Primary Objective
Children with arterial to pulmonary shunts or intravascular stents are at high risk for thrombosis which is associated with increased interventions and mortality. Aspirin is recommended for thrombosis prophylaxis, but some children are at risk for aspirin non-responsiveness. This pathway will standardize the dosing of aspirin and the testing of responsiveness in high-risk children with a goal of decreasing thrombosis.

Inclusion Criteria:
- Patients in Cardiac Care Unit with:
  - Aortopulmonary shunt
  - Intracardiac stent – ductal, pulmonary vein, and atrial
  - Special consideration for new stents in patients with Glenn and Fontan physiology
  - Sano shunt stent

Exclusion Criteria:
- Outpatient and/or in observation status

Recommendations

Aspirin Mechanism of Action rectal versus oral
Aspirin irreversibly inhibits platelet function, decreasing platelet aggregation and clot formation. Aspirin’s half-life is short (20 minutes), and patients immediately begin forming new platelets that are not inhibited. Aspirin is available both enterally and rectally, though rectal formations may have decreased absorption. Aspirin is recommended for high-risk children with congenital heart disease by the American Heart Association. Rates of aspirin non-responsiveness varies between 10 and 80% depending on the study and aspirin non-responsiveness is associated with an increased risk of thrombosis. Aspirin dosing is based on formulary limitations (i.e. dividing tables and suppositories).

Our data from CHMC shows that most shunt/stent thrombosis occurs prior to the initiation of aspirin. Therefore, the first recommendation of this pathway is initiation of aspirin within the first 24 hours after shunt/stent placement. The second recommendation is for aspirin responsiveness testing in accordance with ACTION Network guidelines (unpublished consensus guidelines).

TEG/AA testing
Platelet responsiveness testing at CHMC is achieved using TEG Platelet Mapping ADP/AA. Aspirin inhibits arachidonic acid (AA) metabolism which then prevents platelet aggregation. This test measures that percent of platelets that are inhibited from aggregating through the AA pathway. Two testing points are recommended:
1. 4 hours after initiation of ASA. This allows for an assessment of peak level of inhibition.
2. Prior to the next dose of ASA to measure length of inhibition, given that children with heart disease may regenerate non-inhibited platelets quickly. The inhibition goal for both times is at least 70% inhibition at both time points based on ACTION Network guidelines.

**Bleeding Risk on Aspirin**
Children with congenital heart disease are at risk for both gastrointestinal bleeding and intracranial bleeding and this risk could be increased with more aggressive aspirin dosing, though this has not been well documented in the literature.

**Rationale**
1. Re-intervention on shunts and stents due thrombosis increases hospital length of stay, morbidity, and mortality. CHMC’s current rate of re-intervention is 20%.
2. Most patients that required re-intervention had not received aspirin, suggesting that earlier initiation could decrease the rate of thrombosis and the need for re-intervention.
3. Aspirin non-response is known to increase the risk of thrombus. Children with aspirin non-responsiveness may benefit from second line anti-platelet agents.

**Metrics**
1. Decrease reintervention due to shunt/stent thrombosis to 10% or less by January 2024. (Outcome Metric)
2. Decrease time from procedure (Operating room or cath lab) to aspirin initiation to 1 day (median) by January 2024. (Outcome Metric)
3. Increase Aspirin responsiveness testing in population to 50% by January 2024. (Process Metric)
4. Monitor for GI and intracranial bleeding. (Balancing Metric)

**Team Members**
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ASPIRIN INITIATION FOR HIGH-RISK CARDIAC PATIENTS CLINICAL PATHWAY

EXECUTIVE SUMMARY
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Evidence