Transport of Intravenous Heparin and Nitroglycerine

Coastal Valleys EMS Agency

Why Use Heparin and Nitroglycerine?

- <u>Acute Coronary Syndromes (ACS)</u> are managed with many drugs
- <u>Heparin</u> is used to control coronary *thrombosis* (clot formation)
- <u>Nitroglycerine</u> is useful in the management of *coronary angiospasm*, *cardiac preload*, and *cardiac afterload*

Areas We Will Cover

Acute Coronary Syndromes
Use of Heparin in ACS
Use of Nitroglycerine in ACS

Acute Coronary Syndrome

Is the term that has become commonly used to refer to a patient presenting with ischemic chest pain. The acute coronary syndromes include-

- Unstable Angina
- Non-ST Segment Elevation Myocardial Infarction (STEMI)
- STEMI

It is important to realize that these syndromes represent a <u>dynamic spectrum</u> of disease, and are part of a continuum

Unstable Angina

- Changes in "stable" patterns
- New onset
- Unrelieved with Ntg.

Non-STEMI vs.STEMI Presentations

- Subendocardium
- S-T changes
- No Q-waves

- Transmural
- Half the full thickness
- Q-waves

Acute Coronary Syndromes

Always have the same initiating eventrupture of an unstable, lipid-rich plaque in a coronary artery

Artery vs. Vein Cross Section



Schematic of the tunica layers



Comparison of all 3 layers



Typical Artery

Typical Vein

Stable Coronary Plaques

- Have a thick fibrous cap protecting them from coronary blood flow
- Are not likely to rupture
- Have less lipid mass
- Frequently produce a significantly narrowed coronary artery lumen

Stable Coronary Plaque

Narrowed Lumen

Small Lipid Core

Thick Fibrous Cap

Stable Plaque



Unstable Coronary Plaques

- Have a much thinner fibrous cap
- Are quite susceptible to rupture
- Have a greater amount of lipid mass
- Often *do not* produce significant coronary narrowing

Unstable Coronary Plaque



Vulnerable Plaque





Rupture of a coronary plaque

The fibrous cap ruptures and the lipid core is exposed to the blood stream

Rupture of a coronary plaque-Thrombosis

Platelets aggregate around the exposed lipid core and initiate thrombus formation

Vulnerable Plaque





Fibrin Formation



During coagulation, prothrombrin is converted to thrombin, which acts upon a soluble protein called fibrinogen to create *FIBRIN*, long threadlike compounds which form a mesh-like structure that traps RBCs, WBCs, and more platelets. Fibrin is the major element of a blood clot

Full occlusion of the coronary artery is rare



The *location* of the occlusion within the artery determines *how much* of the myocardium is affected

A *distal* occlusion will affect only a small area of the heart



A *proximal* occlusion will affect a *much larger* area of the heart The amount of occlusion (along with its location within the vessel) helps determine the severity of the Acute Coronary Syndrome

- A small occlusion results in Unstable Angina
- A larger occlusion may result in *Non-ST elevation MI*
- A significant occlusion may result in a *ST Elevation MI* (*STEMI*)

Rupture of a coronary plaque-Angiospasm

As the clot forms an occlusion, the vessel wall injury causes smooth muscle spasm which further narrows the vessel

Myocardial Ischemia

- When the myocardium becomes ischemic, the oxygen that *is* available is diverted into the production of energy to keep the cell alive.
- Little or no oxygen is available for the work of contraction. In cardiac ischemia, the ability of the affected ventricle to eject blood is thus impaired
- PVCs are often generated, potentially causing lethal arrhythmias

Angina Goals

- Perfusion
- Decreased workload
- Prevent infarction
- Intervene in unstable angina

Myocardial Infarction



Cardiac Preload and Afterload

Clearly, if the heart's ability to eject blood is reduced, circulation is impaired.

Other factors that may impair circulation are cardiac *preload* and cardiac *afterload*

What is *Cardiac Preload*?

Cardiac preload is simply the amount of blood that is returned to the heart after circulation through the body. How Does Cardiac Preload Affect The Left Side Of The Heart?

If the left side of the heart has an impaired stroke volume, returning preload can cause the pulmonary vasculature to become engorged, resulting in *Congestive Heart Failure*.

In this instance, it is desirable to decrease the amount of preload, so that the damaged left ventricle can adequately eject the blood it receives.

How Does Cardiac Preload Affect the <u>Right</u> Side of the Heart?

In right ventricular cardiac ischemia, a significant preload is necessary to maintain adequate cardiac output. Without adequate RV stroke volume, there is insufficient blood flow across the pulmonary vasculature to provide gas exchange, and the *left* ventricle receives an inadequate volume of blood to send out to the body.

In this situation, drugs that reduce preload (such as nitroglycerine), while still useful, must be used *carefully*!

"It is important to recognize that patients with RV dysfunction and acute infarction are very dependent on maintenance of RV filling pressures to maintain cardiac output."

International Consensus on Science, AHA

Cardiac Afterload

Simply put, cardiac afterload is the resistance the heart (and in particular, the left ventricle) must overcome to move blood around the body and back to the heart. If the pumping ability of the left ventricle cannot overcome afterload, cardiogenic shock and congestive heart failure will result.

Managing Cardiac Afterload

Vasodilation is useful in reducing cardiac afterload. Vasodilation works in two ways-

- By *reducing* the peripheral vascular resistance
- By *increasing* the vascular space (which also reduces preload)

How can we manage thrombosis, angiospasm, preload, and afterload?

By using agents that will control the size of the clot AND regulate vascular smooth

muscle activity-

Heparin and Nitroglycerine

Minimizing the size of the clot can help control the severity of the infarct

- *HEPARIN* is commonly used to inhibit clot formation, thus controlling clot size
- In low doses, heparin interferes with the ability of platelets to "stick" to each other
- In higher doses, heparin inhibits fibrin formation

HEPARIN

- <u>Class</u>- Anticoagulant
- <u>Action</u>-Interferes with platelet adhesion and conversion of fibrinogen to fibrin

Indications

- Acute Myocardial Infarction
- Pulmonary Emboli
- Disseminated Intravascular Coagulation (DIC)
- Atrial Fibrillation with Embolization
- Deep Vein Thrombosis
- Other embolic disorders

Administration

Heparin is measured in units.

- For use in ACS, an initial IV bolus of 5,000-10,000units is given
- After the initial IV bolus, a continuous IV infusion of 1,000-2,000 units/hour is common

Per CVEMSA protocols-

 Heparin IV infusions of 100 units per cc of D5W or .45NS (for example- 25,000U in 250cc D5W) must be used.

• The maximum delivery rate may be no more than 1,600 units per hour

Precautions

The most common side effect of heparin is increased bleeding. Ask the patient about any bleeding history, such as ulcers. The patient with a history of liver disease or alcoholism may be at risk.

Use with care when the patient is taking oral anticoagulants such as aspirin or coumadin

Heparin is obtained from animal products, and occasional severe allergic reaction and anaphylaxis has been reported Managing Cardiac Preload, Cardiac Afterload, and Coronary Angiospasm May Minimize Myocardial Injury

Reducing these factors can improve coronary blood flow and reduce cardiac workload, easing myocardial oxygen demand and limiting infarct size.

Nitroglycerine, a powerful vasodilator, is effective in managing these three factors.

Nitroglycerine

- Class- Vasodilator
- Action- Nitrates act directly on vascular smooth muscle, causing relaxation.

Vascular Smooth Muscle Relaxation

The peripheral vascular smooth muscle relaxation caused by nitroglycerine enlarges the average arteriolar diameter, which decreases the pressure against which the left ventricle must pump. This means *Cardiac Afterload* is reduced.

This same arteriole enlargement permits blood pooling, which reduces *Cardiac Preload*.

The smooth muscle relaxation also inhibits the coronary angiospasm seen in ACS, improving blood flow to ischemic myocardium.

Administration of Nitroglycerine

- Nitroglycerine is mixed into D5W in amounts of 25mg/250cc (100µg/cc) or 50mg/250cc (200µg/cc)
- Delivery dosage is calculated in µg/minutes. A common starting dose is 5µg/minutes, titrated in 5µg increments

CVEMSA protocols mandate a MAXIMUM rate of 50µg/minute

Considerations In Using Nitroglycerine

- Nitroglycerine is a VERY POWERFUL vasodilator. Frequent BP measurements and pump use are necessary
- Care must be used in the setting of Right Ventricular Infarct/Ischemia
- Sildenafil (Viagra) may potentiate the vasodilatory effects of nitroglycerine

Drug Calculations

Reminders

- Convert all units of measure to the same unit and system
- Assess the computed dosage to determine whether it is reasonable
- Use one method of dose calculation consistently

Administer 800µg/min Dopamine

- You have 200mg/250ml of D5W
- Convert 800 μ g to .8mg - 800 μ g ÷ 1000 = 0.8mg

Administer 1 mg/kg Lidocaine to a Patient Weighing 132 lbs.

•132 lb \div 2.2 = 60 kg

•1 mg x 60 kg =60 mg

Assessment of Computed Doses

You are to administer 8 mg of diazepam (Valium). It is supplied in a 2-mL ampule that contains 10 mg of the drug. Therefore a "*reasonable*" calculation of volume would be less than 2 ml.

Methods of Calculation

 $\frac{D}{H} \ge Q = X$ D = desired dose to be administered H = known dose on hand Q = unit of measure on hand or volume on hand X = unit of measure to be administered

Methods of Calculation

Administer 25 mg of Benadryl. You have a 50 mg vial of the drug. How many milliliters will you give?

 $\frac{25}{50} \operatorname{mg}_{mg} x \ 10 \ \mathrm{mL} = X$ $\frac{25}{5} \operatorname{mg}_{mg} x \ 1 \ \mathrm{mL} = X$

$$5 x 1 mL = X$$

$$X = 5mL$$

Calculating IV Flow Rates

gtt/min = <u>Volume to be infused x drop factor</u> Time of infusion in minutes

Administer 250 mL of normal saline over 90 minutes. Your infusion set delivers 10 gtt/mL

 $gtt/min = \frac{250mL \times 10gtt/mL}{90 minutes} = \frac{2500gtt}{90 min.}$

27.7 or 28 gtt/min

Calculating IV Infusions

gtt/min = <u>Prescribed dose x Drop Factor</u> Concentration of drug in 1 mL

Administer a procainamide infusion at 3 mg/min. You have 1 g of the drug in 250 mL of D5W. The infusion set delivers 60 gtt/mL. How many drops per minute will you deliver?

1 g x 1000 = 1000 mg $1000 \text{ mg} \div 250 \text{ mL} = 4 \text{ mg/mL}$ Calculate drops/min using the IV drip formula $gtt/min = \frac{3 \text{ mg/min x } 60 \text{ gtt/mL}}{4 \text{ mg in 1 mL}} = \frac{180}{4} = 45 \text{ gtt/min}$

Intravenous Piggy-Back

- secondary to the primary IV infusion
- hung in tandem
- infusion times of 20 or 30 minutes to 1 hour

IV Piggy-back Preparation

- Prepare the prescribed medication
- Bleed the air
- Cleanse the primary medication port
- Attach using a needless delivery system
- Tape/secure the needle
- Calculate the flow rate (secondary infusion)

IV Piggy-back Preparation

- Lower the primary infusion reservoir
- Open the piggy-back flow clamp and adjust the flow rate
- Clamp the primary infusion
- Always label the bag with the medication

Mechanical IV Pumps

- Allow more accurate delivery of medications
- Diluted in precise amounts of fluids
- Follow instructions of equipment manufacturer
- Be familiar with the device before using it

Documentation and Reporting

- Document the precise written orders from the transferring physician
- Document medication interruptions and actions taken
- Document pump malfunction that cannot be corrected, the time the medication drop was discontinued and notification times of the base hospital and transferring hosptial

Documentation and Reporting

All calls will be audited by the CVEMSA for:

•Compliance with physician orders

Regional protocols

Actions taken during emergency situations