



CHAMPLAIN MATERNAL NEWBORN REGIONAL PROGRAM
PROGRAMME RÉGIONAL DES SOINS À LA MÈRE
ET AU NOUVEAU-NÉ DE CHAMPLAIN

Newborn Hypoglycemia

Self – Learning Module

Developed by the
Interprofessional Education and Research Committee of the
Champlain Maternal Newborn Regional Program (CMNRP)

2013



TABLE OF CONTENTS

Definition.....	3
1. Physiology.....	3
2. Causes and risk factors.....	4
2.1 Transient hypoglycemia	
2.2 Recurrent hypoglycemia	
3. Signs of hypoglycemia	6
4. Screening, monitoring, and treatment	7
4.1 Blood glucose monitoring	
4.2 Treatment strategies	
4.2.1 Enteral feedings	
4.2.2 Intravenous therapy	
4.2.3 Pharmacotherapy	
5. References.....	11
Additional Resources	12
Acknowledgements	13

Disclaimer: This self-learning module is intended for health care providers caring for term, low-risk newborns. Please refer to institutional policies and procedures.

Definition

Hypoglycemia is the most common metabolic problem seen in the neonate. The term hypoglycemia refers to a low blood glucose concentration. The definition and management of hypoglycemia remain controversial and recommendations vary as to which glucose values require treatment (Canadian Paediatric Society (CPS), 2013; Milcic & Nash, 2008). No uniform standard exists.

Hypoglycemia cannot be described by a single value because it may be different between neonates due to their physiologic immaturity and their current pathophysiology (Cornblath & Ichord, 2000). Every institution should have clearly developed policies regarding neonatal glycemic levels and therapeutic goals that direct their interventions. At present, a plasma glucose concentration of less than 2.6 mmol/L appears to be abnormal for term and preterm newborns and requires intervention (CPS, 2013).

1. Physiology

In utero, the fetus depends on the maternal supply of glucose. After birth, the neonate's glucose supply is disrupted and endogenous glucose metabolism begins. This mobilization of fuel sources is a complex balance between glucose utilization, glucose production and hormonal responses (Milcic & Nash, 2008). Mechanisms for glucose production and utilization include glycogenesis, glycogenolysis, glyconeogenesis, lipolysis, and ketogenesis. Glucose is the predominant fuel for the newborn brain. There is risk of long term neurological damage with prolonged hypoglycemia (CPS, 2013).

Hormones play a vital role in the metabolism of glucose. Insulin is the major blood glucose-lowering hormone. Glucagon, epinephrine and cortisol are the hormones that raise the blood glucose level. The release of these hormones is regulated by the pancreas, adrenal glands and brain (Milcic & Nash, 2008).

Breastfed, formula-fed, and combination-fed newborns follow the same pattern of glucose values with an initial fall in glucose over the first 2 hours of life. Newborn blood glucose levels will gradually rise over the next 96 hours, whether fed or not. Breastfed newborns tend to have slightly lower glucose and higher ketone bodies than formula-fed newborns (Academy of Breastfeeding Medicine, 2006).

2. Causes and risk factors

Several classification systems have been used to define the causes of hypoglycemia. Two basic groups have been identified: (1) conditions associated with diminished hepatic glucose production, or transient hypoglycemia, and (2) conditions associated with excessive insulin production, or recurrent hypoglycemia. Table 1 outlines risk factors that suggest the need for screening.

Table 1: Risk factors of newborn hypoglycemia

Maternal metabolic alterations	Diabetes (pregestational and gestational) Drug therapy (B-Blockers, intrapartum glucose administration) Preeclampsia
Neonatal intrinsic problems	Beta-sympathomimetic exposure Beckwith-Weidemann syndrome Hypothermia Infant of a diabetic mother (IDM) Intrauterine growth restriction (IUGR) Polycythemia Prematurity < 37 weeks Respiratory distress syndrome (RDS) Rh incompatibility Sepsis Small for gestational age (SGA) Large for gestational age (LGA) Twin gestation Perinatal hypoxia-ischaemia Newborns on parenteral nutrition History of poor feeding
Neonatal metabolic disorders	Fatty acid defects Galactosemia Glycogen storage disease Maple syrup urine disease
Neonatal endocrine disorders	Congenital glucagon deficiency Cortisol deficiency Pituitary insufficiency

(ACoRN, 2012; CPS, 2013; Cowette & Loughead, 2002; Hawdon, 2008; Milcic & Nash, 2008)



2.1. Transient hypoglycemia

Diminished hepatic glucose production which may be related to:

- Reduced glycogen stores, decreased gluconeogenesis, and ketogenesis
 - Hyperinsulinemia
 - Metabolism of brown fat
 - Depressed plasma glucose concentration
- *Maternal factors* including intrapartum administration of glucose, drug treatment (oral beta-sympathomimetic agents, propranolol) and diabetes (Cowette & Loughead, 2002; Milcic & Nash, 2008).
 - *Neonatal factors* including idiopathic conditions or adaptation failure, intrauterine growth restriction (IUGR), small for gestational age (SGA), large for gestational age (LGA), prematurity, perinatal stress/asphyxia, hypoxia, cold stress, hyperviscosity, sepsis, congenital heart disease, and congestive heart failure (CPS, 2013; Cowette & Loughead, 2002; Milcic & Nash, 2008).

2.2. Recurrent hypoglycemia

- Secondary to hyperinsulinism or excessive insulin production which may be related to:
 - Islet cell hyperplasia
 - Enzymes disorders that cause disruption in several areas of carbohydrate metabolism
 - Endocrine disorders (pituitary insufficiency and cortisol, glucagon, and epinephrine deficiencies)
 - Umbilical catheter inserted at T11 to L1 (level of the vessels supplying the pancreas or the celiac axis)
- Associated conditions such as Beckwith-Wiedemann syndrome, Rh incompatibility, inborn errors of metabolism, nesidioblastosis, islet cell adenomas, adenomatosis, adrenal insufficiency, and iatrogenic causes such as beta-sympathomimetic exposure,



exchange transfusion, and a high placement of the umbilical artery catheter (Cowette & Loughhead, 2002; Milcic & Nash, 2008).

3. Signs of hypoglycemia

Signs of hypoglycemia are often subtle or may be absent altogether and are usually neurological in nature. Clinical signs of hypoglycemia are non-specific for any particular glucose concentration (Milcic & Nash, 2008). Signs of hypoglycemia are listed in table 2.

Table 2- Signs of hypoglycemia

- Irritability, tremors or jitteriness
- Hypothermia, poor temperature control, sweating
- Poor feeding or intolerance of feedings
- Hypotonia, limpness or lethargy
- Tachypnea, grunting
- Abnormal cry (weak or high-pitched cry)
- Exaggerated Moro reflex
- Cyanosis, pallor
- Abnormal eye movements
- Apnea, irregular rapid breathing
- Seizures, coma
- Cardiac arrest or failure

Note: Often there is a cluster of these signs

(ACoRN, 2012; CPS, 2013; McGowan, Price-Douglas, & Hay, 2006; Milcic & Nash, 2008; Rozance & Hay, 2006)

4. Screening, monitoring, and treatment

Healthy term newborns with a normal perinatal course that are asymptomatic do not require monitoring of glucose levels (CPS, 2013; Deshpande & Ward Platt, 2005). Early feeding is important to prevent hypoglycemia.

4.1 Blood glucose monitoring

- Check serum glucose **at 2 hours of age** if the newborn is considered “at risk” for hypoglycaemia (refer to Table 1). Continue to monitor every 3-6 hours (before feeds) until glucose ≥ 2.6 mmol/L as long as newborn remains well.
- Check serum glucose **immediately** if a newborn is symptomatic or unwell. (ACoRN, 2012; CPS, 2013; Queensland Maternity and Neonatal Clinical Guidelines Program, 2013)

Please refer to the algorithms on the next pages for the screening and immediate management of babies at risk for neonatal hypoglycemia as per the Canadian Paediatric Society (CPS) and ACoRN. Institution specific policies or cut-offs may vary slightly. Close communication with interdisciplinary team members is necessary. Refer to institutional policies regarding when to notify the most responsible healthcare provider (e.g. physician, nurse practitioner, midwife).

Blood glucose concentrations show a cyclic response to an enteral feed, reaching a peak by about an hour after the feed, and the lowest blood glucose level (nadir) just before the next feed is due. Since the purpose of blood glucose monitoring is to identify the lowest blood glucose level, it is most logical to undertake such measurement immediately before a feed (pre-feed) rather than at arbitrary intervals at random intervals between feeds (Deshpande & Ward Platt, 2005).

Canadian Paediatric Society Screening Algorithm

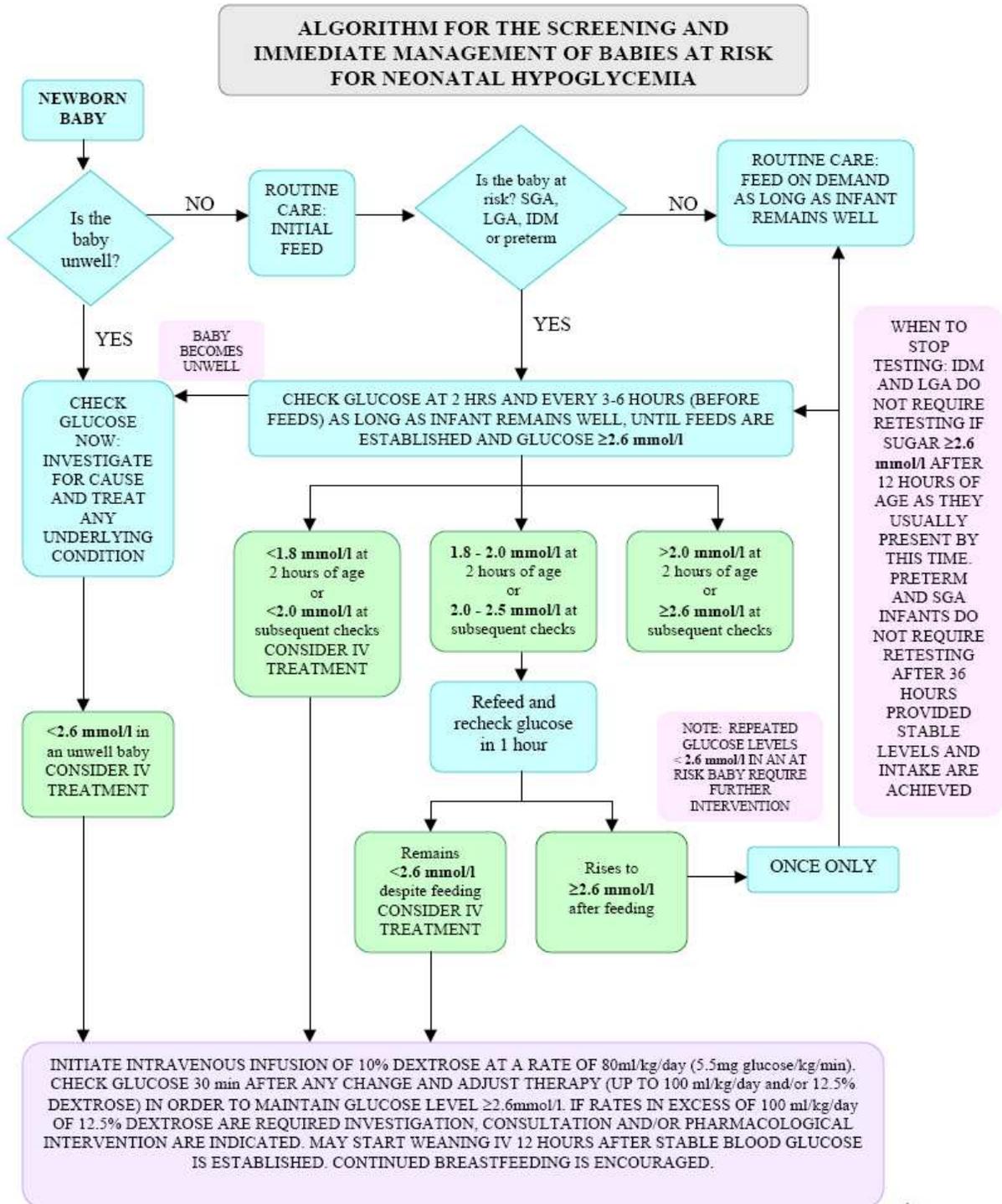


Figure 1) Screening for neonatal hypoglycemia. IDM Infant of diabetic mother; IV Intravenous; LGA Large-for-gestational-age; SGA Small-for-gestational-age. CPS Statement: www.cps.ca/english/statements/FN/fn04-01.htm

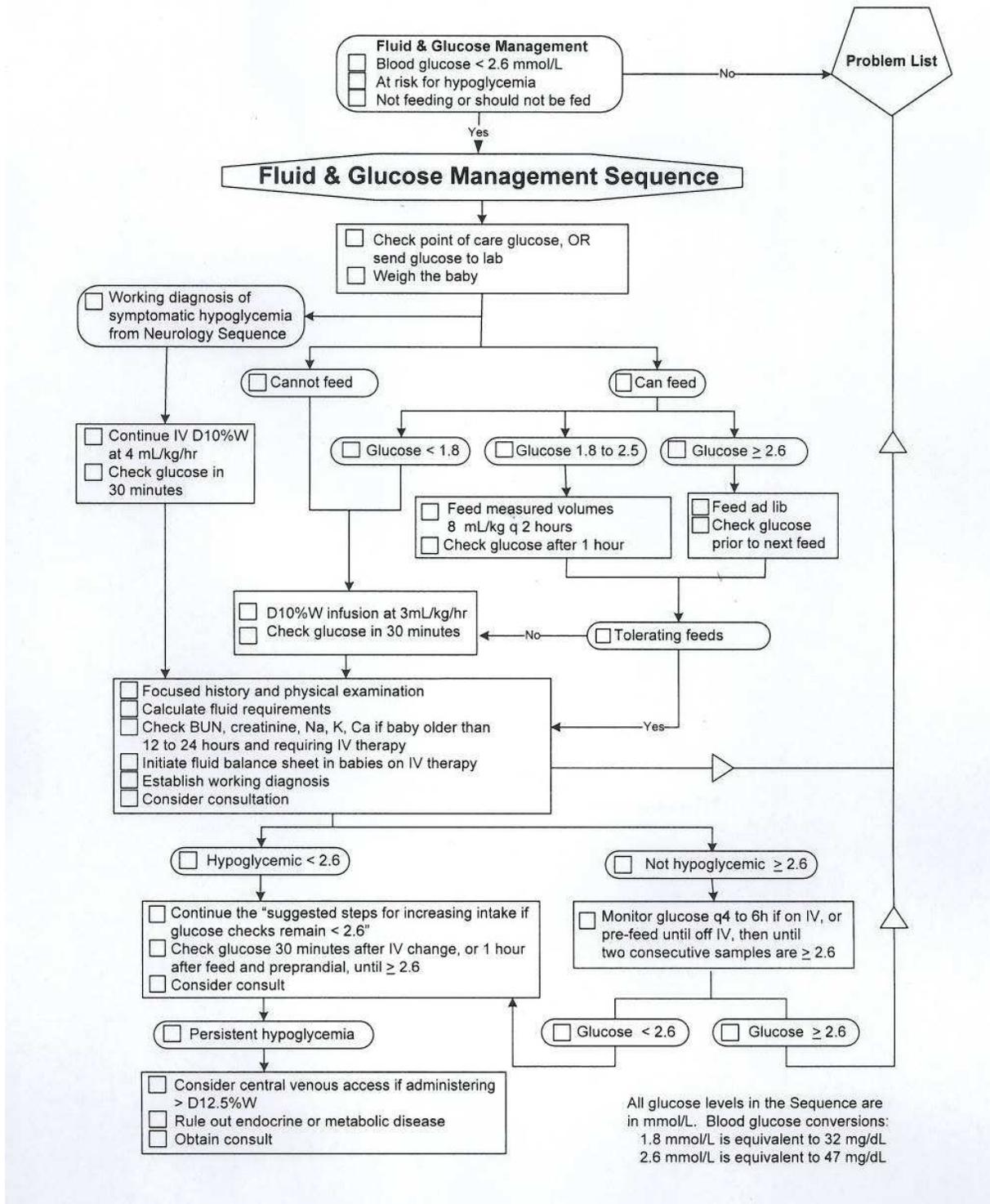


Aziz, Dancey & Canadian Paediatric Society, Fetus and Newborn Committee (2004, reaffirmed 2013), p. 726.



ACoRN Sequence for Hypoglycemia Screening and Management

The Fluid & Glucose Management Sequence



ACoRN Editorial Board, (2012), p. 7-7.

4.2 Treatment strategies

Some strategies can be beneficial in preventing hypoglycemia. One such example would be skin-to-skin contact. Skin-to-skin contact may prevent decreased glucose levels through the promotion of thermoregulation and the establishment of breastfeeding (Provincial Council for Maternal and Child Health, 2012). However, when faced with an at-risk or symptomatic newborn, treatment strategies for hypoglycemia aim to normalize blood glucose levels. These strategies include enteral feedings, especially more frequent breastfeeding, intravenous (IV) glucose infusion, and pharmacotherapy. The baby's condition will help determine which of these strategies are most appropriate (see page 8 and 9 for CPS and ACoRN algorithms).

4.2.1 Enteral feedings

If breastfeeding, manual expression of breast milk and spoon feeding can ensure milk transfer as colostrum is thick and may be difficult for the newborn to extract. Many times, the initiation of early enteral feedings to asymptomatic newborns can prevent or treat hypoglycemia. Note: Parental consent may be required if breast milk substitutes are needed. Refer to institutional policies and procedures.

4.2.2 Intravenous Therapy

Intravenous therapy is indicated when glucose levels remain persistently low despite adequate enteral feeding, if the newborn is too ill to eat, or becomes symptomatic. Glucose infusion rates should be titrated to maintain a blood glucose concentration of ≥ 2.6 mmol/L. As feedings are introduced, the IV glucose infusion should be decreased slowly to avoid fluctuation in glucose delivery (Deshpande & Ward Platt, 2005).

4.2.3 Pharmacotherapy

In some cases, newborns may require agents other than IV glucose infusions to treat their hypoglycemia for example, corticosteroids, or glucagon (Queensland Maternity and Neonatal Clinical Guidelines Program, 2013).

5. References

- ACoRN Editorial Board. (2012). *Acute care of at-risk newborns: A resource and learning tool for health care professionals (2012 update)*. Vancouver, BC: ACoRN Neonatal Society.
- Aziz, K., Dancey, P., & Canadian Paediatric Society, Fetus and Newborn Committee (CPS, 2004, reaffirmed 2013). Position Statement: Screening guidelines for newborns at risk for low blood glucose. *Paediatrics & Child Health*, 9(10), 723-729. Retrieved from <http://www.cps.ca/en/documents/position/newborns-low-blood-glucose>
- Cornblath, M., & Ichord, R. (2000). Hypoglycemia in the neonate. *Seminars in Perinatology*, 24, 136-149.
- Cowette, R. M., & Loughead, J. L. (2002). Neonatal glucose metabolism: Differential diagnoses, evaluation, and treatment of hypoglycemia. *Neonatal Network*, 21(4), 9-19.
- Deshpande, S., & Ward Platt, M. (2005). The investigation and management of neonatal hypoglycaemia. *Seminars in Fetal & Neonatal Medicine*, 10, 351-361.
- Hawdon, J. M. (2008). Investigation and management of impaired metabolic adaptation presenting as neonatal hypoglycaemia. *Paediatrics and Child Health*, 18(4), 161-165.
- McGowan, J. E., Price-Douglas, W., & Hay, W. W. (2006). Glucose homeostasis. In G. B. Merenstein & S. L. Gardner (Eds.), *Handbook of neonatal intensive care* (6th ed.) (pp. 368-390). St-Louis, MI: Mosby.
- Milicic, T. L., & Nash, P. (2008). Neonatal glucose homeostasis. *Neonatal Network*, 27(3), 203-207.
- Provincial Council for Maternal and Child Health. Maternal-Newborn Advisory Committee-Mother Baby Dyad Care Work Group. (2012). *Promotion of mother-baby dyad care*. Retrieved from <http://www.pcmch.on.ca/ClinicalPracticeGuidelines/MotherBabyDyadCare.aspx>
- Queensland Maternity and Neonatal Clinical Guidelines Program. (2013). Queensland Maternity and Neonatal Clinical Guideline: Newborn hypoglycaemia. Retrieved from http://www.health.qld.gov.au/qcg/documents/g_hypogly5-0.pdf
- Rozance, P. J., & Hay, W. W. (2006). Hypoglycaemia in newborn infants: Features associated with adverse outcomes. *Biology of the Neonate*, 90, 87-88.
- Wight, N., Marinelli, K. A., & Academy of Breastfeeding Medicine Protocol Committee. (2006). ABM Clinical Protocol #1: Guidelines for glucose monitoring and treatment of hypoglycemia in breastfed neonates. *Breastfeeding Medicine*, 1(3), 178-184. Retrieved from <http://www.bfmed.org/Media/Files/Protocols/hypoglycemia.pdf>



Additional Resources

American Academy of Pediatrics, Committee on Fetus and Newborn. (2011). Clinical report: Postnatal glucose homeostasis in late-preterm and term infants. *Pediatrics*, 127(3). Retrieved from <http://pediatrics.aappublications.org/content/127/3/575.full.pdf>

Canadian Paediatric Society (CPS) - www.cps.ca

Champlain Maternal Newborn Regional Program (CMNRP) – www.cmnrp.ca

Evans, R. J., Evans, M. K., Brown, Y. M. R., & Orshan, S. A. (2010). *Canadian Maternity, Newborn, & Women's Health Nursing*. (1st Ed.). Philadelphia, PA: Lippincott.

Gosmanov, A., Gosmanov, N., & Gerich, J. (2011). *Glucagon Physiology*. Retrieved from <http://www.endotext.org/diabetes/diabetes2/payment.html>

Kattwinkel, J. (2011). *Textbook of neonatal resuscitation* (6th Ed.). Elk Grove, IL: American Academy of Pediatrics and American Heart Association.

Simpson, K. R., & Creehan, P. A. (2008). *AWHONN's Perinatal Nursing* (3rd Ed.). Philadelphia, PA: Lippincott.

Tappero, E. P., & Honeyfield, M. E. (2009). *Physical Assessment of the Newborn: A Comprehensive Approach to the Art of Physical Examination*. (4th ed.). Santa Rosa, CA: NICU INK.



Acknowledgements

The Champlain Maternal Newborn Regional Program (CMNRP) would like to thank the members of the **Joint Orientation Sub-Committee** for their work on the development of this *Newborn Hypoglycemia Self-Learning Module*.

CMNRP also acknowledges the work of the following groups and health care professionals who have provided feedback and expertise:

- Members of the Interprofessional Education & Research Committee (IERC)
- Pediatricians, Neonatologists, Family Physicians, and Midwives
- CMNRP Perinatal Consultants and Neonatal Nurse Practitioners
- Managers, educators, registered nurses, and registered practical nurses from partner organizations
- Maternal-Newborn Nursing Professors (College and University)