CPB FMEA # 21: Failure to prevent hypotension on CPB following phenylephrine and/or norepinephrine administration: level II.

Friends-

The FMEA this week is Level II of hypotension after the initiation of CPB and failure of phenylephrine or norepinephrine to mitigate the problem. The failure of phenylephrine or norepinephrine to resolve hypotension on CPB increases the risk to the patient. This is not as common as Level I hypotension. This again comes from material supplied to me by Eric Jenkins, CCT, CCP, FPP and Kevin Griffith, CCP from Ann Arbor. Much of this material comes directly from their fine PowerPoint presentation "CPB Disaster Management or When Things Go Wrong, Don’t Scream!" I have focused on the pre-operative use of ACE inhibitors. Although the causation of hypotension on CPB has not been conclusively linked to ACE inhibitor use, a strong association has been noticed. There has even been a stronger association when the patient is a smoker. And although protamine reactions are relatively rare, they are more common with ACE inhibitor use. I have stopped short of describing vasoplegia and the use of methylene blue. Those will come in the Level III FMEA later.

The AmSECT Safety Committee

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FAILURE MODE AND EFFECTS ANALYSIS

FMEA: Failure to prevent hypotension on CPB following phenylephrine and/or norepinephrine administration: level II

EFFECT:

1. Refractory hypotension despite adequate blood flow

2. Inadequate perfusion of vital organs

3. Temporary or permanent organ damage

4. Failure to wean from CPB.

5. Preoperative use of ACE inhibitors is associated with increased postop morbidity possibly related to hypotension during CPB.

CAUSE:

1. Bradykinin accumulation or release during CPB. A history of ACE inhibitor usage, particularly in large doses, is associated with bradykinin release during CPB.
2. Preoperative ACE inhibitor use among smokers further potentiates the kinin response to CPB and causes hypotension during CPB.

3. During CPB a significant rise in kallikrein also leads to the formation of bradykinin. Since the primary site of bradykinin breakdown is the lungs, and the bypass circuit removes the lungs from the circulation, the primary site for removal of excess bradykinin is lost leading to its general accumulation.

4. Reaction to artificial surfaces or some types of membranes; i.e. polyacrylonitrile or cuprophane membranes used in hemodialyzers or ultrafiltrators.

5. Increased bradykinin contributes to protamine associated hypotension in ACE inhibitor patients after CPB.

PRE-EMPTIVE MANAGEMENT;

There are no pre-emptive management processes for this failure. Patients taking ACE inhibitors have a higher mortality after heart surgery. However this may simply co-inside with sicker patients who need this medication. Stopping this medication prior to heart surgery is controversial and should be taken on a case-by-case basis. (\*Patients who are taking ACE inhibitors are more frequently associated with Level II hypotension, but their frequency in the population is less. Such patients would have an RPN of only 4\*4\*2\*2 = 64 as a group, but the Occurrence RPN for individual patients is higher than those not taking ACE inhibitors.)

MANAGEMENT:

1. After utilizing all management interventions for Level I hypotension consider:
2. Epinephrine bolus and/or drip (100 mcgm/1000 mls NS.
3. Arginine Vasopressin (AVP), an endogenous antidiuretic hormone essential for cardiovascular homeostasis and released during baroreflex response. It constricts vascular smooth muscle. Adult Dosage: 1 – 2 units bolus; 1 – 4 units/hr. Pediatric Dosage: 0.0003-0.002 units/kg/min.
4. Failure to wean from CPB will necessitate ventricular support for an indeterminate period.
5. If patient weans from CPB and transits to the ICU, be prepared for extracorporeal support (ECPR) should patient have sudden cardiac arrest in the post-op period.

RISK PRIORITY NUMBER (RPN):

A. Severity (Harmfulness) Rating Scale: how detrimental can the failure be:

1) Slight, 2) Low, 3) Moderate, 4) High, 5) Critical

(I would give this a 4, high.)

B. Occurrence Rating Scale: how frequently does the failure occur:

1) Remote, 2) Low, 3) Moderate, 4) Frequent, 5) Very High

(The occurrence is less frequent than Level I hypotension so the RPN would be a 3.)

C. Detection Rating Scale: how easily the potential failure can be detected before it occurs:

1) Very High, 2) High, 3) Moderate, 4) Low, 5) Uncertain

(The Detectability RPN equals 2 because it is initially more difficult to predict and detect than simple Level I hypotension as CPB progresses.)

D. Patient Frequency Scale:

1) Only a small number of patients would be susceptible to this failure, 2) Many patients but not all would be susceptible to this failure, 3) All patients would be susceptible to this failure.

(All patients are at risk. So the Patient Frequency RPN should be a 3.)

Multiply A\*B\*C\*D = RPN. The higher the RPN the more dangerous the Failure Mode.

The lowest risk would be 1\*1\*1\*1\* = 1. The highest risk would be 5\*5\*5\*3 = 375. RPNs allow the perfusionist to prioritize the risk. Resources should be used to reduce the RPNs of higher risk failures first, if possible.

(The total RPN for this failure is 4\*3\*2\*3 = 72. Patients who are taking ACE inhibitors are more frequently associated with Level II hypotension, but their frequency in the population is less. Such patients would have an RPN of only 4\*4\*2\*2 = 64.)