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ORIGINAL ARTICLE

Prostaglandin E1: Administration implications for the care provider in the treatment of neonatal ductal dependent congenital heart disease

Katherine Taylor Hillig, MSN, NNP*

Regis University Neonatal Nurse Practitioner Graduate Program, Children's Hospital Colorado & Flight For Life Critical Care Transport, 13123 E. 16th Ave, Aurora, CO 80045, USA

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Abstract According to McCance et al. (2010), "congenital heart disease is the leading cause of death, excluding prematurity, during the first year of life. It is estimated that as many as 35% of deaths caused by congenital heart defect occur in the first year of life" (p. 1213). Prostaglandin E1 has been successfully used as the primary temporary treatment for ductal-dependent heart defects in the neonate since the mid 1970's. This drug takes effect rapidly and has a short half life so requires continuous intravenous infusion for successful treatment. Although Prostaglandin E1 does not generally interact with other drugs, it does enhance the effects of some drugs which can in turn increase the risk for adverse side effects in the neonatal population. Prostaglandin E1 does have some significant adverse effects that could be potentially dangerous for the neonate if not treated. It is imperative that the caregivers who are managing these infants are well educated on Prostaglandin E1 and particularly on its side effects.

This literature review explores Prostaglandins E1, its use and effects, side effects and prescriber considerations when managing an infant who is receiving the medication. The articles and studies retrieved during this search revealed that there is a short-coming of recent research on Prostaglandin E1 administration and dosing to support what is now seen in our local tertiary NICU's. Dosing in infants whose ductus arteriosus is open and systemic circulation is not compromised are often started on PGE1 at a much lower dose of 0.0125 mcg/kg/min. These lower initial doses appear to lower the risk of adverse effects such as apnea. There currently is no literature

* Tel.: +1 7202325584.
E-mail address: taylorkatherine@live.com.

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found in this review that supports this change in PGE1 dosing suggesting that more research is needed to support evidence-based practice.

Prostaglandin E1

This literature review will explore the use of Prostaglandin E1, PGE1, or Alprostadil: its use in the treatment of cyanotic heart defects in the neonate, the pathophysiology of ductal-dependant heart lesions, the drugs effect and mechanism of action, along with the potential adverse and side effects that may occur with administration of the drug. Most importantly, this review will explore the implications for the prescriber of the medication and management of the infant receiving PGE. Interestingly, there are not many recent studies on PGE and the appropriate dosing for infants with ductal-dependent lesions. The literature supports the current dosing which has not changed significantly in several decades supporting the need for more recent studies on the administration of PGE1.

Since the mid 1970's, studies were initiated for Prostaglandin E1being used as a successful temporary treatment of ductal-dependant congenital heart defects in the neonate. "In 1981, the Food and Drug Administration approved Alprostadil, injectable prostaglandin E_1 (PGE_1), for use in the treatment of neonates with congenital heart disease" (Chamberlin and Lozynski, 2006, p.158). PGE1 is currently the recommended temporary initial therapy for infants with isolated defects that restrict pulmonary blood flow (e.g., pulmonary stenosis, pulmonary atresia), poor arterial-venous mixing (e.g., transposition of the great arteries), and conditions that interfere with systemic circulation (e.g., interruption or coarctation of the aorta). This life saving pharmacologic intervention of intravenous PGE1 allows for the ductus arteriosus to remain patent, maintaining systemic circulation and oxygenation until the appropriate surgical intervention or cardiac catheterization can proceed. It is important to note that although the intended therapy of PGE1 is to be a short-term intervention, there are some circumstances where long-term therapy, up to several months, has been instituted when surgery must be delayed for infant growth or while waiting for a cardiac transplant (Chamberlin and Lozynski, 2006).

According to McCance et al. (2010), the ductus arterious, (DA), is a vessel where the main and left pulmonary arteries and the lesser curvature of the descending aorta intersect. During fetal circulation, the DA allows for blood to shunt away from the pulmonary artery and flow through the aorta while the fetus is dependent on the placenta for oxygenation. After birth, with the change in pressure from clamping the umbilical cord and terminating placental blood supply, the pulmonary pressure decreases and blood is no longer shunted away from the lungs via the ductus arteriosus but rather flows to the lungs where it is oxygenated and pumped to out to systemic circulation. Due to the change in pulmonary and systemic pressure, and an increase in circulating PaO_2 along with a decrease in circulating endogenous prostaglandin, the DA will constrict and eventually will close between 15 h and two weeks of life (McCance et al., 2010). In only 5—10% of term infant, the DA will remain open and will require intervention for closure (McCance et al., 2010). The closure of the DA can be a critical time for those infants that have a ductal-dependent congenital heart defect, particularly those who are not diagnosed prenatally.

According to McCance et al. (2010) ductal-dependent congenital heart defects have three sub-groups: Obstructive defects, defects decreasing pulmonary blood flow and mixing defects. Defects decreasing pulmonary flow such Tetrology of Fallot and tricuspid atresia will decrease pulmonary blood flow which will cause right to left shunting of blood and these infants will present with hypoxia and cyanosis. Obstructive defects such as coarctation of the aorta, aortic stenosis, pulmonary stenosis and hypoplastic left heart syndrome will have an anatomic narrowing in either the right or left out-flowing vessel which can be sub-valvular, valvular or supravalvular and depending on where the obstruction occurs determines the severity of the symptoms that this infant will present with. These infants may not initially present with cyanosis until the ductus arteriosus starts to close and systemic circulation is impeded. Mixing defects such as transposition of the great arteries, total anomalous pulmonary venous connection or return and truncus arteriosus are dependent on the mixing of saturated blood and desaturated blood mix within