind platelets.

These agents can be given alone or in combination with heparin. Recent study results indicate that using GP IIb/IIIa inhibitors with heparin and reduced-dose thrombolytic therapy improves vessel patency in acute non-Q-wave MI. All three are given intraveneously, but oral GP IIb/IIIa inhibitors are being researched (Gylys & Gold 2000: 3-4; MacCallum, Hanlon & Byrne 1999: 34-35; Miracle & Woods 2000: 19).

These agents increase bleeding time, but after discontinuation, the effects on the platelets disappear within hours. Used along with heparin and aspirin, or with low-molecular weight heparin and aspirin in ACS patients, they show a reduction of MI and death up to 25 times compared to heparin and aspirin alone. Their beneficial effects are seen in patients undergoing PCI and also in those without such procedures. They are expensive (up to $1000 per treatment), which presently makes them prohibitive for routine use. Oral GP IIb/IIIa inhibitor agents, under investigation currently, have a longer plasma life (Verheught 1999: 20-23).

The nurse should not give GP IIb/IIIa inhibitors if the patient is allergic to them, if he has neutropenia, thrombocytopenia, hemostatic disorders, bleeding ulcer, intracranial

bleeding, uncontrolled or severe hypertension, major trauma or surgery within six weeks, aortic aneurysm, aortic dissection, or is pregnant. The nurse should administer these agents cautiously if the patient is lactating.

2.3.4.5 HEPARIN

Heparin has been most widely used for ACS up until now. It combines with antithrombin III, a naturally occurring substance in the body, the function of which is to partially block the effects of thrombin. Heparin increases this effect *at least a thousand fold*, and that in turn greatly slows down the conversion of fibrinogen to fibrin. Heparin's powerful effects on thrombin also greatly increase the risk of bleeding. Its effects are measured by prolongation of the activated partial thromboplastin time (PTT). Therapeutic levels range from 50 to 90 seconds (Sieck 2000:7; Verheugt 1999: 20-23).

For ACS, heparin can be administered intraveneously with a loading dose of 5000 units, followed by a continuous IV infusion of 1000 units/hour. Clotting times should be monitored and the heparin dose should be increased or decreased according to the doctor's orders to maintain a PTT 1.5 to 3 times the control value (usually between 50 to 90 seconds). Heparin also may be given subcutaneously (SQ), usually every 8 hours.

The nurse should not give heparin if the patient is allergic to it, has thrombocytopenia, uncontrolled bleeding, or cannot be monitored regularly with blood coagulation tests. Heparin should not be given during labor or during the immediate postpartum period, or to women older than 60 years who are at high risk for hemorrhaging. It should not be given to patients who have had recent trauma or surgery. Treatment for heparin overdose is 1% protamine sulfate, and the nurse should know that drug dosage of protamine sulfate is based on blood coagulation studies (Gurfinkel 1999: 115-117; Karch 2000: 587-589; Sieck 2000: 7-9).

2.3.4.6 LOW MOLECULAR WEIGHT HEPARIN

Low molecular weight heparin (LMWH) intereferes with the clotting cascade by blocking the conversion of factor X to its activated form. As a result, prothrombin activator cannot activate prothrombin to be converted to thrombin. Enoxaparin (Lovenox) is the LMWH currently being used for ACS. LMWH consists of a smaller molecule of the compounds of which heparin is a mixture. The pharmacological response of LMWH is more predictable than heparin and does require monitoring the PTT. It can be administered subcutaneously, which makes it possible for outpatient use. Full anticoagulation can be achieved with one to two SQ injections daily. LMWH is associated with a lower incidence of thrombocytopenia, it has a longer half-life and causes fewer bleeding complications than standard heparin. Because of its ease in administration and lack of need for monitoring, it has become the preferred treatment for ACS.

Recent studies have shown improvements in mortality for patients admitted with unstable angina or non-Q-wave MI who received LMWH (the ESSENCE trial). These patients also had reduced requirements for diagnostic coronary catheterizations and revascularizations. Follow-up studies on the ESSENCE trial are being performed to investigate the effect of prolonged use of LMWH beyond the acute phase of unstable angina (Scirica 1999: 115-116).

The nurse should not give LMWH if the patient is allergic to it or allergic to heparin or pork products, or in the case of severe thrombocytopenia or uncontrolled bleeding. The nurse should give LMWH cautiously if the patient is pregnant, lactating or has a history of GI bleed. It is given subcutaneously, usually every 12 hours, in the abdominal area at least two inches from the belly button. (Gurfinkel 1999: 115-117; Sieck 2000: 7-9; Roberts 1999: 7-9; Karch 2000: 66-67, 145-146, 330, 460-461, 588-589, 1136, 1140-1141).

Figure 2.4 is a schematic illustration, redrawn by this author, which shows where heparin and low molecular weight heparin exert their antithrombotic effects on the coagulation cascade (Sieck 2000: 7).

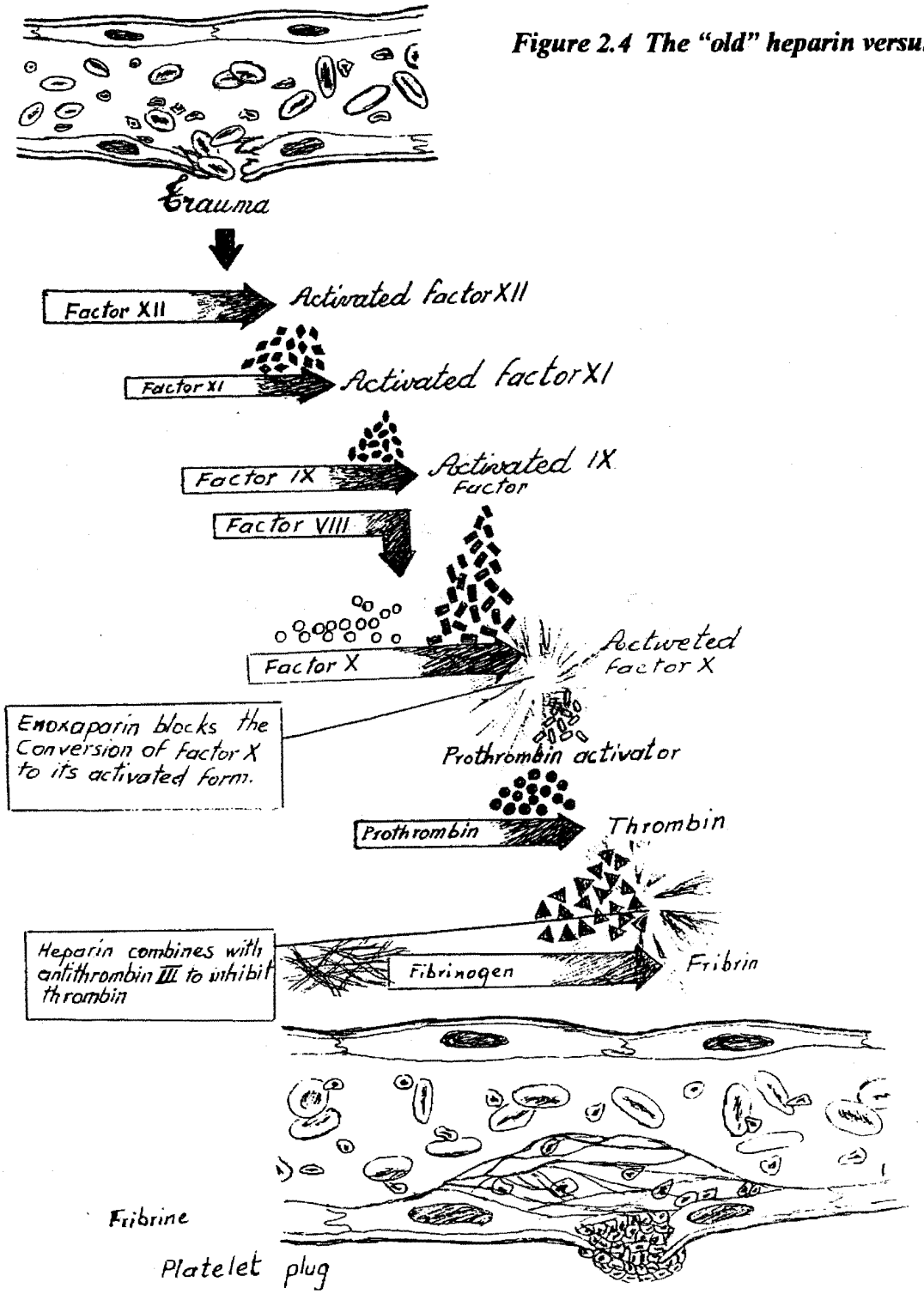


Figure 2.4 The "old" heparin versus the "new"

2.4 ENDOTHELIAL DAMAGE

The endothelium is the layer of cells that forms a blood or lymphatic vessel's inmost lining, as well as that of the heart. The endothelium does more than serve as a semi-permeable between the blood supply and the smooth muscle cells of the vascular wall. It performs many other vital functions as well – it regulates smooth muscle tone, cell proliferation and synthesis and release of vasoactive chemicals; its nonadhesive surface ensures unimpeded blood flow; and in healthy persons, it exhibits antithrombotic and fibrinolytic properties.

By regulating the production of several vasoactive substances, the endothelium maintains equilibrium between vasodilation and vasoconstricition, coagualation and anticoagulation, and cellular growth and inhibition. **Endothelial damage** always causes a shift *toward* vasoconstriction, as well as *toward* blood coagulation and cellular growth (Rockett 1999: 44-46).

2.4.1 FACTORS LEADING TO ENDOTHELIAL DAMAGE

Endothelial damage can occur as a result of several factors. Chronic minimal damage can begin merely as a result of the disturbance of blood flow patterns at vessel bending points and near bifurcations of arterial trees. These local shear forces are enhanced by hypertension, resulting in high shear forces which can damage the endothelium. Microtears in the vessel wall can occur from distension and venous stasis as well as from direct insult from trauma (especially fractures), surgery (including PCI), sepsis and burns. General anesthesia can decrease vascular tone, disrupting the endothelial lining.

Endothelial damage causes an imbalance in several vasoactive substances, which will be discussed in the next section, and also exposes collagen, the underlying connective tissue of the epithelium (Breen 2000: 60-61; Church 2000: 35-36).

2.4.2 FUNCTIONS AND DYSFUNCTIONS OF THE ENDOTHELIUM

2.4.2.1 DILATION

Several substances released from the endothelium mediate blood vessel dilation, two of which are **nitric oxide** and **prostaglandin**. When blood pressure is normal, the endothelium releases small amounts of nitric oxide continuously, overcoming the vessel's natural tendency to constrict, and dilating the vessel enough to maintain homeostasis. When the endothelium is damaged, sufficient amounts of nitric oxide may not be released and the blood vessels do not relax adequately, which can cause elevated blood pressure.

2.4.2.2 CONSTRICTION

The endothelium also produces chemicals that cause vasoconstriction. One of these chemicals, **angiotension-converting enzyme (ACE)** converts peptide angiotension I to angiotension II, a potent vasoconstrictor. Angiotension II *blocks* the release of nitric oxide and prostaglandin. Thus, ACE has a dual effect: converts angiotension I to angiotension II, a powerful vasoconstrictor; and interferes with two vasodilators (nitric oxide and prostaglandin).

Some ACE circulates in the blood continuously, but most of it is in the endothelial tissues. If you could physically take out ACE, angiotension II levels would drop, while nitric oxide and prostaglandin levels would rise, favoring dilation and resulting in a decreased blood pressure (Rockett 1999: 44-49; Weissberg 2000: 247).

2.4.2.3 MONOCYTE AND PLATELET ADHESION

Monocytes, from which macrophages come, cause plaque to be deposited on the endothelium. This will be discussed in the following section on plaque formation. The endothelium helps to regulate the number of inflammatory cells, such as monocytes and macrophages, that bind to the blood vessel wall. When the endothelium is damaged,

more inflammatory cells bind to the vessel wall, thus leading to an increase in plaque formation.

Nitric oxide and prostaglandin also inhibit platelet aggregation and adhesion, and a decrease in these chemicals due to endothelial damage results in platelet aggregation and adhesion, which can lead to thrombus formation. Concurrently, when nitric oxide levels fall, there is a corresponding increase in the oxidation of circulating low-density lipoprotein (LDL) cholesterol. When LDL cholesterol is oxidized, it punctures the endothelial cell walls, causing further damage. Where such damage occurs, platelet aggreation and adhesion also occur (Rockett 1999: 44-49; Karch 2000: 14-15).

2.4.2.4 CELL GROWTH AND INHIBITION

The endothelium regulates several chemical mediators that affect the cellular growth of **vascular smooth muscle (VSM).** Nitric oxide and prostaglandin *inhibit* VSM growth, and angiotension II *stimulates* VSM growth. An imbalance can alter the thickness of both the blood vessel wall and its lumen. This **vascular remodeling** can result in a thickened vessel wall and a narrower-than-usual lumen, leading to hypertension. This type of vascular remodeling can predispose people to other problems such as atherosclerosis, MI, stroke and renal failure (Rockett 1999: 44-49; Fuster et al 1999: 5-9; Weissberg 2000: 247-249).

2.4.2.5 ACE INHIBITOR DRUGS

ACE inhibitor agents are a class or drugs that inhibit tissue-ACE release and allow nitric oxide and prostaglandin levels to rise. For ACS patients, these drugs help to lower blood pressure, inhibit vascular smooth muscle growth, inhibit platelet aggregation and adhesion, and possible slow oxidation of LDL cholesterol. ACE inhibitor agents will be discussed in section 2.9 covering drugs used for treating ACS.

2.4.2.6 SUMMARY OF ENDOTHELIAL DAMAGE

Endothelial damage results in the following phenomena: impairment of vasoregulation, monocyte adhesion, platelet adhesion and vascular smooth muscle growth. These may result in vasoconstriction, hypertension, plaque formation and thrombus formation (Rockett 1999: 44-49; Fuster, Fayad & Badimon 1999: 5-9; Breen 2000: 58-62).

2.5 PLAQUE FORMATION

2.5.1 INTRODUCTION

Atherosclerosis begins as a subendothelial accumulation of lipid rich, monocyte-derived foam cells and associated T-cells which form a non-stenotic fatty streak. As this progresses, the lesions take the form of a core of cholesterol esters surrounded by an endothelialized fibrous cap containing vascular smooth muscle cells (VSMC) and inflammatory cells (mostly macrophages with some T-cells and mast cells). In advanced lesions, there may be new blood vessels and deposits of calcium.

2.5.2 HOW ATHEROSCLEROTIC PLAQUE BEGINS

The earliest detectable physiological manifestation of atherosclerosis is a decreased production of nitric oxide. This is even seen in children with hypercholesterolemia, and is consistent with the hypothesis that increased levels of circulating atherogenic lipoproteins may lead to endothelial dysfunction and subendothelial lipid accumulation. It should be noted, however, that it has not been yet proven which comes first – the endothelial damage or the increased circulating lipoproteins.

As a brief review, endothelial damage leads to a decrease in the production of nitric oxide and prostaglandin, causing vasoconstriction, platelet aggregation, vascular smooth muscle proliferation and monocyte adhesion. These changes brought on by endothelial damage are closely linked with plaque formation, as we shall see.

When levels of circulating lipoproteins in the blood increase, inflammatory **monocytes** are activated and migrate from the arterial lumen to the subendothelial space. It is important to note that these inflammatory cells will not accumulate in the intima in the *absence* of lipids. Monocytes mature into **macrophages**, which have the ability to injest modified lipids and become **macrophage foam cells**. Foam cells can also be appropriately termed as "intracytoplasmic droplets of cholesterol", because the macrophages injest the lipids.

As the number of foam cells increases along with associated T-cells, a **fatty streak** is formed on the arterial lumen wall. The fatty streak is an early atherosclerotic lesion (designated Type II by the American Heart Association) and is not associated with evidence of any structural damage to the endothelium.

As the fatty streak lesion increases in size, it forms a lipid-rich core (designated as Type IV and Va by the American Heart Association) containing cholesterol esters. The vascular smooth muscles respond by forming a **fibrous connective tissue cap** over this lipid core of atherosclerotic plaque. This cap separates the highly thrombogenic lipid-rich core from the arterial lumen and from circulating platelets and proteins of the coagulation cascade. It also gives structural stability to the lesion. This cap also contains some inflammatory cells, most of which are mainly macrophages with some T-cells and mast cells. These macrophages are very active, producing procoagulant tissue factor and many other inflammatory cell mediators, such as tumor necrosis factor, interleukins and metelloproteinases. Some of these macrophages contain cholesterol (foam cells). Vascular smooth muscle cells are the only cell capable of making this cap, and thus they play an important part in maintaining plaque stability and protecting against plaque rupture and consequent thrombosis (Davies 2000: 361-366; Fuster et al 1999: 5-9; Weissberg 2000: 247-252).

2.5.3 HOW ATHEROSCLEROSIS LEADS TO ACS SYMPTONS

The first way atherosclerosis leads to symptoms is if the lesion becomes large enough to restrict blood flow to the point that the oxygen supply cannot meet the demand. This results in tissue ischemia, such as in **chronic stable angina**.

It must be emphasized that plaque growth does not always lead to lumen stenosis. The arteries can remodel and accommodate the expanding atherosclerotic lesion and still maintain a normal or adequate lumen diameter. Thus, atherosclerotic lesions may or may not be clinically silent.

The second way atherosclerosis leads to symptoms is if the lesion ruptures or erodes (this will be discussed in Section 2.6). This triggers platelets to rapidly accumulate and intravascular thrombosis will occur, leading to acute coronary syndromes of **unstable angina** and **myocardial infarction** (Simoons, Boersma & Vandeerzwaan & Deckers 1999: 1-4).

2.5.4 SUMMARY OF PLAQUE FORMATION

To summarize, increased levels of circulating lipoproteins in the blood correlate with endothelial damage and dysfunction. It is not proven yet which happens first. The increased lipid accumulation is followed by inflammatory cells, particularly monocytes, being activated and migrating to the subendothelial space. As activated monocytes adhere to the area, they mature to macrophages which ingest modified lipids and become foam cells. These foam cells initially form a fatty streak, and as the fatty streak lesion increases in size, it forms a lipid-rich core surrounded by a fibrous connective tissue cap. This cap separates the core from the arterial lumen and circulating platelets and proteins, and also gives structure and stability to the core (Fuster et al 1999: 5-9; Davies 2000: 361-364; Weissberg 2000: 247-251).

2.6 PLAQUE RUPTURE

2.6.1 INTRODUCTION

Plaques with a lipid-rich core and an unstable cap are more likely to rupture. A thicker cap is more able to resist mechanical stresses in the body. However, the most important determinant of plaque stability is the *composition,* not necessarily thickness, of the fibrous cap. A larger number of inflammatory cells and a relative smaller number of vascular smooth muscle cells will more easily lead to plaque rupture. The inflammatory cells weaken and destroy the fibrous cap, and vascular smooth muscle cells are not equipped to compensate. The reverse is also true – a smaller number of inflammatory cells and a relative larger number of vascular smooth muscle cells will lead to increased cap stability and less likelihood of plaque rupture.

Therefore, it is not so much the size of the plaque as it is *plaque composition* that determines the outcome. A person can have a small, hemodynamically insignificant atherosclerotic plaque that is clinically silent, and without any warning it can rupture.

When the fibrous cap of an atherosclerotic lesion ruptures, it exposes the highly thrombogenic collagenous matrix and lipid core to the circulating blood and leads to platelet accumulation and aggregation. The clotting cascade is activated, and this leads to fibrin deposition, thrombus formation, and, at the most extreme, vessel occlusion. Small lesions can progress acutely to severe stenosis or total occlusion, and this progression is unpredictable and episodic.

However, vessel occlusion is not inevitable, and it is now clear that episodes of silent, subclinical plaque rupture occur frequently in patients with atherosclerosis. These episodes of non-occlusive plaque rupture trigger the recruitment of new vascular smooth muscle cells to the area, and form a new fibrous cap over the thrombus, thereby increasing the size of the lesion. Thus the size of the atherosclerotic lesions increases as a consequence of repeated episodes of rupture and repair (Davies 2000: 361-366; Fuster et al 1999: 55-59; Weissberg 2000: 247-252).

2.6.2 HOW PLAQUE RUPTURE OCCURS

Plaque disruption depends on both passive and active phenomena. **Passive plaque disruption** usually occurs where the fibrous cap is the thinnest and most heavily infiltrated by foam cells, and therefore the weakest. Vulnerability to rupture depends on the location, size and consistency of the lipid core; and on blood-flow characteristics, especially the impact of the flow on the proximal part of the plaque.

Active plaque disruption involves the action of macrophages, which are capable of degrading the fibrous cap by phagocytosis or secretion of proteolytic enzymes. These enzymes may weaken the fibrous cap and predispose it to rupture (Davies 2000: 361-366; Fuster et al 1999: 55-59; Weissberg 2000: 247-252).

Plaques at risk for future thrombotic events are characterized by:

- large lipid cores (greater than 50% overall plaque volume)
- thin caps
- high density of macrophages, capable of degrading the fibrous cap
- low density of smooth muscle cells in fibrous cap

2.7 VASCULAR REMODELING

Plaque rupture and thrombus formation does not always lead to vessel occlusion. Research has shown that it is clear that episodes of silent, subclinical plaque rupture occur frequently in patients with atherosclerosis. These episodes of non-occlusive plaque rupture trigger new vascular smooth muscle cells to migrate to the area and form a new fibrous cap over the thrombus, thereby increasing the size of the lesion. Thus the size of atherosclerotic lesions increases as a consequence of repeated episodes of rupture and repair (Fuster et al 1999: 5-9; Weissberg 2000: 247-252).

2.8 STATIN DRUGS

Statins are a class of drugs that reduce the rate of progression of pre-existing lesions and the incidence of new lesion formations by lowering serum cholesterol and serum LDLs, and by stabilizing plaque. Section 2.5 discussed how lesions grow by repeated episodes of subclinical rupture and repair, and how silent occlusions can happen when plaque ruptures and thrombosis occurs in the case of a well collateralised myocardial circulation such that no significant ischemia results from the occlusion. The use of statins appears to stabilize plaque, which means repeated plaque rupturing can be prevented, thus halting or slowing growth of existing lesions. By lowering serum cholesterol and serum LDLs, fatty streak and plaque formation can be slowed down or prevented. Statins have only a small effect on reducing the size of pre-existing lesions. (Weissberg 2000: 251).

2.9 DIAGNOSIS OF PLAQUE PRESENCE

Angiography can only detect lesions that impinge significantly on the arterial lumen, and does not give any information on the compostion of the lesion. Since composition, rather than size, determines the likelihood of plaque rupture, angiography is a poor predictor of clinical events. In addtion, as mentioned before, a small lesion undetected by angiography, if unstable, can spontaneously rupture and lead to ACS. The following four diagnostic methods are currently being used and researched to detect the presence and composition of atherosclerotic plaque (Klootwijk & Hamm 1999: S10-S14; Weissberg 2000: 249-251).

- **Electron beam computerized tomography (CT)**
- **Magnetic resonance imaging (MRI)**
- **Positron emission tomography (PET)**
- **Biochemical markers of plaque inflammation**

2.10 ECG CHANGES INDICATIVE OF ACS

In order to understand what the ECG changes represent, a review of a few key terms will be presented with ECG changes relative to each term.

2.10.1 UNSTABLE ANGINA

Angina pectoris (chest pain), the classic symptom of coronary artery disease (CAD), occurs when the heart does not receive a sufficient supply of blood and oxygen (**ischemia** is another term for deficient blood supply to a body part). Angina is considered **unstable angina** if symptoms occur unpredictably or if previously stable conditions become more intense, last longer, or radiate to a different location than usual. Unstable angina is not always caused by exertion – it can occur during rest or sleep, and it is not always relieved by rest and NTG. It indicates severe coronary vessel narrowing from atherosclerotic plaque, a small thrombus, or arterial spasms. Myocardial ischemia is indicated on ECG by ST depression of 1 mm or more above the isoelectric line in 2 or more continguous leads, or T-wave inversion. It is usually a reversible, transient event that is corrected when the blood and oxygen supply to the myocardium is restored (Dennison 1996: 107-108; Sieck 2000: 5-6).

2.10.2 MYOCARDIAL INFARCTION (MI)

Myocardial infarction is the term used to describe irreversible cellular loss and myocardial necrosis that result from prolonged ischemia to a specific area of the myocardium. It is the destruction of a sufficient amount of heart tissue to cause clinical, electrical and/or biochemical changes in the body, and is indicated on ECG by ST-segment elevation of 1 mm or more in 2 or more contiguous leads (Dennison 1996: 110; Thelan et al 1994: 283-284).

2.10.2.1 Q-WAVE MI

A **Q-wave MI**, also called a transmural infarction, affects all three layers of the heart wall, the epicardium, myocardium, and endocardium. It is associated with higher frequency of complete coronary occlusion and infrequent coronary collaterals. It is indicated on ECG by ST-segment elevation of 1 mm or more in 2 or more contiguous leads, and by a Q-wave greater than 0.04 seconds in width and 25% the height of the R-wave. If all 3 layers of the myocardium have been affected, a Q wave appears within a few hours to a few days after the occlusion. The Q-wave may decrease over time following a MI, but it will always be present in a 12-lead ECG (Gylys & Gold 2000: 3-13; McAvoy 2000: 34-39; Statland 1996: 43-51; Church 2000: 34-42; DeWood 1988: 36F-38F).

2.10.2.2 NON-Q-WAVE MI

Some people experience a MI without developing Q-waves. A **non-Q-wave MI** does not affect all three layers of the heart. It is associated with a lower frequency of complete coronary occlusion and is more common with diabetics or those who have developed good coronary collateral circulation. It is indicated on ECG by ST-segment depression which could flip up as the blockage develops into a full-thickness injury. Although non-Q-wave MI was originally considered to be less serious than Q-wave MI, subsequent studies have shown that the incidence of death and recurrent MI is as great as that associated with Q-wave infarctions (Gylys & Gold 2000: 3-13; McAvoy 2000: 34-39; Statland 1996: 43-51; Church 2000: 34-42; DeWood 1988: 36F-38F).

2.10.3 LIMITATIONS OF ECG AS A MARKER OF ACS

Patients with acute MI can frequently present with nondiagnostic ECG's, whether as a result of conduction system delays (e.g. left bundle branch block), nonspecific ST- and T-wave changes, or MI's involving the posterior wall. Improvements in capturing ECG information -- such as 15-lead systems, continuous vectocardiograms, or ST-segment

monitoring – can improve sensitivity of detection of ACS, but still cannot give a definitive diagnosis on all patients.

2.11 CARDIAC ENZYMES (MARKERS)

Following is a discussion of three cardiac enzymes (markers) used to detect a myocardial infarction or rule it out with their normal values and advantages and disadvantages of each.

2.11.1 MYOGLOBIN / Normal: 10-13 units/L

Myoglobin is a low molecular weight protein found in all muscle that is rapidly released into the bloodstream when there is muscle damage. An increase in serum myoglobin is seen earlier than any other cardiac marker, and appears in serum 1 to 3 hours after the onset of chest discomfort; levels peak in 6 to 9 hours and return to normal in 20 to 24 hours. The presence of myoglobin, coupled with clinical signs and symptoms, suggest myocardial injury. Another advantage is that laboratories can perform rapid testing, which means test results are obtained quickly.

Because myoglobin exists in other tissues than just the myocardium, any injury to skeletal muscle can cause a rise in myoglobin levels, including skeletal or neuromuscular disorders, trauma, injections with needles, strenuous exercise, surgery, renal failure, multisystem organ failure, alcohol abuse, hypothermia and hyperthermia. In other words, the test is not very sensitive. Increased levels of serum myoglobin alone cannot be used to rule in a MI, but can be used as an initial screening tool (Ryan 2000: 26-30; Statland 1996: 43-51).

2.11.2 CREATINE PHOSPHOKINASE (CPK) / Normal: 10 – 13 units/L

Creatine phosphokinase (CPK) is an enzyme that exists in cardiac and skeletal muscle, as well as in the brain, and can be fractioned into three closely related enzymes, or

isoenzymes. **CPK-MB** (MB means "myocardial band") is the cardiac component. Once thought to be the "gold standard" for detecting myocardial damage, recent research has shown that CPK-MB readings can be influenced by a number of factors such as trauma, multi-organ damage, high doses of salicylates, strenuous exercise, surgery, myocarditis and rhabdomyolysis.

CPK-MB usually starts to rise 3 to 6 hours after the onset of myocardial damage, peaking in 12 – 24 hours, and returning to normal in 48 – 72 hours. The test is often used to screen for myocardial damage but cannot definitively prove its presence. It is also used to monitor the progress of an MI and the effectiveness of treatment.

Up until recently, CPK-MB analysis was time-consuming, labor-intensive and expensive, making it difficult to obtain rapid results. However, newer analysis systems take faster measurements and improve the turnaround time, but it is still not optimal (Ryan 2000: 26-30; Statland 1996: 43-51).

2.11.3 TROPONINS T AND I

Troponins, which are proteins found in cardiac and skeletal muscle, exist in three different forms: troponin T, troponin I, and troponin C. Troponin C is identical in both muscle types and is useless as a cardiac marker. The structures of **troponin T** and **troponin I** in the cardiac muscle are different from the troponins in skeletal muscle, so they are easily differentiated in lab analysis. Numerous studies have shown the troponins to be as acurate and reliable as CK-MB, and actually better, because of their greater cardiac specificity than CK-MB. In addition, troponin normally is not present in serum until cardiac cell necrosis occurs, so even negligible levels are important and allow for improved diagnosis of cardiac injury, especially in a situation where severe skeletal muscle damage has occurred.

Both troponins remain elevated for longer periods of time (5-9 days for troponin I, and up to 14 days for troponin T). Thus, sensitivity remains high a long time after CK-MB

levels have returned to normal. This long-term delay is advantageous when dealing with patients who delay seeking medical care for a MI because of misleading symptoms.

Another advantage of the troponins is that their serum concentration in acute MI is independent of muscle mass.

2.11.3.1 TROPONIN T / normal: less than 0.1 ng/ml

Cardiac **troponin T** is released 3 to 4 hours after myocardial damage occurs, and you may also get elevated readings with unstable angina and uremia. This limitation makes the test helping in ruling out a MI, but less helpful in diagnosing one. This test is also costly and takes at least 45 minutes to get results.

2.11.3.2 TROPONIN I / normal: less than 0.6 ng/ml

Cardiac **troponin I** is released 4 to 6 hours after myocardial damage, which is helpful again in ruling out an MI. Because levels do not increase in the presence of unstable angina or renal failure, this test is also very helpful in confirming a MI. The test is expensive, but can be done in only 15 – 20 minutes.

Some manufacturers suggest the normal value is anything less than 1.6. The general consensus is that if the troponin I value is between 0.6 – 2.0, the patient probably had a MI, but it should be confirmed with CK-MB and ECG. Without an increase in CK-MB and significant ECG changes, it might possible be acute ischemia. If the value is greater than 2.0, it most probably is a MI (Adams 1999: 127-134; Ryan 2000: 4-8; Statland 1996: 43-51; Ross, Bever, Uddin & Hockman 2000: 29-31).

2.12 ADDITIONAL DRUGS USED TO TREAT ACS AND RELATED SYMTOMS

In Sections 2.3, 2.4 and 2.6 some of the drugs used to treat ACS were discussed. In this section, six other classes of drugs will be covered that are used to treat ACS.

The initial approach to patients with ACS is urgent admission to emergency care with hemodynamic and ECG monitoring, usually done in an ambulance or emergency room of a hospital. After monitoring and venous access is started, rapid general and causal treatment is started.

General treatment for ACS consists of pain relief and limitation of myocardial ischemia. More causal treatment involves antithrombotic therapy. Pain relief can be achieved by NTG and/or morphine sulfate. Anti-ischemic therapy is aimed at decreasing the ischemia in order to prevent myocardial necrosis. Antithrombotic therapy is given to inhibit clot formation and propagation. In cases of suspected total coronary artery occlusion, clot lysis or clot destruction is the main goal.

After these treatments, most patients become free of symptoms. When signs and symptoms of myocardial ischemia recur, patients usually undergo cardiac catheterization and subsequent revascularization, when feasible (Verheugt 1999: 20-23).

2.12.1 MORPHINE SULFATE

Pain relief can be achieved by the use of nitroglycerine, but is mostly guaranteed with **morphine sulfate (MS),** a narcotic agonist analgesic. For pain control, 2 – 4 mg. morphine intravenously may be given every 5 to 10 minutes, or more if needed if ordered by the physician. Besides controlling pain, morphine relieves anxiety and promotes vasodilation. The nurse should use it with caution, as respiratory depression may occur in the or the very ill. Patients already receiving barbiturate general anesthetics are more

prone towards respiratory depression, hypotension, profound sedation or coma if given morphine (McAvoy 2000: 38; Karch 2000: 815-816).

2.12.2 NITROGLYCERINE

Nitroglycerine (NTG), a nitrate, is the most common drug in the acute treatment of myocardial ischemia. Nitroglycerine relaxes vascular smooth muscle with a resultant decrease in venous return and a decrease in arterial blood pressure. It thus reduces left ventricular workload and decreases myocardial oxygen consumption. Sublingual NTG or NTG mouth spray are used in acute treatment of ischemia in ambulatory patients and can also be used as a prophylaxis. In ACS, intravenous NTG is given both as acute treatment and as a maintenance infusion. NTG dosage is usually titrated either on symptoms or on hemodynamic parameters such as heartrate and blood pressure. NTG may also be administered orally (sustained release), topically, transdermally and transmucosally, between lip and gum above incisors (Karch 2000: 859-860; Verheugt 1999: 11-20).

One major drawback with NTG is the rapid development of tolerance. Other side effects include hypotension and headache. The nurse should carefully monitor the patient hemodynamically when administering NTG.

2.12.3 BETA-BLOCKERS

Beta-adrenergic antagonists, or **beta-blockers**, block the beta-adrenergic receptors in the heart, decreasing the influence of the sympathetic nervous system and thereby reducing the excitability of the heart, cardiac workload, oxygen consumption and blood pressure. Beta-blockers also act as antidysrhythmics. By reducing heartrate and myocardial contraction, beta-blockers allow more time for perfusion of the myocardium. They are indicated to treat stable or unstable angina, hypertension and MI.

The benefits of early beta-blocker use after a MI are the antidysrhythmic effect; ability to decrease mortality in the early stage of MI and the ability to decrease the incidence of MI extension and reinfarction.

Major side effects of beta-blockers in acute MI are rare. Beta-blockers should not be used if any of the following conditions are present: hypotension, bradycardia, AV block or congestive heart failure. These agents should be used with caution in patients with bronchospasmic lung disease (e.g. asthma). For such, cardioselective beta-blockers (e.g. metoprolol or esmolol) may be given; noncardioselective beta-blockers (e.g. propanolol) should not be used (Dennison 1996: 115; Moser, Frazier, Worster & Clark 1999: 11-15; Verheugt 1999: 11-20).

2.12.4 CALCIUM CHANNEL BLOCKERS

Calcium channel-blockers are antianginal and antihypertensive. They inhibit the movement of calcium ions across the membranes of cardiac and arterial muscle cells, which results in the slowing of the velocity of conduction of the cardiac impulse, depression of myocardial contractility, and dilation of coronary arterioles. These effects lead to decreased cardiac work, decreased cardiac energy consumption, and increased delivery of oxygen to the myocardial cells. These drugs are appropriate for patients experiencing hypertension and cardiac ischemia.

Side effects include hypotension, bradycardia, and fatigue. The nurse should monitor the patient hemodynamically while the drug is being titrated to therapeutic dose (McAvoy 2000: 36; Verheugt 1999: S1121; Karch 2000: 31-32).

2.12.5 ACE INHIBITORS

Section 2.4 briefly discussed some of the effects of **angiotension-converting enzyme inhibitor agents (ACE inhibitors)** – vasodilation with resulting decreased blood

pressure, decreased platelet aggregation and adhesion, decreased LDL oxidation, and decreased vascular remodeling. ACE inhibitors are recommended for a minimum of 6 weeks (preferably longer) after a MI, as they decrease ventricular remodeling that occurs after a MI, preserving ventricular function. Recent research shows that these drugs also decrease the risk of recurrent MI (Topol 1998: 571-572).

Studies have shown that up to 14% of patients who experienced an acute MI later develop left ventricular dysfunction as a result of loss of myocardial cells and subsequent ventricular remodeling. Over time, ventricular remodeling results in a loss of use of the infarcted tissue and a thinning of the infarcted zone. The remaining noninfarcted tissue then becomes more dilated in an attempt to maintain adequate flow, and this leads to chronic heart failure and death. Patients with both symptomatic and asymptomatic ventricular dysfunction are at risk for developing symptomatic heart failure, as well as other cardiac dysrhythmias, fatigue, decreased exercise tolerance, and overall decreased quality of life. For these reasons, the prevention of left ventricular dysfunction is a priority in the treatment of acute MI survivors. Because early intervention is the key to preserving left ventricular wall function, ACE inhibitors should be started within 72 hours after a MI. After 4 to 6 weeks of therapy with ACE inhibitors, patients who do not show evidence of left ventricular dysfunction can stop treatment (Moser et al 1999: 13; McAvoy 2000:37; Fuster et al 1999: 58-62).

Side effects of ACE inhibitor agents include hypotension (especially in salt/volume depleted patients), tachycardia, congestive heart failure, fatal pancytopenia and gastric irritation. The nurse should closely monitor the patient's fluid status, blood pressure and heartrate and renal function. The nurse should give these drugs should be given shortly before or after meals to prevent gastric irritation.

2.12.6 THROMBOLYTICS

Reperfusion therapy with **fibrinolytic agents, or thrombolytics,** is standard care for patients presenting to the hospital within 6 hours of MI symptoms and who have

significant ST-segment elevation on ECG (some hospitals give thrombolytics if the patient presents within 12 hours of onset of MI symptoms).

These drugs work by converting inactive plasminogen to active plasmin, an enzyme that is responsible for breaking down fibrin clots. The primary goal of these drugs is to decrease mortality by rapidly restoring blood flow to the occluded infarct-related coronary artery(s).

Streptokinase and urokinase are the first generation of fibrinolytic agents currently in use. Newer agents are more clot selective than streptokinase. They are alteplase (tPA, Activase); retaplase (rPA, Retavase) and anistreplase (APSAC, Eminase).

Data from several large-scale studies have shown that the choice of thrombolytic agents is less important to survival than is the delay between onset of symptoms and initiation of treatment. Patients in the RAPID II study who received treatment within 6 hours after the onset of symptoms had better overall patency than did the patients receiving treatment between 6 – 12 hours (Kloner & Birnbaum 1997: 42-43).

Absolute contraindications to thrombolytic therapy are: active internal bleeding; history of CVA; recent (within 2 months) intracranial or intraspinal surgery or trauma; known bleeding disorders; severe uncontrolled hyptertension; prolonged or traumatic CPR; pregnancy; streptokinase contraindicated if the patient has received streptokinase within the last 6 months (rPA can still be given).

Relative contraindications are major surgery or trauma within 10 days; cerebrovascular disease; recent GI or GU bleeding; high likelihood of thrombus on the left side of the heart (e.g. mitral stenosis with atrial fibrillation); subacute bacterial endocarditis; significant liver dysfunction; patients receiving oral anticoagulants; diabetic hemorrhagic retinopathy; advanced age (over 70 years); any condition in which bleeding constitutes a significant hazard or would be particularly difficult to manage because of its location.The nurse should monitor closely for any adverse side effects of thrombolytic therapy such as

hemorrage, reocclusion, allergic reactions or reperfusion dysrhythmias, in addition to nursing care given when administering antithrombotic agents. Reperfusion dysrhythmias are usually transient and treatment is usually required only for sustained ventricular tachycardia, ventricular fibrillation and symptomatic bradycardia or block. Check for allergic reactions (to streptokinase) such as urticaria, fever, bronchospasms, dyspnea and dysrhythmias (Dennison 1996: 114-115; Roberts 1999: 7-9; Verheugt 1999: S1121).

2.13 PERCUTANEOUS CORONARY INTERVENTION

If ST-segment elevation persists despite medical treatments, or if chest discomfort continues despite a definitive diagnosis, the physician may choose invasive treatments such as **percutaneous coronary intervention (PCI)**, a technique where the blocked vessels are mechanically opened up by angioplasty, atherectomy, or stent placement. Coronary artery bypass graft **(CABG)** surgery may be needed if the coronary vessels are totally occluded or untreatable by other methods. It is not within the scope of this research paper to discuss CABG surgery; however, PCI will be briefly discussed in order to understand the range of treatment options for ACS.

2.13.1 PERCUTANEOUS TRANSLUMINAL CORONARY ANGIOPLASTY

Percutaneous transluminal coronary angioplasty **(PTCA)** involves the insertion of a catheter through the radial or femoral artery, advancing it to the coronary arteries, and inflating the balloon-tipped end of the catheter intermittently in the area of coronary artery stenosis caused by plaque. This dilates the stenotic area and improves blood flow through it. PTCA is an alternative to both traditional medical management of coronary artery disease and CABG surgery, and is a valuable adjunt to thrombolytic therapy in some cases (Thelan et al 1994: 330; Dennison 1996: 109).

2.13.2 CORONARY ATHERECTOMY

Coronary atherectomy is the removal of plaque from a coronary artery by cutting, shaving or grinding off the plaque.

Research and practical experience have shown that PTCA with a balloon catheter is not the best therapy in patients with complex, tortuous coronary lesions. Complication rates for balloon angioplasty are higher for longer (10 20 mm long), highly calcified type lesions. Complications include inability to cross a lesion with a typical PTCA wire and balloon, aprupt closure of the vessel, major arterial dissection and restenosis. Consequently, other approaches and treatment have been sought.

In traditional PTCA, inflation of the balloon catheter causes distention of the vessel wall. Rotational atherectomy, the most commonly used type of atherectomy, causes minimal swelling of the arterial wall and allows treatment of lesions that are not easily approached with traditional angioplasty alone (Thorbs, Barbiere, Wayland & Morgan 2000: 77-80).

2.13.3 CORONARY ARTERY STENTS

Coronary artery stent placement involves the use of a stainless steel wire coil that acts as a scaffolding device to support a coronary artery and maintain patency after PTCA. The stent is introduced over a guidewire in a region previously dilated by PTCA to prevent acute closure and restenosis, as well as to obtain a larger vascular lumen diameter.

2.13.4 COMPLICATIONS OF PTCA, ATHERECTOMY AND STENT PLACEMENT

The following complications can occur as a result of any one of these PCIs: coronary artery dissection, caused by catheter trauma; cardiac tamponade, caused by cardiac perforation; dysrhythmias; acute reocclusion or closure caused by trauma to the intima initiating the clotting cascade or coronary artery spasm; chronic restenosis caused by intimal hyperplasia; hypotension or bradycardia (vagal reaction) caused by increased parasympathetic nervous system stimulation during sheath removal; and subacute stent thrombosis.

Although stents have decreased restenosis rates and the need for target vessel revascularization, their use activates the GP-IIb/IIIa receptors on platelet surfaces and predisposes to thrombus formation. This is reflected by the clinical syndrome known as **subacute thrombosis**, an event differing from abrupt closure or restenosis. Abrupt closure usually occurs within 24 hours following PTCA, whereas subacute stent thrombosis occurs in 1% to 3% of patients and develops 2 to 14 days after stent placement (Futterman & Lemberg 2000: 70-76; Thorbs et al 2000: 77-84).

2.13.5 NURSING MANAGEMENT FOR PCI PATIENTS

The nurse should check the catheter puncture site frequently for bleeding or hematoma formation. Neurovascular checks should be performed frequently to assess for peripheral ischemia related to femoral or radial artery thrombosis. The nurse should monitor for signs and symptoms of retroperitoneal hemorrhage by checking for back or flank pain, postural tachycardia and/or hypotension. The affected limb should be kept flat and immobile and the head of bed should not be elevated anymore than 30 degrees (if femoral site used) for the first 8 hours after PTCA. The patient should be on continuous cardiac monitoring, and the nurse should check for ECG changes (in particular ST- or T-wave changes) and onset of chest pain, indicated myocardial ischemia (Dennison 1996: 109-110; Futterman & Lemberg 2000: 70-76; Thorbs et al 2000: 77-841).

2.13.6 FACILIATED PCI

Until recently, fibrinolytic drugs have been the treatment of choice for achieving rapid revascularization, with coronary angioplasy reserved as an option if the former did not achieve vessel patency. Traditionally routine PCI of the stenotic infarct-related artery following fibrinolytic therapy has not been recommended, because some evidence suggested that the procedure was not useful or effective and was perhaps even harmful. However, recent studies show that PCI is facilitated by a combination of therapies that *enhance* reperfusion. Preclinical and clinical investigations of fibrinolytic therapy plus GP-IIb/IIIa inhibitors have shown that the dose of fibrinolytic therapy can be reduced by

approximately 25% to 50% when administered with GP-IIb/IIIa inhibitors. Fibrinolysis occurs more rapidly and completely and is more stable with combination therapy, as reflected by improved artery patency, which results in lower mortality and reinfarction rate and higher PCI success. This concept of administering this type of pharmacotherapy prior to angioplasty and stent placement is currently referred to as **facilitated percutaneous coronary intervention,** or **FPCI**. The results are rapid relief of angina, restoration of coronary and myocardial blood flow, improvement in myocardial preservation without significant risks of bleeding, and reduced incidence of death and reinfarction.

The rationale for lytic therapy in conjunction with PCI and GP-IIb/IIIa inhibitors is that the earlier an infarct-related artery can be opened, the more myocardium can be preserved. Thrombolytic therapy initiated immediately in the field or in the emergency room can facilitate reperfusion before cardiac catheterization. A patent or partially patent artery would be technically easier to perform angioplasty and stenting on (Futterman & Lemberg 2000: 72-74).

2.14 NEW AND UPCOMING TREATMENTS FOR ACS

2.14.1 CONTINUOUS ST SEGMENT MONITORING

The static 12-lead ECG is likely to miss important trends in ST-segment recover or reevaluation during the early dynamic period of ischemia, injury, or infaction because it documents electrical complexes of a single moment. **Continuous ST-segment monitoring** is a non-invasive, continuous method for monitoring coronary patency. Most current models of cardiac monitoring offer ST-segment monitoring capability, and stand-alone ST-segment monitors are also available, making continuous ST-segment monitoring accessible to everyone in all hospital locations.

In addition to monitoring for reperfusion and reocclusion in ACS, ST-segment monitoring can help guide pharmacologic and mechanical intervention. Another

diagnostic advantage is that one can obtain an ECG immediately and concurrently with changes in pain. Most ST-segment monitoring systems can be set to capture a 12-lead ECG at 1- to 30-minute intervals and any time an alarm is triggered. These ECGs are plotted to form a trend, revealing ST-segment changes over time to help the nurse better asses her patient (Kucia & Stewart 2000: 4-13).

2.14.2 PLAQUE STABILIZATION

Research has shown that it is not necessarily the size of a plaque lesion, but rather the plaque composition that determines plaque stability. Studies have shown improved coronary and brachial endothelial function with several risk factor modifications and drugs that have also been shown to reduce cardiovascular events and ischemia in patients with coronary heart disease. These include cholesterol lowering, blood pressure reduction, smoking cessation, and administration of estrogen, ACE inhibitors, statins and antioxidant vitamins. These measures help stabilize existing plaque and prevent the occurrence of future atherosclerotic lesions (Topol 1998: 560-561).

2.14.3 BIOCHEMICAL MARKERS OF INFLAMMATORY CELLS

Pathological studies have revealed several common characteristics of plaques that have ruptured and caused clinical events. These features include a thin, fibrous cap; a large lipid core; numerous macrophages and other inflammatory cells; and a relative small number of vascular smooth muscle cells. Inflammatory cells can weaken the fibrous cap in several ways, leaving it vulnerable to rupture. Thus inflammatory cells can destroy the make-up of the fibrous cap. Researchers are working to establish reliable biochemical markers that will indicate the presence of these inflammatory cells at plaque sites and it is likely that biochemical measures of inflammation, along with measurement of conventional risk factors, will be used to guide the selection of patients at highest risk (Weissberg 2000: 247-252).

2.14.4 DETECTION OF PLAQUE PRESENCE AND COMPOSITION

Magnetic resonance imaging (MRI), as mentioned earlier, gives anatomical detail, but no information on the inflammatory activity within the plaques. **Positron emission tomography (PET)** shows little anatomical information but gives the opportunity to measure and monitor plaque inflammatory cell content and activity.

2.14.5 ORAL GP-IIb/IIIa INHIBITORS

Although very expensive and still under research, it is hopeful that oral GP-IIb/IIIa inhibitors can be developed for use for both in- and out-patient use to help prevent or treat ACS. These drugs block the final pathway of platelet aggregation and are currently the most effective antiplatelet medications on the market.

2.14.6 COMBINATION DRUG THERAPY

LMWHs and GP-IIb/IIIa drugs have been used separately either alone or in combination with other medications up until now to treat ACS. Randomized trials are now underway to test the effectiveness of using the two drugs concurrently to treat ACS.

2.15 CONCLUSION

Identifying and managing acute coronary syndromes (ACS) – unstable angina, non-Q-wave and Q-wave MIs – is a challenge for nurses. The goal of therapy is to maintain perfusion through the coronary arteries while limiting any areas of myocardial damage. The whole spectrum of ACS has been investigated thoroughly in recent decades and have resulted in in-depth knowledge of the pathophysiology and in extensive experience with different treatment methods. In addition to being familiar with current approaches to treating ACS, it is vitally important that the critical care nurse have a thorough knowledge of the pathophysiology of ACS in order to understand the rationale behind approaches to treating ACS.

CHAPTER 3

RESEARCH DESIGN AND RESEARCH METHODOLOGY

3.1 INTRODUCTION

The research design and method are discussed in detail in this chapter. The aim of this study is reiterated for clarity purposes, which was to explore and describe the respondents' perceptions as to how they rate their knowledge of ACS, the pathophysiology of ACS and current approaches to and rationales for treatment, and based on the results of the findings, to formulate an in-service training session about ACS for critical care nurses. ***Table 3.1*** on page 52 is a summary of the content.

Table 3.1 Research Design and Method

AIM	DATA GATHERING	SAMPLING & POPULATION	VALIDITY & RELIABILITY	DATA ANALYSIS
Explore and describe the respondents' perceptions as to how they rate their knowledge regarding aspects of ACS: its pathophysiology, current available treatments, rationales for such treatments and nursing care, and to formulate an in-service training session on ACS.	Implementation of a questionnaire	Convenience sample of voluntary critical care nurses of all qualifications, regardless of staff appointment, in a state hospital in East Texas.	Literature review, professional experts' critique, Cronbach's alpha and factor analysis.	Descriptive and inferential statistics.

3.2 RESEARCH DESIGN AND METHOD

3.2.1 RESEARCH DESIGN

The design of this research study was a quantitative, descriptive and contextual study in which a sample survey was performed:

- The study is quantitative in order to investigate the perceptions of critical or acute care nurses as to how they rate their knowledge regarding ACS, and also to access the magnitude and reliability of relationships between independent variables and nurses' knowledge of ACS (Polit & Hunger 1995: 651).
- It is descriptive because it aims at describing and documenting phenomena, such as the respondents' age, number of years nursing experience and education for this specific context (Polit & Hunger 1995: 52, 178).
- It is contextual because this study focuses on the knowledge of critical care nurses regarding ACS and specifically the critical care nurses at a state hospital in East Texas.

As instrument a questionnaire was used to describe the critical or acute care nurses' rating of their knowledge of ACS. Scales were chosen for answering questions instead of using open-ended questions. The reason for this is because the researcher assumed that with scales, the participants could indicate what they *perceived* they knew regarding ACS, and this would be less threatening to them than open- or close-ended questions, in which their knowledge about ACS would actually be tested.

In a survey information from populations regarding the prevalence, distribution, and interrelations of variables can be obtained within those populations. One of the greatest advantages of survey research is its flexibility and broadness of scope. It can be applied to many populations, it can focus on a wide range of topics, and its information can be

used for many purposes. Questionnaires in particular are easy to administer and economical (Polit & Hunger 1995: 187-188).Questionnaires are self-administered, i.e. the same respondent reads the questions on a form and gives an answer in writing.

Care was taken in the development of the questionnaire to word the questions clearly, simply and unambiguously. The specific context in which this research study was held was in a state hospital in East Texas affiliated with the University of Texas.

3.2.2 RESEARCH METHOD

The purpose of the literature study was three-fold: first, to establish a conceptual framework for the research study; secondly, to identify the criteria to be considered as critical points for this study, and thirdly, to formulate an educational in-service training session on ACS for critical care nurses in the hospital where this study is performed.

3.2.2.1 DATA GATHERING

The data was gathered by means of a questionnaire. The development of the questionnaire was as follows:

- development phase
- types of questions
- refinement of questionnaire
- confirmation phase

Development phase

The literature study was used to determine which criteria were to be considered as critical points in the study. The questionnaire using scales was chosen by the researcher because it is not expensive, it requires less time to administer compared to other types of data collection methods, and it provides the possibility of complete anonymity (Burns & Grove 1993: 374).

A cover letter (see ***Annexure I***) accompanied the questionnaire, explaining the purpose of the study, the name of the researcher, and assuring anonymity.

Types of questions

All the questions regarding the respondents' knowledge in the second section are of such a nature that the respondent had to evaluate him/herself on a rating scale. The reason for using these kinds of questions is that graded alternatives give the researcher more information regarding intensity as well as direction of opinion. It also gives the respondents the opportunity to express a range of views (Burns & Grove 1993: 377-379; Polit & Hungler 1995: 256, 277). The respondents do not feel threatened by the questions – in other words, they do not feel that somebody is checking on what they know or do not know about ACS. They are actually rating or evaluating their own knowledge, which contributes to a non-threatening situation. Scales are popular amongst nurse researchers, are easy to administer, and do not require a group of expert judges to put together the questions (Abdellah & Levine 1979: 260-262; Polit & Hungler 1984: 281-283). The questions in section one regarding demographic general information are a combination of fill-in-the-blank and multiple-choice questions.

Compilation of questionnaires

The questionnaire (see ***Annexure II***) consists of two sections. The first section covers general information about the respondents. The second section covers information about ACS. ***Table 3.2*** gives a layout of the questionnaire.

Table 3.2 Layout of Questionnaire

SECTION	TOPIC	NUMBER & DESCRIPTION OF QUESTION
1 - General information	Demographic data	Seven questions: 1-7 Age, gender, marital status, level of education, staff appointment, number of years nursing experience in general and number of years critical care nursing experience. This data was used to describe the sample population. Additionally, this study looks at relationships between these independent variables (demographic data) and the opinion of the respondents on their level of knowledge of ACS as portrayed by the answers to the questionnaire.
2 – Knowledge of ACS	Definition of ACS	One question: 1 Describes the opinion of the respondents on their level of understanding of the definition of ACS.
	Pathophysiol-ogy of ACS	One question: 2 Reflects the opinion of the respondents on their ability to describe the pathophysiology of ACS to patients
	Patient education regarding in-hospital management of ACS	One question: 3 Reflects the opinion of the respondents on their ability to explain to patients about non-invasive and invasive treatments performed in the hospital aimed at managing ACS.
	Physiology of clot formation and lysis.	One question: 4 Describes how the respondents feel they would benefit from an understanding of the physiology of thrombus formation and lysis.

		NUMBER & DESCRIPTION OF QUESTION
	How hypertension can lead to ACS	Two questions: 5 and 6 Reflects the opinion of the respondents on the level of their understanding of how hypertension can lead to endothelial damage, plaque formation, plaque rupture and thrombus formation, and their ability to explain this to the patients.
	Q-wave and non-Q-wave MI	One question: 7 Reflects the opinion of the respondents regarding their knowledge about the differences between a Q-wave and non-Q-wave MI.
	Unstable angina or a non-Q-wave MI progressing to an acute MI.	One question: 8 Describes the opinion of the respondents on their knowledge of how unstable angina or a non-Q-wave MI can progress to an acute MI.
	Cardiac markers.	Three questions: 9, 10, 11 Reflects the opinion of the respondents on their knowledge about the three main cardiac markers, how to explain their interpretation to patients, and the respondents' knowledge about the advantages and disadvantages of each cardiac marker in diagnosing a MI.
	ECG readings.	One question: 12 Describes the opinion of the respondents on their understanding of what ECG changes indicate myocardial ischemia, injury and infarction.
	Sheath removal	One question: 13 Reflects the respondents' opinion on their level of experience in sheath removal after PCI.

		NUMBER & DESCRIPTION OF QUESTION
	Low molecular weight heparins (LMWH)	One question: 14 Describes the respondents' opinion on their level of knowledge regarding drug actions of LMWH and advantages and disadvantages of using LMWH instead of unfractioned heparin.
	Glycoprotein IIb/IIIa inhibitor agents	Three questions: 15, 16, 17 Reflects the opinion of the respondents on their level of knowledge about the use of GB IIb/IIIa drugs for treating unstable angina and non-Q-wave MI, their level of experience in administering these agents, and their level of knowledge about how these agents are used after PTCA/stent procedures.
	Thrombolytic agents	Three questions: 18, 19, 20 Reflects the opinion of the respondents on their level of knowledge about actions, uses and potential complications of administering thrombolytic agents, as well as describes their level of knowledge of the contraindications to giving thrombolytics and/or IIb/IIIa inhibitor agents.
	Beta-blockers	One question: 21 Describes the opinion of the respondents on their level of knowledge on the rationale of administration of beta-blockers to patients who have recently experienced a MI.
	Regularity of patient education	One question: 22 Describes the respondents' regularity in providing patients education regarding ACS pathophysiology and medications used to treat ACS.

		NUMBER & DESCRIPTION OF QUESTION
	Medications for ACS patients	Two questions: 23 and 24. Reflects the opinion of the respondents on their level of knowledge about several medications prescribed for ACS patients (calcium channel blockers, long-acting nitrates, beta-blockers, angiotension-converting enzyme (ACE) inhibitors,aspirin, clopidogrel, ticlopidine and warfarin) at time of hospital discharge, as well as the respondents' ability to explain the uses and side effects of these drugs to their patients.

Refinement of questionnaire

The critical care nurse educator at the hospital where this study was performed was approached and agreed to be of assistance to review the questionnaire. She has extensive expertise in intensive care nursing as well as critical care education. Changes to the questionnaire were made according to her suggestions.

Confirmation phase

The questionnaire was revised and presented to the Nurse Executive at the hospital for approval of distribution (see ***Annexure III: Permission Letter***)..

3.2.2.2 POPULATION AND SAMPLING

Population

The target population was the critical or acute care nurses at a state hospital in East Texas. This included nurses working in the Intensive Care Unit (ICU), Operating Room (OR), Emergency Room (ER), Cardiac Catheterization Laboratory (cath lab), and

Telemetry (5 East). The nurses working in these units during the month of May 2000 were asked to take part in this study. They participated regardless of their critical care training and experience status. Full-time, part-time and PRN (session) staff were included in this study. This hospital was chosen because the researcher works there, and was interested to know how much those working with her know about ACS. She also wants to help them increase their knowledge about ACS and help them improve their nursing care for such patients. Written permission was obtained to from nursing management of this hospital to distribute the questionnaire (see ***Annexure III***).

Sampling

Convenience sampling was used in this research. Convenience sampling entails the use of the most conveniently available people for use as subjects in a study. An advantage is that it is inexpensive. A weakness in this method is that available subjects may be atypical of the population in regards to the critical variables being measured. This can limit the extent of generalization (Polit & Hunger 1995: 232). The questionnaires were put in the critical care nurses' mailboxes of the units mentioned above. Instructions were given on the cover sheet for the nurse to fill out the questionnaire at work and to return it to a large manila envelope conveniently posted in each unit for collection of the questionnaires. Seventy questionnaires were passed out. A total of 25 were returned, but only 19 were completely filled out (n=19).

3.2.2.3 VALIDITY AND RELIABILITY

Validity refers to "the degree to which an instrument measures what it is supposed to be measuring". **Internal validity** is "the degree to which it can be inferred that the experimental treatment (independent variables), rather than uncontrolled, extraneous factors, is responsible for observed effects". **External validity** refers to how well the research findings can be generalized to other settings or samples (Polit & Hunger 1995: 353, 644). Reliability refers to the degree of consistency with which the instrument (in

this case, the questionnaire) measures the thing it is supposed to be measuring (knowledge of ACS).

The questionnaire is a product of the literature study, conceptual framework and the contribution of professionals. Its validity was confirmed by the evaluation of the professionals mentioned earlier. In addition, the researcher did not bias her scale. As indicated earlier by the researcher, critical thinking is vitally important to the nursing process and will result in a comprehensive plan of care for patients with the most potential for success. The researcher assumes that in order to provide optimum care for ACS patients, the nurse must be able to make independent judgments and decisions *which arise from a sound knowledge base* and the ability to integrate that knowledge of information within the context where it is applied (in this case, critical care hospital units). Additionally, clinical practice expertise -- developed through clinical experience -- is a very important element influencing the nursing process and is what consistently characterizes the nurse expert (Thelan et al 1994:4).

Internal validity was not easy to maintain. The researcher could only assume the participants answered the questions honestly. However, the non-threatening nature of the questions and assured anonymity and confidentiality could have enhanced honesty.

External validity "is concerned with the extent to which study findings can be generalized beyond the sample used in the study" (Burns & Grove 1993). It was not achieved in this study, because the researcher was not able to control the rate of return of the questionnaires, which may be a factor in the low rate of return. Out of 70 questionnaires distributed, only 25 were returned, and only 19 of those returned were completely filled out. It can be further mentioned that looking at the demographic data, the mean age was 47 years and the mean nursing experience was 18 years, of which both are probably higher than at many other hospitals. The hospital where this research study was performed is a state hospital that has excellent benefits – vacations, health insurance, and pension plans – which is a major reason why employees remain working at this institution over long periods of time.

Reliability of the questionnaire was confirmed by Cronbach's alpha and factor analysis. This was determined before the questionnaire was distributed. Cronbach's alpha is a widely used method to determine internal consistency of the instrument used in a research study. In other words, it measures whether or not items in the instrument correlate with each other. The normal range of values is between 0.0 and +1.0, and higher values reflect a higher degree of internal consistency. In the case of this study, Cronbach's alpha was 0.9416, reflecting a high degree of internal consistency, i.e. a high degree of correlation between the subject items in the questionnaire. This means there is consistency in what the items measure – they measure what they are supposed to measure (Abdellah & Levine 1979: 281-283; Polit & Hunger 1984: 347-351).

3.2.2.4 METHOD OF DATA COLLECTION

Data collection

Before approaching the Nurse Executive for permission to run this study, the researcher approached the ICU manager and the critical care nurse educator. The researcher explained the purposes of this study and presented the questionnaire along with an outline of a proposed in-service training session on ACS to be given. Anonymity and confidentiality of each participant were assured, as any information collected would not be linked in any way to their names.

Each nurse has his/her own mailbox at the hospital, and a questionnaire was placed in the mailboxes of all nurses working in the ICU, OR, ER, cath lab and 5 East. A large manila envelope marked "ACS Questionnaires" for collection of the questionnaires was placed in each of the five units and instructions given to the nurses to fill out the questionnaire at work and return it to the designated manila envelope posted in their respective unit. This set of instructions could also have contributed to reliability. The questionnaires were distributed during the first week of May 2000 and the collection envelopes were picked

up at the end of May 2000. Out of 70 questionnaires distributed a total of 25 were returned. Only 19 of these 25 questionnaires were filled out completely.

Control of external variables

Control of external variables in this study was not strict. The questionnaires were placed in respondents' mailboxes and they were given almost a month's time in which to return them to the collection envelopes. However, in this questionnaire knowledge was not tested. The respondents had to rate their knowledge on a scale, which eliminated the need amongst the respondents to share knowledge as would have been the case in order to perform better in a "test" type questionnaire. For the same reason, the respondents would not have taken the questionnaires home with them and/or consulted outside sources of information for the answers, because they only had to give an opinion on how they rate their own knowledge. The researcher was available during work hours for answering any queries.

Additional to the above, the questionnaire was developed by the researcher after a thorough literature study was done on the subject of ACS. A literature review helps a researcher develop a broad conceptual context into which a research problem will fit, and also serves to provide the researcher with a perspective on the problem necessary for interpreting the results of his/her study (Polit & Hungler 1995: 70).

Indicators of adequate competency

A standardized answer sheet was used for retrospective evaluation of the questionnaire. In 22 of the 24 questions, circling "1) (strongly agree) is an indication that the respondent believes he/she has a thorough understanding of what is asked. Two out of the 24 questions (4 and 15) are the reverse: by answering with a "7" (strongly disagree) the respondent indicates he believes he/she has a thorough understanding of what is asked.

3.2.2.5 DATA ANALYSIS

Data analysis was done by means of descriptive and inferential statistics. Descriptive statistics were used to document and describe responses on the questionnaire, including what percentage of critical care nurses displayed competency in each area measured. Inferential statistics were used to infer relationships between the independent variables (age of respondents, number of years nursing experience, number of years critical care nursing experience, educational level of respondents) and the respondents' perception as to how they rate their knowledge regarding ACS.

3.3 SUMMARY

The design of this research study was a quantitative, descriptive and contextual study. A questionnaire was used as an instrument to ascertain the opinion or perception of the respondents on their knowledge about ACS. Convenience sampling was used. The questionnaire was a product of a literature study, conceptual framework, and contribution of professionals.

Reliability was measured by Cronbach's alpha and factor analysis before the questionnaire was distributed. Control of external variables was explained. The rate of return of the questionnaires was low, given the number originally distributed in the nurses' mailboxes.

CHAPTER 4

RESULTS OF THE STUDY

4.1 INTRODUCTION

The results of this research study are presented and discussed in this chapter. Where possible, data has been presented in tables to clarify and simplify the results. The competency indicator will be reviewed, followed by a description of the participants of the research study. The data analysis is presented in different sections: analysis of the complete scale, percentages of participants having adequate knowledge in each subject area, and correlational analysis between the independent variables (age, years nursing experience, years critical care nursing experience, and level of education and the test scores).

4.2 COMPETENCY SCORES

Critical or acute care nurses are highly specialized nurses, and the researcher assumes that they have a solid knowledge of critical care nursing in order to provide the skills and competent intensive care for patients whose conditions may change rapidly and who require both close monitoring and critical care intervention. Critical care nurses ought to, ideally, know the pathophysiology of coronary artery disease and understand the rapidly evolving treatments for ACS.

Competency, or adequate knowledge of a respondent in his/her opinion regarding a particular subject area, is indicated by circling a "1" (strongly agree) or a "2" (agree) in all questions except questions 4 and 15, in which case it is the reverse – circling a "6" (disagree) or "7" (strongly disagree) indicates adequate knowledge/competency regarding the area questioned.

4.3 PARTICPANTS IN THE RESEARCH STUDY

4.3.1 DEMOGRAPHIC INFORMATION

Table 4.1 outlines the demographic profile of the respondents. Of the 19 respondents, 17 were female, and 14 were married. The largest number of nurses had an ADN (three-year registered nurse Associate Degree of Nursing), and seven had a BSN (four-year registered nurse Bachelor Degree of Science in Nursing). Two nurses had a Master's degree. The largest number of nurses (eight) work in ICU, with seven working in ER, three in 5-East (Telemetry), and one in the cardiac catheterization laboratory (cath lab). No one from OR returned a questionnaire.

The mean age was 47 years old, and the mean number of years nursing experience was 18 years. The mean number of years experience in critical care nursing was 8 years.

Table 4.1 Demographic Profile of Participants

Sample size (N): 19

Gender	**Marital status**
Male 2 Female 17	Married 14 Single 0 Divorced 5 Widowed 0
Education level	**Unit worked**
ADN 11 BSN 7 MA, MN, MSN 1 PhD 0	ICU 8 ER 7 OR 0 Cath lab 1 5 East 3
Mean age: 47 years **Mean number of years nursing experience:** 18 years **Mean number of years critical care nursing experience:** 8 years	

Table 4.2 shows the mean age level and the mean number of years experience in nursing and in critical care nursing in the ADN group (n=11) and the BSN/MSN group (n=8). The BSN and MSN nurses were grouped together for simplicity's sake, partly because of such a small number (one) of MSN nurses, but mainly because a BSN is the basic requirement before continuing on to a MSN. The means in the two groups were very similar. The mean age of ADN nurses was 46 years, and the mean age of BSN/MSN nurses was 44 years. The mean number of years nursing experience for ADN nurses was 11 years and the mean for BSN/MSN was 8 years. The mean number of years critical care nursing experience for ADN nurses was 11 years and the mean for BSN/MSN nurses was 8 years.

Table 4.2 The mean age, years nursing experience and years critical care nursing experience with regards to educational level

	ADN Nurses			*BSN/MSN Nurses*	
Variable	***N***	***Mean***		***N***	***Mean***
Age	***11***	***47***		***8***	***45***
Years nursing experience	***11***	***18***		***8***	***19***
Years critical care nursing experience	***11***	***10***		***8***	***9***

Table 4.3 shows a further breakdown regarding educational level, showing how many registered nurses work in the different critical care units studied. Of the 8 nurses working in ICU, 75% of them (6) were ADN nurses. Of the 7 working in ER, a greater percentage, 57% (4) were BSN/MSN nurses, versus 43% (3) who were ADN nurses. The nurse from the cath lab was an ADN nurse. Of the three (3) registered nurses working in 5-East, 2 of them were BSN/MSN nurses and 1 was an ADN nurse.

Table 4.3 The number of nurses working in specific units with regards to educational level

Educational data	Units Where Nurses Work				
	ICU	ER	CATH	5 East	Total
Number ADN nurses	6	3	1	1	11
Percentage ADN nurses	75%	42.86%	100%	33.33%	57.89%
Number BSN/MSN nurses	2	4	0	2	8
Percentage ADN/BSN nurses	42.11%	57.14%	0%	66.67%	42.11%
Total number	8	7	1	3	19
Total Percentage	42.11%	36.84%	5.26%	15.79%	100%

4.4 ANALYSIS OF THE DATA

4.4.1 ANALYSIS OF THE COMPLETE SCALE

Table 4.4 shows an analysis of the complete scale. The sample size, n=19, reflects the number of participants who filled out the questionnaire entirely. This table shows the mean score for each question. A mean score is computed by summing all the scores for each question and dividing by the number of subjects (participants). The lowest score recorded is question 12, with a mean score of 2.76 (note: question 4 has a mean score of 2.16, but as mentioned before, in this question and in question 15, adequate knowledge would be indicated by a "6" or a "7", not a "1" or "2" as in the other questions). These averages indicate that as a whole sample, adequate knowledge and expertise does not occur in regards to any area questioned.

Table 4.4

Analysis of Complete Scale

Question	Sample Size	Mean	Standard Deviation
Q1	19	2.89	1.20
Q2	19	3.16	1.30
Q3	19	3.05	1.13
Q4	19	2.16	1.21
Q5	19	3.00	1.29
Q6	19	3.16	1.30
Q7	19	3.11	1.29
Q8	19	3.21	1.13
Q9	19	3.16	1.71
Q10	19	2.95	1.35
Q11	19	3.89	1.20
Q12	19	2.76	0.93
Q13	19	3.89	2.34
Q14	19	3.58	1.46
Q15	19	2.79	1.65
Q16	19	3.89	1.76
Q17	19	4.00	1.80
Q18	19	3.16	1.26
Q19	19	2.84	1.42
Q20	19	2.95	1.48
Q21	19	3.47	1.47
Q22	19	3.63	1.32
Q23	19	3.68	1.34
Q24	19	3.68	1.45

Reliability		
	Chronbach's Alpha	.9416
	Principle Component Analysis	
	One Factor	
	Variance Accounted for	.4537

4.4.2 PERCENTAGES OF CRITICAL CARE NURSES HAVING ADEQUATE KNOWLEDGE IN THEIR OPINION

Table 4.5 shows the percentage of respondents having adequate knowledge/expertise in their opinion regarding individual subject areas tested. The topic is listed with the questions relevant to that topic, along with the number of nurses and corresponding

percentage who scored a "1" or "2" (or a "6" or "7" in questions 4 and 15) indicating competency in their opinion.

This table indicates that only in one area do more than 50% of the respondents have adequate knowledge in their opinion, and that is in the subject of thrombolytic agents (53% of the respondents in question 19). In all the other questions, the percentage of respondents having adequate knowledge in their opinion regarding ACS ranges from 0 to 47%, reflecting an overall knowledge deficit regarding ACS amongst the participants.

Table 4.5 Percentage of nurses having adequate knowledge in their opinion regarding individual subject areas.

Topic	**Question, number and percentage of respondents having adequate knowledge in their opinion regarding individual topics of ACS**
Definition of ACS	Question: 1 19 Respondents answered this question. Nine or 47% of the respondents are of the opinion they understand the definition of ACS
Pathophysiology of ACS	Question: 2 19 Respondents answered this question. Seven or 37% of the respondents are of the opinion they are able to explain to their patients the pathophysiology of ACS.
Patient education regarding in-hospital management of ACS.	Question: 3 19 Respondents answered this question. Six or 32% of the respondents are of the opinion that they can competently explain in-hospital management of ACS to their patients.

	Question, number and percentage of respondents having adequate knowledge in their opinion regarding individual topics of ACS
Physiology of clot formation	Question: 4 19 Respondents answered this question. None of the respondents are of the opinion that they have an adequate knowledge of the physiology of clot formation. They would all benefit to know more about it.
How hyptertension leads to ACS	Question: 5 and 6 Question 5: 19 Respondents answered this question. Four or 21% are of the opinion that they have an adequate understanding of how chronic hypertension can lead to endothelial damage, progressing to plaque formation, plaque rupture and thrombus formation. Question 6: 19 Respondents answered this question. Five or 26% are of the opinion that they are able to explain to their patients why chronic hypertension can lead to endothelial damage, progressing to plaque formation, plaque rupture and thrombus formation.
Q-wave and non-Q-wave MI	Question: 7 19 Respondents answered this question. Seven or 37% are of the opinion that they have an adequate knowledge regarding the difference between a Q-wave and non-Q-wave MI.
Progression of unstable angina or non-Q-wave MI to acute MI.	Question: 8 19 Respondents answered this question. Four or 21% are of the opinion that they adequately understand what factors contribute toward causing unstable angina or non-Q-wave MI to progress to an acute MI.

	Question, number and percentage of respondents having adequate knowledge in their opinion regarding individual topics of ACS.
Cardiac markers	Question: 9, 10, 11 Question 9: 19 Respondents answered this question. Eight or 42% are of the opinion that they have adequate knowledge regarding the cardiac markers myoglobin, CPK-MB and Troponin (I and T), and are able to interpret these tests correctly. Question 10: 19 Respondents answered this question. Eight or 42% are of the opinion that they are able to adequately explain to their patients the interpretation of cardiac marker values. Question 11: 19 Respondents answered this question. None are of the opinion that they adequately understand the advantages and disadvantages of the cardiac markers in diagnosing a non-Q-wave and a Q-wave MI.
ECG readings	Question: 12 19 Respondents answered this question. Nine or 47% are of the opinion that they have adequate knowledge regarding ST segment and T-wave changes indicative of myocardial ischemia, injury and infarction
Sheath removal	Question: 13 19 Respondents answered this question. Seven or 37% are of the opinion that they are experienced in sheath removal.
Low molecular weight heparin	Question: 14 19 Respondents answered this question. Four or 21% are of the opinion that they have adequate knowledge regarding the drug actions of LMWH and understand the advantages and disadvantages of using them verses heparin.

	Question, number and percentage of respondents having adequate knowledge in their opinion regarding individual topics of ACS.
GB IIb/IIIa inhibitors	Question: 15, 16, 17 Question 15: 19 Respondents answered this question. Three or 16% are of the opinion that they have adequate knowledge regarding GB IIa/IIIb inhibitors and their use in treating unstable angina and non-Q-wave MI. Question 16: 19 Respondents answered this question. Six or 32% have administered GB IIb/IIIa inhibitors after PCI and stent procedures and are of the opinion that they have an adequate understanding of the rationale for the use of these drugs. Question 17: 19 Respondents answered this question. Five or 26% are of the opinion that they are able to adequately explain to their patients the rationale for using GB IIb/IIIa inhibitors after PCI procedures.
Thrombolytic agents	Question: 18, 19, 20 Question 18: 19 Respondents answered this question. Seven or 37% are of the opinion that they have adequate knowledge regarding the therapeutic actions and indications for thrombolytic agents. Question 19: 19 Respondents answered this question. Ten or 53% are of the opinion that they have adequate knowledge of, and are able to recognize, potential complications arising from the use of thrombolytic agents. Question 20: 19 Respondents answered this question. Nine or 47% are of the opinion that they have adequate knowledge regarding contraindications of administering thrombolytics and/or GB IIb/IIIa inhibitors.

	Question, number and percentage of respondents having adequate knowledge in their opinion regarding individual topics of ACS.
Beta-blockers	Question: 21 19 Respondents answered this question. Five or 26% are of the opinion that they have adequate knowledge regarding the rationale for administering beta-blockers to patients who have recently undergone a MI.
Regularity of patient education	Question: 22 19 Respondents answered this question. Three or 16% regularly provide education to their patients regarding ACS pathophysiology and the medications used to treat ACS.
Medications for ACS patients	Question: 23, 24 Question 23: 19 Respondents answered this question. Two or 11% are of the opinion that they have adequate knowledge regarding the drug actions and uses for certain medications prescribed for ACS patients being discharged from the hospital. Medications include: calcium-channel blockers, long-acting nitrates, beta-blockers, ACE inhibitors, ASA, clopidogrel, ticlopidine and warfarin. Question 24: 19 Respondents answered this question. Three or 16% are of the opinion that they able to adequately explain to their patients before discharge the uses and side effects of the medications listed in question 23.

4.4.3 CORRELATIONAL ANALYSIS

Annexure IV , ***The Correlational Analysis,*** is a detailed breakdown illustrating the correlation between three of the independent variables, namely age, number of years

nursing experience, and number of years critical care nursing experience as well as the scores on each question. The level of significance (p-value) in such an analysis is an index of how probable it is that the findings are reliable. In other words, *significance* means that "the obtained results are unlikely to have been the result of chance (Polit & Hunger 1994: 408). For example, if a report indicates that a finding was significant at the 0.05 level, that means that only 5 times out of 100 would the obtained result be haphazard or due to chance. A *nonsignificant* result indicates that the result could have occurred as a result of chance. This analysis reflects that, out of 75 different findings, only 11 of them were found to be significant. Significance in this study = p-value less than/equal to 0.16. Normally if a p-value is less than .05 it is said to be significant. However, in this research study, because the sample size was small, significance must be judged differently, and if the p-value was less than or equal to 0.16, it could be said that a trend toward significance occurred.

Of the 11 significant findings, two have *positive relationships* (question 4 and 22). A positive relationship between two variables exists when there is a tendency for high values on one variable to be associated with high values on the other. In question 4, as age increases, the respondents' scores increase. In the case of this particular question, this means that increased age correlates with the respondents' opinion that they *would not* benefit from a thorough understanding of clot formation and lysis physiology. In other words, the older a respondent, the more he/she is of the opinion that he understands clot formation and lysis physiology and does not need further understanding. In question 22, as the years of nursing experience increase, so do the respondents' scores increase, indicating that the more years nursing experience the respondents have, the less regularly they provide patient education to patients diagnosed with unstable angina, non-Q-wave and Q-wave MI. The other 9 significant findings have negative (inverse) relationships, indicating that as the variable amount decreases, the scores increase.

It is interesting to note that the smallest p-values (where p is less than 0.01 and p is less than 0.05 correspond with questions 19 and 20 respectively) correlate with the findings in ***Table 4.5*** in which the highest percentage of respondents who feel they have adequate

knowledge regarding ACS was also found to be in questions 19 and 20 (57% of the nurses had adequate knowledge regarding thrombolytic agents in these two questions). This emphasizes that thrombolytic agents is the subject area about which most of the nurses feel they are most knowledgeable.

It should also be noted that of the 11 significant findings, 7 of them indicate that as the number of years *critical or acute care nursing experience* increased, so did the respondents rate their adequacy of knowledge regarding ACS. The researcher assumed before conducting this study that this would be true.

With so few findings significant, it is safe to say that age, number of years nursing experience and number of years critical or acute care nursing experience did not have an impact on the scores, i.e. there is no significant correlation between the questionnaire scores and these three independent variables. This apparent trend could possibly be due to the small sample size. Typically a larger sample size will increase the probability of the findings being significant. However, let it be noted that the number of years critical care nursing experience has the most impact on score values in this analysis.

4.4.4 EFFECT OF EDUCATIONAL LEVEL ON SCORES

Annexure V, The Effect of Education Level on the Total Scale and Individual Questions, displays the mean scores of the total scale and the individual questions and reflects whether or not there is significance between the scores and the nurses' level of education. Normally when comparing two groups, one uses a t-test. However, ANOVA (analysis of variance), which was used in this analysis, is an alternative to t-test. F-value is a statistic resulting from ANOVA, and the p-value is derived from the F-value. Of the 25 findings reported, 9 (36%) are significant (where p-value is less than or equal to 0.16). With 36% of the findings significant, the researcher concludes there is a trend toward the level of education significantly influencing the overall scores.

In every one of these significant findings, with the exception of one (question 1), a lower score corresponds with the ADN nurses, indicating that the ADN nurses were more of the opinion that they had adequate knowledge. The researcher expected the opposite would occur. ADN nurses graduate from a two-year associate degree of nursing school (all prerequisites must be completed before entering the nursing program). BSN nurses graduate from a more rigorous and demanding four-year university nursing program. The researcher expected the BSN group to be the ones perceiving they have adequate knowledge regarding ACS, which turned out not to be the case in these findings.

4.5 SUMMARY

Both the analysis of the complete scale and the analysis of the percentages of respondents having adequate knowledge individual subject areas reflect an overall knowledge deficit amongst the participants regarding ACS in their opinion. Thrombolytic agents appear to be the subject that the respondents feel they are most knowledgeable about. Age and years of general nursing experience did not impact scores in any significant way. However, the number of years critical or acute care nursing experience did have a significant impact, which the researcher had predicted. The level of education appeared to have a significant impact on scores, but surprisingly, the group with the lesser amount of education (ADN nurses) scored better than the group with a longer, more demanding education (BSN nurses).

With these findings in mind, an educational in-service on ACS will be prepared and given to the critical or acute care nurses at the hospital where the study was performed. All content to be taught will be reviewed first by the hospital's critical care nurse educator and one other critical care nurse with extensive cardiovascular intensive care nursing experience. Attendance will be voluntary. 4.2 hours of continuing nursing education will be awarded to each participant upon completion of the class. The goal of this in-service training session is to broaden and deepen critical care nurses' knowledge base of ACS so they may make more informed independent judgments, resulting in improved nursing care.

CHAPTER 5

CONCLUSIONS, RECOMMENDATIONS AND GUIDELINES FOR IN-SERVICE TRAINING SESSION

5.1 INTRODUCTION

The researcher's interest in cardiac nursing and the interest to discover how much critical or acute care nurses feel they know about ACS motivated the researcher to undertake this study. A literature study was subsequently completed and a research tool developed: a questionnaire with a rating scale.

The implementation of the questionnaire was done by the researcher. The data was processed and presented in the previous chapter. The goal of this study was twofold: first, to explore and describe the opinion of critical or acute care nurses in a specific setting regarding their knowledge of ACS, and secondly, to develop a four-hour in-service training session on ACS for all critical care nurses where this research study was performed. This chapter is concerned with recommendations and presenting an outline of the final goal, formulating an in-service presentation on ACS for critical care nurses.

5.2 CONCLUSIONS

The conclusions are divided up into demographic data and knowledge level.

5.2.1 DEMOGRAPHIC DATA

- The participants were full-time or part-time staff in one of four critical care units: ICU, ER, cath lab and 5-East (Telemetry).

- The mean age of the participants was 47 years old. The mean number of years nursing experience was 18 years, and the mean number of years critical care nursing experience was 8 years.

- One (1) nurse had a master's degree level of education. Of 18 registered nurses, 7 had a BSN degree and 11 had an ADN degree.

- 75% of the respondents working in ICU who filled out the questionnaires were registered nurses who had ADN degrees, whereas 66% of the registered nurses working in 5-East (Telemetry) who filled out the questionnaires had a BSN or MSN degree. Slightly more than half (57%) of the nurses working in ER had a BSN or MSN degree, and the one nurse from the cath lab had an ADN degree.

5.2.2 KNOWLEDGE LEVEL

Critical or acute care nursing involves the use of high-level critical thinking skills within the nursing process, and a sound knowledge base is vital to independent judgments and decision making. If the nurse has a knowledge deficit in a particular area, his/her ability to think critically and make the necessary clinical decisions will be hindered. She will not have a thorough knowledge of the disease process and treatments for her patients, which hinders her ability to give the best nursing care possible.

A questionnaire with a rating scale was used to ascertain the opinion of critical care nurses regarding their knowledge of ACS. On all questions except two (questions 4 and 15), if a person circles a "1 or "2", the researcher assumed that indicated the respondents felt they have an adequate knowledge regarding the particular aspect of ACS being questioned. The reverse held for questions 4 and 15, in which case circling a "6" or "7" indicated the respondents felt they have an adequate knowledge. The research findings indicated an overall knowledge deficit regarding ACS amongst the participants.

The analysis of the percentage of critical care nurses indicating they feel they have adequate knowledge and expertise regarding each individual subject area tested revealed that in only one area, thrombolytic agents, were over 50% of the opinion they have adequate knowledge. In all other subject areas, the percentages ranged from 1 to 48%,

reflecting that the respondents feel they have an overall knowledge deficit regarding ACS.

The correlational analysis revealed that neither age nor number of years nursing experience had any significant effect on the scores. However, 8 of the 11 significant findings showed a correlation between increased number of years critical or acute care nursing experience and an increased perception of what the respondents feel they know. These findings were reflected in questions 8, 12, 14, 16, 17, 18, 19 and 20, where the p-value was found to be equal to or less than 0.16. Questions 8, 12, 14, 16 and 17 covered, respectively, the factors contributing toward unstable angina, non-Q-wave and Q-wave MI; ST-segment and T-wave changes indicating myocardial ischemia, injury or infarction; the drug actions of low molecular weight heparin and the advantages and disadvantages of using them versus heparin; administration of GB IIb/IIIa inhibitors to patients after PTCA and stent procedures; and ability to explain the rationale for use of GB IIb/IIIa inhibitors after PTCA and stent procedures. Questions 28, 19 and 20 covered the subject of thrombolytic agents, their use, drug actions and potential complications.

The level of education had a positive significant effect on 9 out of the 24 items questioned. Interestingly, the nurses with ADN degrees (two-year RN degree) had scores reflecting a perception of greater knowledge than the nurses with four-year BSN degrees.

5.3 OUTLINE OF ACS EDUCATIONAL IN-SERVICE TRAINING SESSION

Based on the research study findings, it is apparent that a knowledge deficit regarding ACS exists amongst the participating critical care nurses in this study. A four-hour in-service training session will be presented for all critical care nurses in the hospital where the study was performed. Attendance will be voluntary, and continuing education credits will be awarded to all participants who successfully complete the class. The premise behind presenting this training is that formal education in critical care nursing enhances the knowledge and skills of nurses working in the critical care environment (Sole 1997:58). As the nurses broaden their knowledge base regarding ACS, they will be more

equipped to think critically and make sound decisions within the nursing process and provide optimum care for their patients. ***Annexure VI*** gives an outline of a four-hour in-service training session to be presented to critical care nurses. The information covered in the literature review in Chapter 2 will be utilized to present this in-service training session.

5.4 LIMITATIONS OF STUDY

5.4.1 SAMPLE SIZE

Because of a small sample size, it was difficult to defend the validity of the findings. Many of the findings turned out to be nonsignificant, and it is possible that with a larger sample size, more of the findings would have been found to be significant.

5.4.2 INTERNAL VALIDITY

Internal validity was difficult to achieve. The method of distribution and collection of the questionnaires was not well controlled, and consequently the researcher had to assume that the participants did not consult outside sources of information. The small sample size, in contrast with the number of questionnaires distributed in nurses' mailboxes, may be due in part to the lack of control over the method of distribution and collection. It would have been more advantageous to assemble the nurses together and have them answer the questionnaire under supervision.

Although validity was enhanced in a certain sense by the fact that the knowledge of the respondents was not actually "tested", but that the respondents actually had to rate their own knowledge, it might also have had a negative side. In this situation, they did not feel threatened, but it cannot be said that all those rating their knowledge for example as a "1" or "2" would be able to score the same in a formal test. A formal test would thus had been a more objective instrument.

5.4.3 EXTERNAL VALIDITY

External validity was also difficult to achieve. The sample size was small, which made it difficult to generalize the study findings to a larger population. In addition, the mean age and mean number of years nursing experience were fairly high (47 years and 18 years, respectively) and the researcher questions whether these figures accurately reflect the general population of critical care nurses. The convenient sample used is not representative of the total population. The more representative the study population is of the target population, the greater is the accuracy with which generalizations can be made from the sample to the total population (Abdellah & Levine 1979: 155).

5.4.4 QUESTION 22

This question was poorly worded and did not fit in with the other questions because it asked whether or not education was provided regularly to patients. The question did not provide any indication of the participants' knowledge about ACS. It should have been eliminated during the refinement phase before the questionnaires were printed and distributed.

5.4.5 QUESTIONS 4 AND 15

These questions were worded in such a way that adequate competency was indicated by circling a "6" or "7", instead of circling a "2" or "3" as in the other questions, and this made the analysis more complicated. It would have been easier if these two questions were worded so that adequate knowledge could be indicated in the same manner as in all the other questions. This was overlooked during the refinement phase of developing the questionnaire.

5.5 RECOMMENDATONS

5.5.1 RECOMMENDATIONS REGARDING CRITICAL CARE NURSING EDUCATION

- Each topic covered in the literature study should be specifically included in curriculum for the in-service training session.
- The in-service training session should be given on a regular basis. Rather than present all the material in one training session, as is planned, cover specific topics on a weekly or bi-weekly basis on a on-going basis. The nurses will be more able to absorb the information if a smaller amount of subject material is covered regularly. Additionally, one can review the material covered during previous training sessions to ensure that the participants are actually learning the subject material being presented.

5.5.2 RECOMMENDATIONS FOR CRITICAL OR ACUTE CARE NURSING PRACTICE

- Nursing care plans should be made specifically for ACS patients. This will ensure that there is a continuity of care from nurse to nurse, and that all aspects of nursing care for ACS patients are being performed.

5.5.3 RECOMMENDATIONS FOR FURTHER RESEARCH

- An instrument should be developed specifically in a "test format". Such an instrument is more objective and is not difficult to score or analyze.
- A larger population should be chosen, and the study should be repeated.
- The instrument should be refined as suggested.
- A comparative study could be made between using instrument of this study and using a test-like instrument measuring knowledge, and then correlate the data

regarding the comparisons. It would be interesting to determine the correlation between what the respondents actually knew and how the rate their knowledge.

5.6 SUMMARY

The purpose of this research study was two-fold -- first, to explore and describe the knowledge level of critical or acute care nurses regarding acute coronary syndromes (ACS), and secondly, to design an in-service training on ACS for the critical care nurses in the hospital in which the study was performed. The results of the study revealed an overall knowledge deficit regarding ACS amongst the nurses participating in the study. Because of the small sample size, it is difficult to generalize these findings to the total population of critical care nurses. An in-service education training class on ACS has been prepared and will be offered to all critical care nurses in the hospital where the study was performed. This is based on the premise that formal education in critical or acute care nursing enhances the knowledge and skills of nurses working in the critical care environment. It is hoped that as the nurses gain knowledge and expertise in this particular area, they will share this knowledge with others.

This research study was based on the premise that critical thinking is absolutely vital to the nursing process. Critical or acute care nursing is challenging, and critical care nurses should have a sound knowledge base and the ability to integrate information within the context in which it is gathered in order to provide the skilled and competent intensive care for patients whose conditions may change rapidly. Nurses who care for ACS patients should know the pathophysiology of coronary artery disease and understand the rapidly evolving treatment options for ACS in order to provide optimum nursing care and be able to answer questions and provide guidance to their patients and other members of the healthcare team.

Dear Critical Care Nurses, ***Annexure I***

Enclosed you will find a questionnaire about ***Acute Coronary Syndromes (ACS)***. The purpose of this questionnaire is to ascertain at what level of knowledge of ACS are critical care nurses at **UTCH** at, and then analyze this data and present an in-service in the near future about ***ACS*** that will inform us, as nurses, about the pathophysiology of ***ACS*** and how we can provide optimum patient care for our patients diagnosed in this continuum. This data will also be presented in a dissertation on ***ACS*** that I am writing.

Please take a few minutes to fill this questionnaire out and then put it back in the **manila envelope** labeled ***Acute Coronary Syndromes Questionnaire*** that is posted near your mailboxes. **Please fill this out at work and do not take it home!**

For confidentiality's sake, please do not write your name on the questionnaire. **Simply sign and date this page below,** which will inform me that you have given me your written consent to use the questionnaire you filled out, **and then detach this page and put both this page and your completed questionnaire in the manila envelope.** Another reason for signing this page .... I would like to give you a *thank you* for helping out, and I can only do that if you sign your name! **:)**

Thank you for participating in this study .... I truly appreciate your help, and hope that we will all learn more about this interesting subject!

Sincerely,

Carol Price R.N.

NAME _____

UNIT I WORK IN _____

DATE _____

ANNEXURE II

General Information: fill in the blank or circle appropriate answer

1. Age _____

2. Gender: male / female

3. Marital status: married / single / divorced / widowed

4. Education level: LVN / ADN / BSN / MA, MN, MSN / PhD

5. What unit you work in: ICU / ER / OR / Cath Lab / 5 East

6. Years experience as a nurse _____

7. Years experience in critical care nursing _____

The following questions will be answered using a scale of 1 - 7.

1= strongly agree
2 = agree
3 = somewhat agree
4 = neither agree nor disagree
5 = somewhat disagree
6 = disagree
7= strongly disagree

1. I understand the definition of "Acute Coronary Syndromes" (ACS)

1 2 3 4 5 6 7

2. I am able to explain to my patients the pathophysiology of acute coronary syndromes

1 2 3 4 5 6 7.

3. I am able to explain in-hospital management (including non-invasive and invasive) of acute coronary syndromes to my patients.

1 2 3 4 5 6 7

4. I would benefit from a thorough understanding of clot formation and lysis physiology.

1 2 3 4 5 6 7

5. I understand the pathophysiological reasons why chronic hypertension can lead to endothelial damage, plaque formation, plaque rupture and thrombus formation.

1 2 3 4 5 6 7

6. I am able to explain to my patients why chronic hypertension can lead to endothelial damage, plaque formation, plaque rupture and thrombus formation.

1 2 3 4 5 6 7

7. I understand the difference between a Q-wave and a non-Q-wave myocardial infarction (MI).

1 2 3 4 5 6 7

8. I understand what factors contribute toward unstable angina and a non-Q-wave MI progressing to an acute MI.

1 2 3 4 5 6 7

9. I am familiar with cardiac markers (CPK-MB, myoglobin, Troponin I and Troponin T) and am able to interpret these tests correctly.

1 2 3 4 5 6 7

10. I am able to explain to my patients the interpretation of cardiac markers.

1 2 3 4 5 6 7

11. I understand the advantages and disadvantages of each of the cardiac markers in diagnosing a non-Q-wave and Q-wave MI.

1 2 3 4 5 6 7

12. I am familiar with the ST segment and/or T-wave changes indicative of myocardial ischemia, injury and infarction.

1 2 3 4 5 6 7

13. I am experienced in sheath removal after PTCA and stent placement.

1 2 3 4 5 6 7

14. I understand the drug actions of low molecular weight heparins and the advantages/disadvantages of using them verses using heparin.

1 2 3 4 5 6 7

15. I would benefit from learning more about glycoprotein IIb/IIIa receptor inhibitors (abciximab=Reopro, eptifibatide=Integrilin, tirofiban=Aggrastat) and their use in treating unstable angina and non-Q-wave MI.

1 2 3 4 5 6 7

16. I have administered GB IIb/IIIa inhibitors to patients after PTCA and PTCA/stent procedures and have understood the rationale for these drugs.

1 2 3 4 5 6 7

17. I am able to explain to my patients the rationale for using IIb/IIIa inhibitors after PTCA/stent procedures.

1 2 3 4 5 6 7

18. I understand the therapeutic actions and indications for thrombolytic agents (alteplase, anistreplase, reteplase, streptokinase, urokinase).

1 2 3 4 5 6 7

19. I have a thorough knowledge of, and am able to recognize, potential complications that can occur with patients receiving thrombolytic agents.

1 2 3 4 5 6 7

20. I am aware of the contraindications to administering thrombolytic agents and/or GB IIb/IIIa inhibitors.

1 2 3 4 5 6 7

21. I understand the rationale behind administering beta-blockers for patients who have recently undergone a non-Q-wave or Q-wave MI.

1 2 3 4 5 6 7

22. I regularly provide patient education regarding disease pathophysiology and drug actions and their side effects to my patients diagnosed with unstable angina, non-Q-wave and Q-wave MI.

1 2 3 4 5 6 7

23. I understand the drug actions and use for the following medications used for patients discharged from the hospital: calcium channel blockers, long-acting nitrates, beta blockers, angiotension-converting enzyme (ACE) inhibitors, ASA, clopidogul, ticlopidine, and warfarin.

1 2 3 4 5 6 7

24. I am able to explain to my patients before they are discharged the uses and side effects of the drugs listed in question # **23**.

1 2 3 4 5 6 7

ANNEXURE III

Carol G. Price R.N.
P.O. Box 1400
Van, TX 75790
tel. (903) 963-3267
email: ywam@writeme.com

April 17, 2000

Ms. Anne DeWitt, RN, MSN
Nurse Executive
University of Texas Health Center
11937 US Highway 271
Tyler, TX 75708-3154

Dear Anne,

I am in the process of writing my dissertation of limited scope for my Master's degree in Advanced Clinical Nursing through the University of South Africa. Part of the requirement for this degree is that I conduct a research study. I am writing to ask your permission to conduct this research study at UTHC.

I would like to do a research study at UTHC to explore and describe the knowledge of critical care nurses regarding *Acute Coronary Syndromes* (ACS). The title of the research is: *THE KNOWLEDGE OF CRITICAL CARE NURSES REGARDING ACUTE CORONARY SYNDROMES.* The study will involve the use of a questionnaire to ascertain the nurses' knowledge level regarding ACS. Enclosed you will find a copy of the questionnaire.

Participation in this study will be voluntary for critical care nurses working in ICU, OR, ER, cardiac cath lab and 5-East (Telemetry). As you can see from the cover letter to the nurses, anonymity and confidentiality will be ensured, and informed consent forms are signed by all participants.

One of my purposes in this study is to prepare an in-service training class on ACS once I have ascertained the knowledge level regarding ACS amongst the nurses.

Would you please look over the questionnaire, let me know if there are any changes you would recommend, let me know if it is alright if I proceed with this research study, using this questionnaire?

Thank you so much!

Sincerely,

Carol Price R.N.

Annexure IV

Correlational Analysis

Question		Age	Nurse Experience	Critical Care Experience
Total	Correlation	.0560	-.0180	-.3134
	Sample Size	17	19	19
	Probability	ns	ns	ns
Q1	Correlation	.0737	-.1047	-.0671
	Sample Size	21	24	24
	Probability	ns	ns	ns
Q2	Correlation	.0875	.1258	-.1632
	Sample Size	21	24	24
	Probability	ns	ns	ns
Q3	Correlation	.1073	.0994	-.0646
	Sample Size	21	24	24
	Probability	ns	ns	ns
Q4	Correlation	.3590	-.0068	-.0436
	Sample Size	21	24	24
	Probability	p = .11	ns	ns
Q5	Correlation	.2665	-.0139	-.2187
	Sample Size	21	24	24
	Probability	ns	ns	ns
Q6	Correlation	.2305	-.0483	-.2574
	Sample Size	20	23	23
	Probability	ns	ns	ns
Q7	Correlation	-.0553	.0385	-.2000
	Sample Size	21	24	24
	Probability	ns	ns	ns
Q8	Correlation	-.0238	-.2583	-.3285
	Sample Size	21	24	24
	Probability	ns	ns	p = .12

Correlational Analysis
(cont)

Question		Age	Nurse Experience	Critical Care Experience
Q9	Correlation	-.0763	-.1797	-.1888
	Sample Size	21	24	24
	Probability	ns	ns	ns
Q10	Correlation	.2208	.1387	-.1705
	Sample Size	21	24	24
	Probability	ns	ns	ns
Q11	Correlation	-.2117	-.2067	-.2796
	Sample Size	21	24	24
	Probability	ns	ns	ns
Q12	Correlation	-.1334	.1321	-.3093
	Sample Size	21	24	24
	Probability	ns	ns	p = .14
Q13	Correlation	.1406	.0537	-.2258
	Sample Size	21	24	24
	Probability	ns	ns	ns
Q14	Correlation	-.3138	-.2349	-.3404
	Sample Size	21	24	24
	Probability	p = .16	ns	p = .10
Q15	Correlation	.2387	.0735	-.0906
	Sample Size	21	24	24
	Probability	ns	ns	ns
Q16	Correlation	-.1000	.0367	-.3543
	Sample Size	20	23	23
	Probability	ns	ns	p = .10
Q17	Correlation	-.1407	-.0435	-.3533
	Sample Size	20	23	23
	Probability	ns	ns	p = .10

Correlational Analysis
(cont)

Question		Age	Nurse Experience	Critical Care Experience
Q18	Correlation	-.1326	-.2123	-.3459
	Sample Size	21	23	23
	Probability	ns	ns	p = .11
Q19	Correlation	-.1303	-.3182	-.5465
	Sample Size	21	23	23
	Probability	ns	p = .14	p < .01
Q20	Correlation	-.2170	-.1805	-.4186
	Sample Size	21	23	23
	Probability	ns	ns	p < .05
Q21	Correlation	.0304	.2613	-.2553
	Sample Size	21	23	23
	Probability	ns	ns	ns
Q22	Correlation	.2023	.3103	-.0177
	Sample Size	20	22	22
	Probability	ns	p = .16	ns
Q23	Correlation	.1906	.1647	-.0319
	Sample Size	19	21	21
	Probability	ns	ns	ns
Q24	Correlation	.1372	.0679	-.0841
	Sample Size	20	22	22
	Probability	ns	ns	ns

Annexure V

Effect of Education Level on the Total Scale and Individual Questions

Question	Level	Sample Size	Mean (sd)	F-value	p-value
Total	ADN	11	65.44 (16.88)	2.81	p = .12
	BSN/Masters	8	82.40 (20.37)		
Q1	ADN	11	2.82 (1.08)	2.61	p = .12
	BSN/Masters	8	2.13 (0.64)		
Q2	ADN	11	2.73 (1.10)	0.29	ns
	BSN/Masters	8	2.50 (0.63)		
Q3	ADN	11	2.55 (1.13)	0.20	ns
	BSN/Masters	8	2.75 (0.71)		
Q4	ADN	11	1.91 (1.04)	0.01	ns
	BSN/Masters	8	1.88 (0.83)		
Q5	ADN	11	2.91 (1.38)	0.99	ns
	BSN/Masters	8	2.38 (0.74)		
Q6	ADN	11	2.91 (1.38)	0.01	ns
	BSN/Masters	7	2.86 (0.90)		
Q7	ADN	11	3.00 (1.34)	0.99	ns
	BSN/Masters	8	3.25 (1.28)		
Q8	ADN	11	3.18 (1.33)	0.14	ns
	BSN/Masters	8	3.38 (0.74)		
Q9	ADN	11	2.73 (1.68)	0.03	ns
	BSN/Masters	8	2.63 (0.52)		
Q10	ADN	11	2.36 (0.92)	2.19	p = .16
	BSN/Masters	8	3.00 (0.93)		
Q11	ADN	11	3.82 (0.98)	0.58	ns
	BSN/Masters	8	3.50 (0.76)		
Q12	ADN	11	2.45 (0.82)	0.02	ns
	BSN/Masters	8	2.50 (0.76)		

Effect of Education Level on the Total Scale and Individual Questions (cont)

Question	Level	Sample Size	Mean (sd)	F-value	p-value
Q13	ADN	11	2.73 (2.20)	4.37	$p < .05$
	BSN/Masters	8	4.88 (2.23)		
Q14	ADN	11	2.91 (1.38)	3.24	$p = .09$
	BSN/Masters	8	4.00 (1.20)		
Q15	ADN	11	2.45 (1.04)	0.16	ns
	BSN/Masters	8	2.75 (2.12)		
Q16	ADN	10	2.90 (1.29)	7.51	$p < .05$
	BSN/Masters	8	4.75 (1.58)		
Q17	ADN	10	3.10 (1.20)	5.63	$p < .05$
	BSN/Masters	8	4.88 (1.96)		
Q18	ADN	11	2.64 (0.81)	0.14	ns
	BSN/Masters	7	2.86 (1.68)		
Q19	ADN	11	2.09 (1.14)	0.64	ns
	BSN/Masters	7	2.57 (1.40)		
Q20	ADN	11	2.18 (1.08)	1.45	ns
	BSN/Masters	7	3.00 (1.83)		
Q21	ADN	11	3.18 (1.25)	0.15	ns
	BSN/Masters	7	3.43 (1.40)		
Q22	ADN	11	3.18 (0.60)	5.90	$p < .05$
	BSN/Masters	6	4.50 (1.64)		
Q23	ADN	10	3.00 (1.15)	4.38	$p = .06$
	BSN/Masters	6	4.33 (1.37)		
Q24	ADN	11	3.00 (1.10)	4.85	$p < .05$
	BSN/Masters	6	4.33 (1.37)		

ANNEXURE VI

OUTLINE OF IN-SERVICE TRAINING SESSION ON ACS

1. Define Acute Coronary Syndromes (ACS)	A. Define: 1. Unstable angina 2. Non-Q-wave MI 3. Q-wave MI
2. Correlate the process in the clotting cascade, where thrombus formation occurs, and at which point in the clotting cascade specific medications exert their affects.	A. Factors triggering initiation of process – extrinsic and intrinsic. B. Mechanisms of platelet aggregation and platelet plug. C. Thrombus formation. D. Clotting cascade and thrombus formation. E. Pharmacologic agents interfering in thrombus formation. 1. Aspirin 2. Second-generation platelet inhibitors: ticlopidine and clopidogrel (Ticlid & Plavix). 4. GP IIb/IIIa inhibitors 5. Heparin 6. Low molecular weight heparins
3. Describe the physiology, including results of damage, of the endothelium.	A. Physiology of the endothelium. B. Factors involved with endothelium damage. C. Results of damage.
4. Correlate endothelium damage to ACS.	A. Endothelium damage sequelae. B. Treatment with ACE inhibitors.
5. Explain the process of coronary artery plaque formation	A. Mechanism of plaque formation. 1. Serum lipids 2. Fatty streaks 3. Monocytes 4. Macrophages and foam cells B. Lipid core formation.

	C. Fibrous cap stabilization. D. Occlusion 1. Partial 2. Total
6. Correlate the mechanisms of plaque rupture and ACS.	A. Mechanisms of plaque rupture. B. Plaque rupture sequelae 1. Ischemia 2. Non-Q-wave MI 3. Q-wave MI C. Use of statins.
7. Identify specific ECG changes in unstable angina, non-Q-wave MI and Q-wave MI.	A. Q-wave B. ST-segment elevation or depression C. T-wave
8. Describe the measurement, advantage and disadvantage of each of the three most commonly used cardiac markers. Include normal and abnormal values.	A. Myoglobin B. CK-MB C. Troponins (I and T)
9. Compare and contrast the various drugs used in the treatment of ACS including mechanisms of action, side effects and nursing actions.	A. Morphine sulfate B. Nitrates C. Beta-blockers D. Calcium-channel blockers E. ACE inhibitors F. Thrombolytics
10. Correlate rationale for current ACS treatments with the knowledge of pathophysiology related to ACS.	A. Diagnostic: angiography. B. Interventions / Procedures: PCI, FPCI C. Pharmacologic
11. Compare the UTHC protocols for chest pain, acute MI and/or ACS to current medical treatment guidelines.	A. Chest Pain Rapid Evaluation Pathway / Protocol B. ACS Pathway / Protocol
12. Acknowledge ongoing research and their possible implications in future treatment of ACS.	A. Current research 1. Projects 2. Future predictions

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