Nursing Care Plan

A Client with Acute Myocardial Infarction

Betty Williams, a 62-year-old psychologist, is admitted to the emergency department with complaints of severe substernal chest pain. Mrs. Williams states that the pain began after lunch, about 4 hours ago. She initially attributed the pain to indigestion. She described the pain, which now radiates to her jaw and left arm, as “really severe heartburn.” It is accompanied by a “choking feeling,” severe shortness of breath, and diaphoresis. The pain is unrelieved by rest, antacids, or three sublingual nitroglycerin tablets (0.4 mg).

Oxygen is started per nasal cannula at 5 L/min. Central and peripheral intravenous lines are inserted. A 12-lead ECG and the following labwork are obtained: cardiac troponins, CK and CK isoenzymes, ABGs, CBC, and a chemistry panel. Morphone sulfate relieves Mrs. Williams’s pain.

Mrs. Williams’s medical history includes type 2 diabetes, angina, and hypertension. She has a 45-year history of cigarette smoking, averaging 1.5 to 2 packs per day. Family history reveals that Mrs. Williams’s father died at age 42 of AMI, and her paternal grandfather died at age 65 of AMI. Mrs. Williams is taking the following medications: tolbutamide (Orinase), hydrochlorothiazide, and isosorbide (Isordil).

Based on ECG changes and cardiac markers, an acute anterior MI is diagnosed. Mrs. Williams has no contraindications to thrombolytic therapy and is deemed a good candidate. Intravenous alteplase (t-PA, Activase) is given by bolus followed by intravenous infusions of alteplase and heparin. She is transferred to the coronary care unit (CCU).

ASSESSMENT

Dan Morales, RN, is Mrs. Williams’s primary care nurse. Mrs. Williams is alert and oriented to person, place, and time. Vital signs are T 99.6°F (37.5°C), P 118, R 24 with adequate depth, and BP 172/92. Auscultation reveals an S4 and fine crackles in the bases of both lungs. The ECG shows sinus tachycardia with occasional PVCs. Her skin is cool and slightly diaphoretic. Capillary refill is less than 3 seconds, and peripheral pulses are strong and equal. Her nail beds are pink.

A triple-lumen central line is in place. Nitroglycerin is infusing at 200 mcg/min in the distal lumen; the alteplase infusion is in the middle lumen; and a heparin infusion is in the proximal lumen. The peripheral intravenous line has a saline lock. Mrs. Williams states, “The pain is better since the nurse in the ER gave me a shot. But it has been coming and going. I would rate it a 4 right now, but it was terrible before. The doctor told me that this drug I’m getting will quickly open up the artery that is blocked. I hope it works! Do many people get this drug?”

DIAGNOSES

Based on the assessment, Mr. Morales identifies the following nursing diagnoses:

- Acute pain related to ischemic myocardial tissue
- Anxiety and fear related to change in health status
- Ineffective protection related to the risk of bleeding secondary to thrombolytic therapy
- Risk for decreased cardiac output related to altered cardiac rate and rhythm

EXPECTED OUTCOMES

The expected outcomes specify that Mrs. Williams will:

- Rate chest pain as 2 or lower on a pain scale of 0 to 10.
- Verbalize reduced anxiety and fear.
- Demonstrate no signs of internal or external bleeding.
- Maintain an adequate cardiac output during and following reperfusion therapy.

PLANNING AND IMPLEMENTATION

The following interventions are planned and implemented during the immediate phase of Mrs. Williams’s hospitalization.

- Instruct to report all chest pain. Monitor and evaluate pain using a scale of 0 to 10. Titrate intravenous nitroglycerin infusion for chest pain; stop infusion if systolic BP is below 100 mmHg. Administer 2 to 4 mg morphine intravenously for chest pain unrelied by nitroglycerin infusion.
- Encourage verbalization of fears and concerns. Respond honestly and correctly misconceptions about the disease, therapeutic interventions, or prognosis.
- Assess knowledge of CHD. Explain the purpose of thrombolytic therapy to dissolve the fresh clot and reperfuse the heart muscle, limiting heart damage.
- Explain the need for frequent monitoring of vital signs and potential bleeding.
- Assess for manifestations of internal or intracranial bleeding: complaints of back or abdominal pain, headache, decreased level of consciousness, dizziness, bloody secretions or excretions, or pallor. Test all stools, urine, and vomitus for occult blood. Notify physician immediately of any abnormal findings.
- Monitor for signs of reperfusion: decreased chest pain, return of ST segment to baseline, reperfusion dysrhythmias (e.g., PVCs, bradycardia, and heart block).
- Continuously monitor ECG for changes in cardiac rate, rhythm, and conduction. Assess vital signs.
- Treat dangerous dysrhythmias or other cardiac events per protocol. Notify the physician.
- Discuss continuing cardiac care and rehabilitation.
**CARDIAC RHYTHM DISORDERS**

Heart muscle contracts in response to electrical stimulation. In the normal heart, electrical stimulation produces a synchronized, rhythmic heart muscle contraction that propels blood into the vascular system. Changes in cardiac rhythm affect this synchronized activity and the heart’s ability to effectively pump blood to body tissues.

**THE CLIENT WITH A CARDIAC DYSRHYTHMIA**

A cardiac dysrhythmia is a disturbance or irregularity in the electrical system of the heart. Cardiac dysrhythmias may be benign or have lethal consequences. Prompt recognition and quick action of a lethal dysrhythmia can be life saving.

Dysrhythmias develop for many reasons. Not all are pathologic; some alterations in cardiac rhythm occur in response to events such as exercise or fear. For example, a rapid heart rate due to exercise, fever, or excitement is a normal response to the body’s demand for oxygen or to stimulation of the sympathetic nervous system. Slow heart rates also may be normal. Athletic heart syndrome, which results from long-term training on the heart muscle, allows the heart to beat more slowly and forcefully while maintaining cardiac output and tissue perfusion. Many athletes have a heart rate of less than 60 beats per minute. Aging affects cardiac rhythm as well (see p. 000).

Regardless of cause, a dysrhythmia can significantly affect cardiac performance, depending on heart muscle health. The client’s response to the dysrhythmia is key in determining the urgency and type of treatment needed.

**PHYSIOLOGY REVIEW**

Cardiac muscle is unique. Unlike skeletal muscle tissue, cardiac muscle can generate an electrical impulse and contraction independent of the nervous system.

**Conduction Pathways**

Electrical activity of the heart is normally controlled by the cardiac conduction system, a network of specialized cells and conduction pathways that initiate and spread electrical impulses that cause the heart to beat (see Figure 28–7). Pacemaker cells spontaneously generate electrical impulses at a regular rate. Specialized conduction tissue rapidly transmit these impulses to myocardial cells. Myocardial muscle cells contract in response to the impulse. Electrical stimulation of heart muscle always precedes mechanical contraction.

Pacemaker cells are found throughout the heart. The sinoatrial (SA) or sinus node is the primary pacemaker of the heart. It usually fires at a regular rate of 60 to 100 BPM, initiating impulses that are conducted throughout the heart. The sinus node impulse spreads through the atria via the interatrial pathways. Conduction fibers narrow through the atrioventricular (AV) node, briefly delaying impulse conduction. This delay allows atrial muscle to contract, delivering an extra bolus of blood to the ventricles before they contract (the atrial kick). The AV node also controls the number of impulses that reach the ventricles, preventing extremely rapid heart rates. From the AV node, the impulse travels down the bundle of His, the right and left bundle branches, and to the Purkinje fibers of the ventricular conduction system. The Purkinje fibers terminate in ventricular muscle, prompting mechanical contraction, or systole.

If the sinus node fails, secondary pacemakers in the AV node (with an intrinsic rate of 40 to 60 BPM), and the Purkinje fibers (intrinsic rate of 15 to 40 BPM) take over as the pacemaker at a slower rate. This provides backup mechanism for electrical stimulation of the heart.

**Electrophysiologic Properties**

Four unique properties of cardiac cells allow effective heart function. Three properties are electrical; the fourth is cardiac muscle’s mechanical response to electrical stimulation.