Conquering Complications: Care of the Cardiac Surgery Patient in the Immediate Post op Period

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Many concepts apply to CMC patients!

Test blueprint

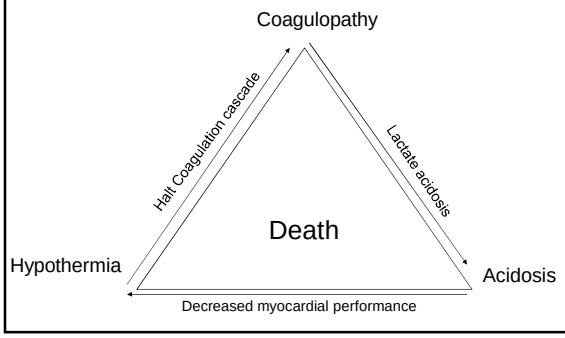
CMC

- Cardiac tamponade
- ABGs

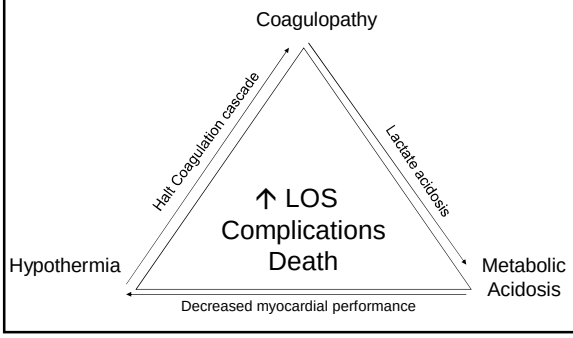
CSC

- Complications of cardiac surgery
- Tamponade
- Bleeding

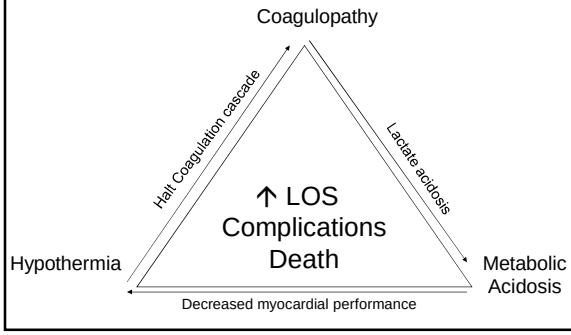
Trauma Triad of Death



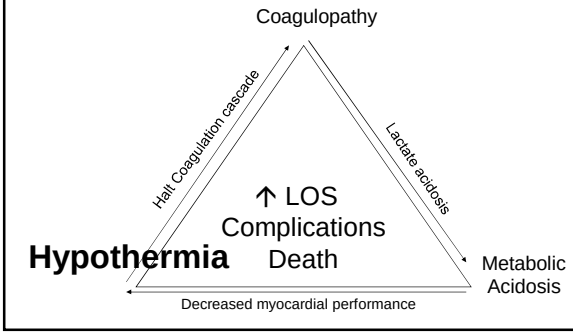
Cardiac Surgery Triad of Disaster



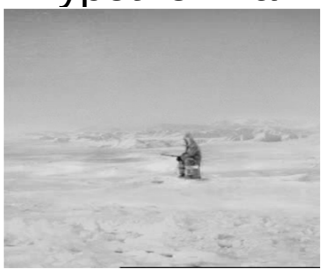
Hypoperfusion Starts the Triad of Disaster



Cardiac Surgery Triad of Disaster



Hypothermia



https://www.youtube.com/watch?v=3Wz8MNQF_F4

Causes of Heat Loss

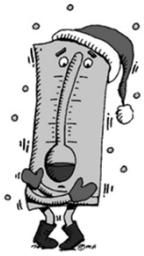
- Cooled during cardiopulmonary bypass
- Cold OR room
- Cool room and/or fan on
- Cold fluids
  - 1 unit of pRBC can lower body Temp 0.25° C
  - 1 liter of fluids unit can lower body Temp 0.5° C
- No blankets
- Head uncovered

Alarming Consequences of Hypothermia

1. Increased oxygen debt
   - Cold hemoglobin can not release oxygen to the cells
   - Left shift of the oxyhemoglobin dissociation curve
   - Prolonged ventilation
2. Increased lactic acid production
   - Change from aerobic to anaerobic metabolism
   - Leads to acidosis
3. Coagulopathy
   - Prolonged clotting cascade
   - Platelet dysfunction – platelets are extremely temperature dependent
   - Altered fibrinolytic system
4. Altered cardiovascular function
   - Decreased cardiac output/contractility
   - Risk of arrhythmias
   - Increased SVR due to vasoconstriction

Alarming Consequences of Hypothermia (cont)

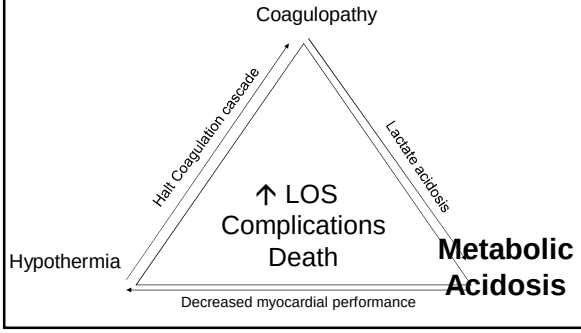
5. Hyperglycemia
   - Decrease insulin production
6. Increased Risk of Infection
   - Impairs neutrophil function
   - Tissue hypoxia from vasoconstriction
7. Altered drug metabolism
   - Delayed emergence from anesthesia
8. Shivering
   - Increases myocardial oxygen demand and consumption



Rewarming techniques

- Warm room – no fan
- Warm blankets – keep patient covered
- Bare Hugger
- Use blood warmer to give blood products
  - Have blood warmer and bare hugger in room

Cardiac Surgery Triad of Disaster



Acidosis Pathophysiology

From Allen C Wolfe Jr., MSN, RN, CFRN, CCRN, CMTE
Clinical Education Director/Clinical Specialist,Air Methods Corporation Denver, Colorado

Ph < 7.20 =decrease in cellular function causing failure to produce ATP (energy stores) to transport proteins across cells and slows or stops transmission of messages

Driving in a Snow Storm

Alarming Consequences of Acidosis

- Decreased cardiac contractility & cardiac output
- Impaired response to catecholamine
  - (ie intropes are not effective)
- Increased PVR
- Vasodilation - decreased SVR
- Bradycardia
- Increased arrhythmia risk
- Coagulopathy
- Compensatory hyperventilation.

Pathophysiology of Acidosis

Hypothermia causes peripheral vasoconstriction and impairs oxygen delivery to the tissues

Hypovolemia caused by bleeding reduces tissue perfusion

cellular metabolism changes from aerobic to anaerobic
↓
serum lactate levels rise
↓
metabolic acidosis develops

Identify Cause of Lactic Acidosis

- Type A Lactic Acidosis
- Type B Lactic Acidosis

Treat the Cause!

Metabolic Acidosis

- Type B Lactic Acidosis
  - Occurs in the absence of tissue hypoxia
  - May be catecholamine-induced metabolic effect (especially with epinephrine)
  - May be caused by hyperglycemia & alterations in fatty acid metabolism

Injury from surgery disrupts normal regulatory mechanisms

- Plasma glucose elevates during surgery
- Insulin secretion is depressed during surgery

Elevated blood levels of catabolic hormones render patient resistant to insulin.

Metabolic changes are proportional to the degree of surgical stress.

Ketones form as a result of incomplete oxidation.

- Large quantities of body fluid are needed for renal excretion of glucose and ketones.
- This may result in hypovolemia.

The inability of kidneys to remove excess glucose with hypovolemia prompts further rise in glucose.

Metabolic Acidosis
may develop within hours.

K+ increases as response to hyperglycemia.

K+ is redistributed from within the cell to the extracellular space as a result of increased plasma osmolality.

Insulin corrects the abnormality.

Date/Time	1600	1830	2030	2300	0200	0600
PH	7.47	7.32	7.27	7.19	7.28	7.38
PCO2	31	42	44	34	33	34
PO2	192	156	92	115	85	87
CO2 conc	24	24	20	14	16	21
O2 Sat	99	99	95	97	95	96
BE	-1	-4	-8	-14	-10	-5
O2 flow Rate/mode	100% IMV 10	80% IMV 10	60% IMV10	60% IMV 10	50% IMV 6	50% CPAP
K+	3.9	4.0	2.9	3.5	4.9	
Glucose				414	322	221
Treatment			Regular insulin 5 Units IV 20 SQ 2 ambs Bicarb		20 units SQ insulin	

Date/Time	2400	0600
PH	7.40	7.26
Pco2	34	46
PO2	110	91
CO2 conc	22	22
O2 Sat	98	96
BE	- 3	- 7
O2 flow	50%	50%
Rate/mode	IMV10	IMV 4
K+	5.0	5.3
Glucose		327
Treatment		

If Potassium is LOW, Be Careful when giving...

- Insulin
- Calcium
- Digoxin

Serum glucose should not decrease more than 75 - 100 ml/dl per hour to prevent…

- Hypoglycemia
- Hypokalemia
- Cerebral Edema

Severe hypoglycemia can lead to coma and death!

Admission ABGs

	Patient A	Patient B
ph	7.29	7.33
pCO_2	60	41
$p0_2$	132	100
TCO_2	31	22
O_2 %	98	98
BE	-1	-6

Interpret these

1. Pt A = Respiratory Acidosis
   Pt B = Metabolic Acidosis
2. Pt A = Metabolic Acidosis
   Pt B = Respiratory Acidosis
3. Both metabolic Acidosis
4. Both Respiratory Acidosis

Metabolic Acidosis

Ongoing Metabolic Acidosis
means something is not being perfused

- Type A Lactic Acidosis
  - Reflects impaired tissue oxygenation & anaerobic metabolism resulting from circulatory failure
  - The lactate ion more than the acidemia contributes to potential cardiovascular dysfunction

The Value of Lactate

- Serum lactate levels are used to assess the acid-base state and adequacy of tissue perfusion
- By product of anaerobic metabolism if tissue hypoxia (from hypoperfusion) exists
- A change from aerobic to anerobic metabolsim

- Lactate is primarily excreted by liver.
- Treatment: Treat the Cause
- $NaHCO_3$ (Bicarb) is truly only a "Band-Aid" and should only be used for severe metabolic acidosis = ph < 7.2 & HCO_3 < 6mqEq/L

Treat the Cause!

The Value of Lactate

Serial lactate levels predictor of perfusion

- Normal <2.5mmol/L
- Mild acidosis 2.5-4.9mmol/L **(mortality 25-35%)**
- Moderate acidosis 5.0-9.9mmol/L (**mortality 60-75%)**
- Severe acidosis > 10mmol/L **(mortality > 95%)**

Shoemaker, WC et al. Textbook of critical care. 1995. WB Saunders

Lactate Levels

"Surviving Sepsis 2014"

- Normal 1-2
  - Cells are alive & well
- Moderate 2 – 4
  - DECREASED cellular perfusion
  - Cells STRUGGLE to survive
  - May indicate severe sepsis
- Severe > 4
  - COMPLETE TISSUE HYPOXIA
  - Cells DIE
  - Hypotension refractory to adequate fluid resuscitation indicates septic shock

Surviving Sepsis Campaign 2014

Stages of Shock

- Compensated
  - Maintains end organ perfusion
  - BP is maintained by ↑ heart rate (tachycardia)
- Uncompensated
  - Decreases micro-vascular perfusion
  - S/S of end organ dysfunction
  - Hypotensive
- Irreversible
  - Progressive end organ dysfunction

Serum Lactic Acid Levels

- May be the first indication that something is wrong
- Excess lactate demonstrates measurement of tissue oxygen debt
- Results in metabolic acidosis due to tissue hypoperfusion and "starvation"

Serum Lactic Acid Levels

- Increasing lactic acid levels mean the tissues are hypoperfused and patient is getting worse.
- Decreasing lactic acid levels mean the tissues are getting perfused and the patient is getting better.

Do not draw lactate levels more than every 4 hours
Lactate level takes 4 hours to rise and 4 hours to release

Base deficit/excess

- Amount of total base (buffer) that is needed to achieve acid-base balance.
- BD/BE is depicted by $HC0_3$-Cl, phosphates, sulfates, proteins and organic acids. To figure BD/BE, lab uses the PH, PaCo2 and Hct.
- Normal -2- +2
- If < - 2, the patient is not perfusing

Base deficit

- Normal -2 to +2
- Mild Hypoperfusion: -2 to -5
- Moderate Hypoperfusion: - 6 to -14
- Severe Hypoperfusion: < - 15

Source: Davis J et al: Journal of Trauma, 1996. Davis K et al. Journal of Trauma, 2002

Which patient are you more concerned about?

	1.0600	2.0800
PH	7.37	7.35
PCO2	36	32
PO2	77	87
CO2	22	18
O2%	94	96
BE	-4	-8

Acidosis Summary

- Acidosis is the #1 negative inotrope!
- Acidosis decreases cardiac contractility!
- Treat acidosis so intropes work!
  - Direct therapy to improve oxygen delivery and reducing demand and thus decrease acid production.

Treat the Cause!

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Ask the Experts

Use of Bicarbonate in Patients With Metabolic Acidosis

Q Please elaborate on why bicarbonate is not used unless the ph is 7.2 or less. Many nurses, particularly cardiovascular nurses, have difficulty understanding this when the patient's blood pressure is low and the patient is acidotic with large negative base excesses. Please explain the physiological basis for the decision and clarify why "it doesn't work" or "it is not beneficial."

A Myra F. Ellis, RN, MSN, ACCRN-CSC, replies:

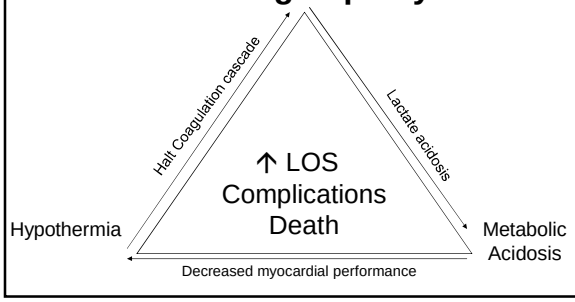
Bicarbonate therapy is sometimes prescribed to treat acute metabolic acidosis, an acid-base disorder that is characterized by a primary decrease in the concentration of bicarbonate ions (HCO_3), a compensatory decrease in the partial pressure of carbon dioxide (Pa_{CO_2}), and a decrease in serum pH.[1] Acidosis is considered severe when the pH is less than 7.20. Assuming an appropriate ventilatory response, this blood pH would be associated with a serum HCO_3 concentration of less than 10 mEq/L.[2] Two general mechanisms cause metabolic acidosis: a true HCO_3 deficit (such as renal or gastrointestinal losses) or a net addition of strong acids (such as toxins, diabetic ketoacidosis, or lactic acid production).[1,3] Bicarbonate replacement is beneficial for patients with true bicarbonate deficits, but not in patients with metabolic acidosis caused by an increase in acid production.[1,2]

Most experts currently recommend reserving bicarbonate replacement for patients with severe acidemia (pH < 7.1) and serum HCO_3 of 4 mEq/L or less because of the potential harms associated with its use.[2,3] The Surviving Sepsis Campaign recently published recommendations against the treatment of acidosis in patients with pH exceeding 7.15 even for the purpose of improving hemodynamic status.[3] Although bicarbonate may increase the pH, the available evidence supports its use only in cases of severe acidosis.

The pathologic effects of severe metabolic acidosis are well documented and affect multiple systems. Severe acidosis causes relaxation of vascular smooth muscle (vasodilatation) and vasoconstriction. Severe acidosis causes decreases in both blood pressure and cardiac output as a result of decreased myocardial contractility. In addition, acidemia decreases the binding of norepinephrine to its receptors and is associated with hyporesponsiveness to endogenous and infused catecholamines.[3,4] Patients in severe acidosis have a predisposition to cardiac arrhythmias such as ventricular fibrillation, which can contribute to sudden death.[3,4] Acidosis adversely affects cell functions such as enzymatic reactions, generation of adenosine triphosphate, fatty acid biosynthesis, and bone formation and restoration.[3] Patients are more susceptible to infection because of the suppressed immune response and leukocyte function.[3]

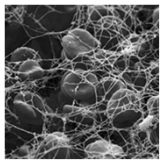
It seems logical that if acidosis causes multiple cellular dysfunctions, correcting pH with alkaline

Cardiac Surgery Triad of Disaster **Coagulopathy**



Causes of Coagulopathy

- Hypothermia
- Acidosis
- Underlying diseases
- Medications
- Dilation with fluids



Causes

- Mechanical Causes
  - Bleeding from suture lines
  - Clip comes off graft
  - Aortic or ventricular rupture
  - Chest wall bleeders
- Abnormal clotting factors due to
  - Preop anticoagulant meds
  - Systemic heparinization during CPB
  - Breakdown of factors during CPB

Signs & Symptoms

- CT bleeding > 100 – 200 cc/hr
- Low or labile B/P
- Low CVP or PAD
- Falling SvO_2 and CO/CI
- Abnormal clotting Factors
- Bleeding from line sites, incisions

Treatments

- Monitor CT output. May need to replace CT output cc for cc with packed cells
- Keep sedated and keep B/P < 140 to prevent stress on suture lines
- Keep CT patent by gently milking and stripping
- Use warming blanket to keep normal thermic.
  - Hypothermia interferes with clotting factors

Treatment: Blood and Blood Products

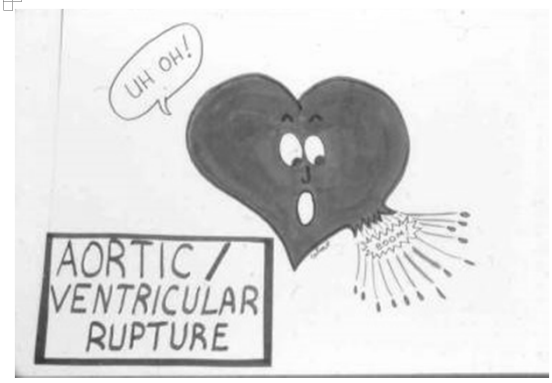
- Give blood and blood products
- FFP for ↑ PT or PTT
- Platelet Phoresis for ↓ Platelet count
- Cryoprecipitate for ↓ Fibrinogen level
- Packed cells for ↓ H & H

Rule of thumb

- Replace CT output ml for ml
- Minimum after every 4th unit pRBCs
  - Calcium Chloride
  - FFP
- Recommend 1 pRBC to 1 FFP

Treatments

- Pharmacological Interventions
  - Protamine to reverse effects of systemic heparinization
  - Aminocaproic Acid (Amicar) to inhibit conversion of plasminogen to plasmin
  - Desmopressin to improve platelet function
  - Recombinant Activated Factor VII (NovaSeven) stimulates the generation of thrombin
- May need to return to surgery to repair mechanical cause of bleeding



Keep blood on HOLD --- communicate with blood bank that you have a bleeder

- May need to use type specific blood

Massive Hemorrhage Protocol

UnityPoint Health Methodist/Proctor	Page # 1 of 7	Section: Emergency Situations	Policy #: H-14
CARE COORDINATION	Approved by CNO:		Date: 12/9/15 Review by: 12/9/16
	Approved by Pathologist: Elizabeth Bauer-Marsh MD		
	Supersedes: 9/10/13, 7/23/14 Date Revised: 12/9/15		
	Primary Responsible Parties:	Jeanette Murray Elizabeth Bauer-Marsh, MD	
	Secondary Responsible Parties:	Amy Hill	
	Joint Commission Standard:	PC	
SUBJECT:	MASSIVE HEMORRHAGE / TRANSFUSION		

I. POLICY:
In the event of a life threatening massive hemorrhage, a physician may initiate the massive hemorrhage/transfusion policy. Acute Care areas are to transfer patients to a Critical Care unit (e.g. ICU, CVICU).

II. PURPOSE AND STANDARD:
To ensure effective restoration of circulating volume (euvolemia), arrest of bleeding where there is major blood loss/massive hemorrhage, restoration of oxygen-carrying capacity (RBC's), and correction of hemostatic abnormalities/coagulopathy.

Massive Hemorrhage Protocol

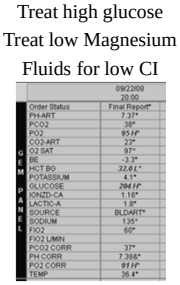
Cooler #	PRBC's	Plasma (FFP) Expire in 24 hrs	Platelets – Given per Lab results	Cryoprecipitate – per physician order – for fibrinogen <100mg/dL; 8 min. to thaw
1	4 units O neg. uncrossmatched			
2	6 units	4 units - Given after 10 units PRBC's and/or per labs	1 apheresis	
3	6 units	4 units	1 apheresis	2 units (pooled)
4	6 units	4 units	1 apheresis	2 units (pooled)

Physician can adjust orders at any time as needed

81 y/o male CABG x 3 LAD, Marginal, PDA
Admission vital signs & labs.
What are your priorities?

	Adm 2000
Art B/P	142/52
MAP	83
HR	68
Temp	97.2
PAS/PAD	32/17
CVP	14
SVO_2	69
CO	3.5
CI	1.8
SVR	1685
SpO_2	97
UO	750
CT	210

Treat high glucose
Treat low Magnesium
Fluids for low CI



Postop recovery going well. Extutated at 0015.
Now what?

	Adm 2000	2100	2200	2300	2400	0100	0200
Art B/P	142/52	148/57	106/38	151/45	161/51	142/47	138/48
MAP	83	86	62	82	94	79	79
HR	68	64	83	74	85	81	86
Temp	97.2	97.7	99	99.1	98.6	99.3	99.1
PAS/PAD	32/17	36/17	29/14	28/12	36/16	23/10	22/11
CVP	14	16	13	12	12	8	8
SVO_2	69	72	64	71	71	70	66
CO	3.5	4.4	3.3	4.5	6.1	4.5	3.8
CI	1.8	2.2	1.7	2.3	3.1	2.3	1.9
SVR	1685	1308	1186	1234			1392
SpO_2	97	100	100	97	97	98	99
UO	750	250	375	175	250	425	100
CT	210	40	60	20	30	250	290

1. Routine surgery – continue to monitor
2. Bleeding – call surgeon
3. Hypertensive – start antihypertensive

It's 0500. What are your priorities?

	2400	0100	0200	0300	0400	0500
Art B/P	161/51	142/47	138/48	111/40	113/43	91/38
MAP	94	79	79	64	71	58
HR	85	81	86	79	86	108
Temp	98.6	99.3	99.1	99.5	99.5	99.7
PAS/PAD	36/16	23/10	22/11	23/10	26/11	20/12
CVP	12	8	8	8	8	6
SVO_2	71	70	66	63	62	59
CO	6.1	4.5	3.8	3.5	4.2	3.6
CI	3.1	2.3	1.9	1.8	2.1	1.8
SVR			1392	1161		1006
SpO_2	97	98	99	100	97	98
UO	250	425	100	115	185	
CT	30	250	290	130	300	190
	200 cc IV fluid	288 cc PRBC			300cc PRBC 40 cc platelets	

It is 0700, calculate the CT output and the blood products given…. Are you keeping up with the bleeding?

Chest tube output 1660 ml
0100: 250
0200: 290
0300: 130
0400: 300
0500: 190
0600: 200
0700: 300

	2400	700
Art B/P	145/44	91/38
HR	82	108
PAS/PAD	28/12	20/10
CVP	12	6
SVO_2	71	59
CO	4.5	3.6
CI	2.3	1.8
SVR	1186	1006
Temp	98.6	97

- Pt received 3 units of pRBCs

0800 sedated to keep from bleeding. Did not return to OR

	0500	0600	0700	0800	0900	1000	1100	1200	1300	1400
Art B/P	91/38	117/40	109/42	116/47	106/43	113/46	148/73	145/54	130/58	141/56
MAP	58	75	67	71	65	69	92	83	84	84
HR	108	92	101	92	90	88	83	85	101	99
Temp	99.7	99.3	99.5	99.3	99	99.1	98.8			
PAS/PAD	20/12	37/15	43/16	32/17	31/16	67/20	51/20	414/19	33/19	41/20
CVP	6	9	8	12	11	12	13	11	10	11
SVO_2	59	56	61	66	67	65	68	70		68
CO	3.6	4.9	4.2	3.6	3.8	3.5	3.7	3.8		3.4
CI	1.8	2.5	2.1	1.8	1.9	1.8	1.9	1.9		1.7
SVR	1006		1160	1265		1370				
SpO_2	98	98	94	96	95	94	93	93	92	91
UO		75	75	105	475	375	200	425	325	150
CT	190	200	300	70	50	250	150	50	50	50
		500 cc Hespan, 300 cc PRBC, 250 cc albumin		325 cc PRBC, 195 cc platelets	325 cc PRCs, 299 cc platelets	325 cc PRBC	325 cc PRBC			

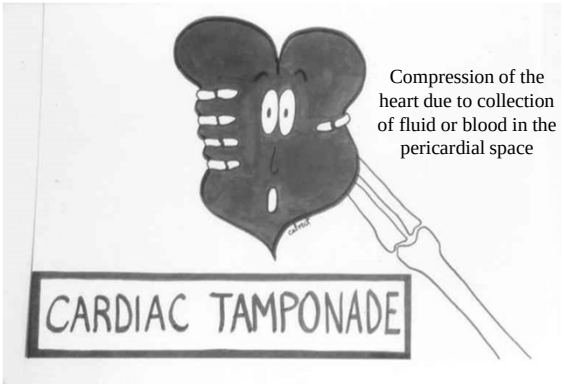
2nd Case:
Based on these coag values, what blood products do you expect to be ordered?

Hbg	7.3
HCT	21.3
Platelets	186
PT	21.7
INR	2.23
PTT	43.4
Fibrinogen	108
Lactic Acid	3.1

1. Packed cells
2. Packed cells, FFP
3. Packed cells, FFP, Cryoprecipitate
4. Packed cells, FFP, Cryoprecipitate. platelets

After 5 pRBCs, 1 platelet pheresis, 3 FFP, 10 cryo. Would you anticipate any further blood products?

Hbg	7.3	9.8
HCT	21.3	28.1
Platelets	186	115
PT	21.7	18.7
INR	2.23	1.09
PTT	43.4	32.5
Fibrinogen	108	240
Lactic Acid	3.1	2.8



Compression of the heart due to collection of fluid or blood in the pericardial space

Cardiac Tamponade: Causes

- Blood accumulated in the chest from:
  - CTs clotted off and unable to drain excess blood
  - Epicardial wire removal
- May occur quickly within minutes of hours or may occur slowly over days or weeks

Cardiac Tamponade: Signs & Symptoms

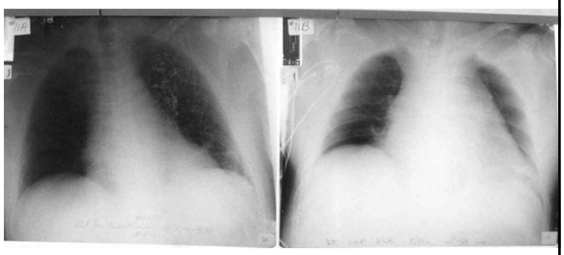
- Hypotension
- Low urine output
- Rising & equalization of CVP & PAD
- Falling SVO_2, CO/CI
- Sudden decrease in CT output
- Widening mediastinum on CXR
- Neck Vein Distention
- Tachycardia
- Pulses Paradox > 20 mmHG
- Diminished heart sounds

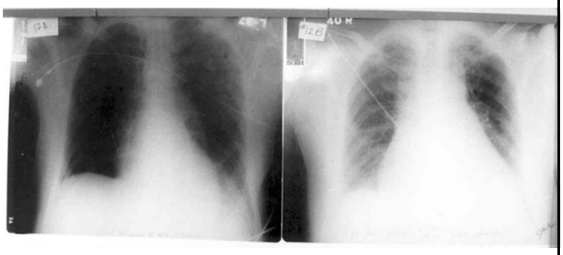
For tamponade that occurs slowly may also see these S/S:

- Shortness of Breath
- Chest Pain
- Ischemic changes on EKG
- Nausea

Cardiac Tamponade

- Beck's Triad
  - Hypotension
  - Neck vein distention
  - Muffled heart sounds





Cardiac Tamponade: Treatment

- Urgent surgical exploration to evacuate excess blood & correct cause of the tamponade
- Bedside echo may be used to make differential diagnosis between tamponade & LV failure
- Administer fluids & inotropes or Calcium Chloride until patient can be returned to OR
- Prepare for possible exploration of chest at bedside

DOS **POD #1**

	1900	2400	0500	0900	1100	1500	1900	2100
Art B/P	106/85	136/66	122/56	147/59	149/59	140/65	134/62	123/60
MAP	72	81	90	79	76	81	80	77
HR	68	99	105	106	103	111	118	118
PAS/PAD	32/15	32/18	40/21	36/21	35/21	38/23	32/18	32/21
CVP	10	11	15	14	15	16	12	15
SVO_2	63	64	65	56	55	49	43	41
CO	5.8	8.3	11	8.8	8.4	6.7	7.0	6.1
CI	2.5	3.5	4.8	3.8	3.6	2.9	3	2.8
SVR	964	674	634	559	616		743	
SpO_2	96	96	93	92	94	93	92	92
SV	85	83				62		
UO	600	325	1000	60	125	400	75	150
CT	60	100	150	50		50		75

	2200	2300
Art B/P	123/59	92/47
MAP	76	68
HR	124	125
PAS/PAD	33/22	37/26
CVP	16	19
SVO_2	40	32
CO	5.2	3.8
CI	2.2	1.6
SVR		
SpO_2	92	92
SV	42	30
UO	40	30
CT		

It's 2300, what do you want to do?

1. Fluids for hypovolemia
2. Surgery for tamponade
3. Inotropes for cardiogenic shock
4. Diuretics for fluid overload

2300 – started tamponading
Started on Dopamine 2.5 mcgkg/min
Epi 3.07 mcg/min
Milrinone 0.5 mcg/kg/min
1300 – back to OR
1600 – back to CVICU post removal of blood

POD #2

	2200	2300	0400	0800	0900	1100	1300	1600
Art B/P	123/59	92/47	129/67	141/67	108/68	101/62	118/71	126/59
MAP	76	68	88	83	78	73	80	75
HR	124	125	129	137	147	110	107	125
PAS/PAD	33/22	37/26	34/22	34/25	48/21	33/24	37/26	29/20
CVP	16	19	16	19	17	19	23	25
SVO_2	40	32	37	48	40	42	45	72
CO	5.2	3.8	5.3	6.3	6.9	3.2	4.2	7.9
CI	2.2	1.6	2.3	2.7	2.9	1.4	1.8	3.3
SVR				670		1521	1316	831
SpO_2	92	92	90	91	91	95	95	99
SV	42	30	41		42		39	63
UO	40	30	550	180		45	60	
CT						25		

TAMPONADE!

Case Study Pacemaker/ Central Line Insertion

46 y/o several hours post pacemaker insertion at outlying hospital

- c/o severe chest pain
- BP drops to 80/50, HR 108, RR 22
- **PMH:** coronary stent, MI, diabetes
  - On ASA and Effient
- What are some potential complications of pacemaker (or central line insertion) that you would suspect?

Central Line & Pacemaker Insertion

Potential Post Procedure Complications

Immediate:
- Bleeding
- Arterial puncture
- Arrhythmia
- Air Embolism
- Pneumothorax
- Hemothorax

Delayed:
- Infection
- Venous thrombosis
- Pulmonary emboli
- Catheter migration
- Catheter embolization
- Myocardial perforation
- Nerve Injury

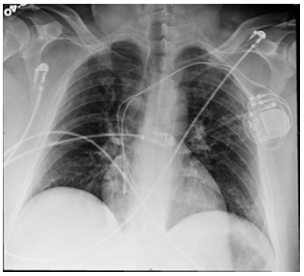
Myocardial Perforation

Symptoms:
- Signs of tamponade (heart is compressed from fluid)
  - Hypotension
  - Decreased urine output
  - Distended neck veins
  - Tachycardia
  - Dyspnea
  - Fluid noted around heart on CXR and/or echo

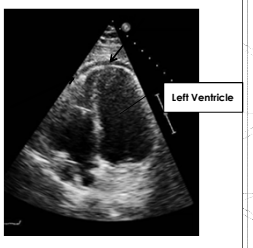
Treatment:
- Fluids to maintain hemodynamic stability
- Pericardiocentesis if hemodynamically compromised

Chest Xray Ordered

- Note water bottle shape of heart rather than the typical cardiomyopathy size
- Suspicion for pericardial effusion (fluid around heart)



- Echocardiogram reveals small amount of blood at base of heart confirming pericardial effusion from myocardial perforation



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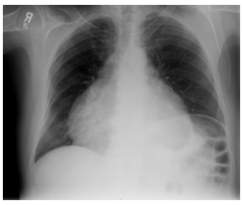
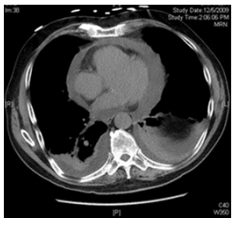
Another Patient with Large Pericardial Effusion



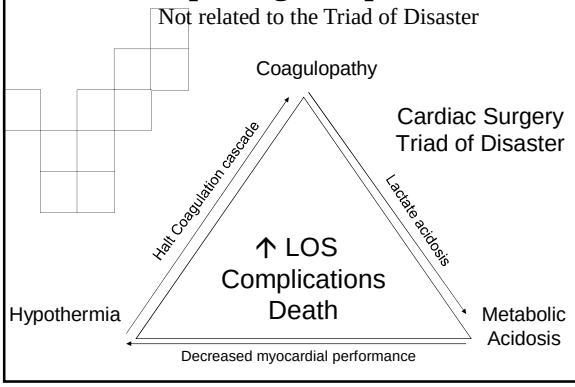


And the rest of the story…

- Patient was given fluids and monitored
- Effusion resolved on its own
- Both pacemaker and central lines can result in myocardial perforation

Conquering Complications:

Not related to the Triad of Disaster



Cardiac Vasoplegic Syndrome post Cardiac Surgery

Sara Caruso, RN, BSN, CCRN-CSC

CABG x 3, on Insulin drip, Epinephrine drip, & precedex drip

PMH: ½ ppd smoker x20 years, depression with use of SSRI's, EF = 30%, HF -- Coreg and Lisinopril

	Admission Vitals
MAP	60
CVP	3
CO/CI	4.5/2.5
SVR	1012

250ml of 5% albumin x 2 administered with no change in SVR/CVP, Neosynephrine drip is started

CABGx3, on Insulin drip, Epinephrine drip, and precedex drip

PMH: ½ ppd smoker x20 years, depression with use of SSRI's, EF = 30%, HF -- Coreg and Lisinopril

	Admission Vitals	**One Hour Later after albumin & Neosynephrine**
MAP	60	40
CVP	3	1
CO/CI	4.5/2.5	4.2/2.1
SVR	1012	770

What is Cardiac Vasoplegic Syndrome? (Vasodilatory Shock)

- Arterial vasodilatory state resistant to the usual vasopressors post cardiac surgery
- Severe and persistent form of hypotension with:
  - Normal or high cardiac output
  - Low CVP and PAOP
  - Decreased systemic vascular resistance (SVR) <800
- Low filling pressures that are poorly responsive or unresponsive to volume
- 5- 8 % of all patients post cardiac surgery
- Increased morbidity and mortality

Why does this happen?

- Huge inflammatory response post cardiac surgery -- large quantities of nitric oxide released
  - Cardiopulmonary bypass
  - Surgical trauma
  - Blood loss
  - Blood transfusions
  - Hypothermia
  - Neutralization of heparin with protamine
- Nitric Oxide produces profound vasodilation and vasoplegia

Result: Loss of vasomotor tone and vasodilation

At Risk population:

- Preoperative heart failure
  - EF < 35%
  - End stage HF requiring assist device
- Numerous preop antihypertensive medications
  - ACE inhibitors, ARBs, Beta Blockers, Calcium Channel blockers
- Use of pre and post Amiodarone and Phosphodiasterase inhibitors (Milrinone)

Treatment for Cardiac Vasoplegic Syndrome

Methylene Blue (Tetramethylthionin chloride)

- Interferes with the nitric oxide pathway and inhibits the vasorelaxant effect on smooth muscle
- Can raise mean arterial pressures while minimizing the use of vasopressors
- Usually given pre-op or inter-op to prevent CVS
- Bolus dose of 1-2mg/kg over 10-20 min followed by an infusion of 0.25mg/kg/hr for 48-72 hours, do not exceed 2mg/kg

Treatment for Cardiac Vasoplegic Syndrome

Phenylephrine, Norepinephrine or Vasopressin

- Treats refractory hypotension when used in conjunction with Methylene Blue
- Potent vasoconstrictor

Nursing Role

- Early identification of Cardiac Vasoplegic Syndrome
- Notification to cardiac surgeon
- DO NOT KEEP GIVING VOLUME!

CABGx3, on Insulin drip, Epinephrine drip, and precedex drip

PMH: ½ ppd smoker x20 years, depression with use of SSRI's, EF = 30%, HF -- Coreg and Lisinopril

	Admission Vitals	**One Hour Later after albumin & Neosynephrine**
MAP	60	40
CVP	3	1
CO/CI	4.5/2.5	4.2/2.1
SVR	1012	770

Needs Methylene Blue and Vasopressin

Walk To Recovery

♥ Chairs for Meals

♥ Scheduled Walks
1st Walk: Between 06 – 08
2nd Walk: Before Lunch
3rd Walk: Before 1330
4th Walk: Before Dinner
5th Walk: After Dinner
6th Walk: Before Bed

Case Study

You are admitting 66 y/o male after CABG x 1, AVR
PMH: Diabetes, CAD, Hyperlipidemia

	Admission 2122
pH	7.33
pCO2	50
pO2	79
TCO2	28
02 sat	95
BE	0.5
Hemoglobin	11.2
Hematocrit	36
Glucose	125
Potassium	4.8

Based on ABGs and CXR, what do you want to do?

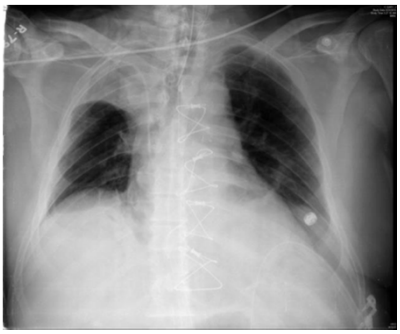
0245 – about 5 hours post op

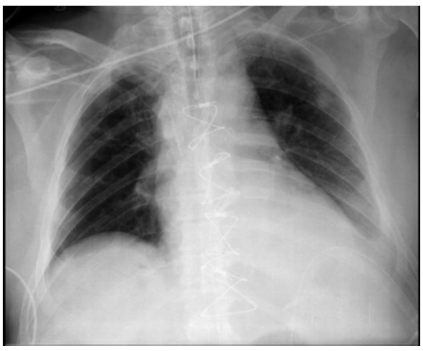
- SpO_2 drops to 90 – 91%
- BP 95/62
- HR 106
- One hour prior:
  - BP 118/81, HR 87, RR 12,
  - $Sp0_2$ 100 on 80% vent

	Admission 2122	0200	0248 Now
pH	7.33	7.47	7.49
pCO2	50	36	36
pO2	79	81	56
TCO2	28	27	29
02 sat	95	97	91
BE	0.5	2.5	4.1
Hemoglobin	11.2	11.5	10.9
Hematocrit	36	37	35
Glucose	125	147	133
Potassium	4.8	4.6	4.4

Clear bilateral lung sounds except diminished right upper lobe

CXR 0315

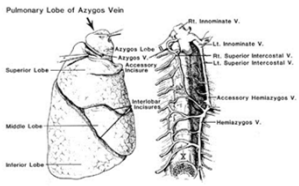




CXR at 0530 after ET tube pulled back 2 cm suctioned with mucomyst for tan secretions.

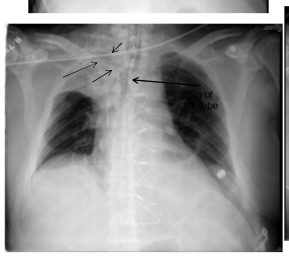
Azygos Lobe

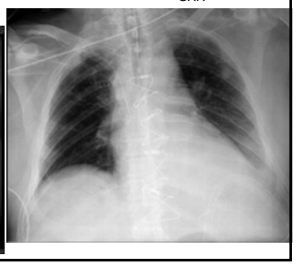
- Right upper lobe bronchus comes off trachea versus right main bronchus
- A rare congenital variation of the upper lobe of the right lung
- An anatomically separated part of the upper right lobe
- Not associated with any morbidity but can cause technical problems in thoracoscopic procedures



Admission CXR

Post suction CXR





Treatment of Cardiac Arrest

Cardiac Surgery Advanced Life Support

UnityPoint Health Peoria
Heart of IL AACN – President
www.cherylherrmann.com

Cardiac Arrest…

- In the immediate postop recovery in a cardiac surgery patient is typically related to reversible causes
  - Tamponade
  - Bleeding
  - Ventricular arrhythmias
  - Blocks associated with conduction problems
- Survival to discharge can be up to 79%
  - If treated promptly

EACTS Guidelines

European Association for Cardio-Thoracic Surgery


Guideline

Guideline for resuscitation in cardiac arrest after cardiac surgery

Joel Dunning[a], Alessandro Fabbri[b], Philippe H. Kolh[c], Adrian Levine[d], Ulf Lockowandt[e], Jonathan Mackay[f], Alain J. Pavie[g], Tim Strang[h], Michael I.M. Versteegh[i], Samer A.M. Nashef[j,*], on behalf of the EACTS Clinical Guidelines Committee

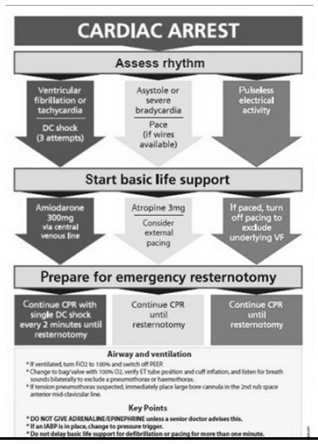
2009

Cardiac Advanced Life Support-Surgery

Key Concepts

- Assess Rhythm
- Shock before Compressions
- Pace/Atropine – not Epi
- Identify reversible causes
- Early resternotomy

CALS-S



Pulseless Electrical Activity (PEA)

- Cardiac surgery patients who arrest with PEA are typically experiencing treatable causes
  - Hypovolemia -- severe
  - Hypoxia
  - Tamponade
  - Tension pneumothorax
- Prompt treatment results in good outcomes
- To assess for causes of PEA/nonschockable rhythm
  - Consider the 4 "Hs" and 4 "Ts

Assess for Reversible Causes

Four Hs	Four Ts
Hypoxia *	Tamponade *
Hypovolemia*	Tension Pneumothorax
Hypokalemia/ Hyperkalemia	Thromboembolism
Hypothermia	Toxin

* = Most common causes of cardiac surgery arrests

Treatment of PEA causes

Hypoxia	Hypovolemia and Tamponade
• Treat per airway management and assessment	• Severe hypovolemia is typically due to bleeding • Severe hypovolemia and tamponade both require emergent resternotomy to correct

Steps to Ensure Adequate Airway and Ventilation

- Check endotracheal tube (ET) position and end tidal carbon dioxide ($EtCO_2$) waveform and reading
- Listen for an ETT airleak and verify that is properly inflated
- Listen and look for bilateral breath sounds.
  - Consider removing the patient from the ventilator and give 100% oxygen via bag-mask-valve to more easily assess lung sounds and determine lung compliance
  - If bilateral lung sounds are present, reconnect the ETT to ventilator.

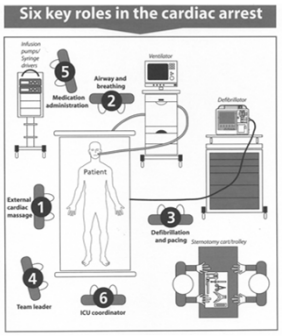
Steps to Ensure Adequate Airway and Ventilation (continued)

- Feel the trachea to verify it is midline.
- If a tension pneumothorax is suspected, insert a large bore needle into the 2nd intercostal space, mid-clavicular line.
- If unable to ventilate the patient with a bag-mask-valve, attempt to suction the ET tube.
  - If unable to pass the suction catheter, ETT occlusion or malposition should be suspected.
  - Remove the ETT and ventilate with a bag-mask-valve.

Prepare for Emergency Resternotomy

Six Key Roles

1. External cardiac massage
2. Management of airway and breathing
3. Defibrillation
4. Team leader
5. Medication administration
6. ICU nursing Coordinator



Warning Signs of Trouble

- Tachycardia
  - Persistent tachycardia is a compensatory mechanism
- Cool extremities
- Diminished peripheral pulses
- Changes in mentation
- Decreased urine output
- Hypotension

You will save more lives by being ***METICULOUS*** than by being BRILLIANT

Don't miss the obvious!