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Cause specific management of shock in neonate

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Shock is characterized by inadequate oxygen delivery to tissues to meet demand because of circulatory failure. The immediate aim of management of neonatal shock is to optimize perfusion, delivery of oxygen and nutrients to the tissues. Understanding the pathophysiology of neonatal shock helps to recognize and classify shock in the early compensated phase and initiate appropriate treatment. Hypovolemic shock in neonate is usually due to antepartum hemorrhage, post-natal blood loss iatrogenic or secondary to disseminated intravascular coagulation or vitamin K deficiency or excessive insensible water loss in extreme pre-terms. Cardiogenic shock in the neonate may be caused by myocardial ischemia due to severe intra-partum asphyxia, arrhythmias, primary structural heart disease, mechanical reduction of cardiac function or venous return secondary to tension pneumothorax or diaphragmatic hernia and disturbance of transitional circulation due to persistent pulmonary hypertension in newborn, or patent ductus arteriosus in premature infants. Distributive shock caused by neonatal sepsis, vasodilation, myocardial depression or endothelial injury and obstructive shock is caused from tension pneumothorax or cardiac tamponade. The immediate aim of management of neonatal shock is to optimize perfusion and delivery of oxygen and nutrients to the tissues. The American College of Critical Care Medicine estimates 60 minutes is the average time needed to provide adequate circulatory support and block the development of shock. The first step in managing shock in the newborn during the first 5 minutes is to recognize cyanosis, respiratory distress and decreased perfusion. This should be followed immediately by airway access and ventilation to optimize oxygenation. Rapid peripheral, central venous or intraosseous access is of primary importance in the initial management of the newborn in shock. Any baby with shock and hepatomegaly, cyanosis or a pressure gap between upper and lower limbs should be treated with prostaglandin within 10 minutes of birth until congenital heart disease is excluded. Inotropes like dopamine, dobutamine, epinephrine and norepinephrine are indicated *via* IV or IO route before central access is achieved when myocardial contractility remains poor despite adequate volume replacement. Delay increases mortality 20-fold.

Biography

Mohammad Monir Hossain is currently working as Professor of Neonatal Medicine, NICU and Critical Care of Pediatrics at the Bangladesh Institute of Child Health (BICH) and Dhaka Shishu (Children) Hospital. He has received his PhD from the University of Dhaka for his research work on neonate receiving intensive care in 2006. After his graduation (MBBS) in 1987, he completed Doctor of Medicine in Pediatrics (MD) in 1997. He became Fellow (FCPS) of Bangladesh College of Physicians & Surgeons in 1999 and Royal College of Physicians and Surgeons of Glasgow (FRCP Glasg) in 2009, Royal College of Physicians of Edinburgh (FRCP Edin) in the same year and Royal College of Pediatric & Child Health (FRCPCH), UK in 2010. He has been serving as an Assistant Professor, Associate Professor and Professor at Bangladesh Institute of Child Health and Dhaka Shishu (Children) Hospital since 2001. He has authored several publications in various journals and books. His publications reflect his research interests in critical care in neonatology. He was the Executive Editor of *Bangladesh Journal of Child Health (BJCH)*. His interests includes neonatal shock, pulmonary hemorrhage, PPHN, septic shock and DIC and critically ill neonates receiving intensive care.

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