Equine Neonatal Medicine

Edited by

David M. Wong

Pamela A. Wilkins

WILEY Blackwell

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Book Dedication

This book is dedicated to my wife, Kristine, and daughters, Olivia, Pip, and Estelle, and their steadfast support of me over the years as an equine veterinarian; to Pam Wilkins, my emergency and critical care mentor and friend; to all my colleagues at the Lloyd Veterinary Medical Center (Iowa State University), Marion duPont Scott Equine Medical Center (Virginia Tech), and New Bolton Center (University of Pennsylvania) – it has been a pleasure to work together through so many foaling seasons with you; and to all the mares and foals that we have had the honor to provide veterinary care to.

David M. Wong

I would like to dedicate this book to those who came before us, the editors of the original Equine Clinical Neonatology book, Anne Koterba, Willa Drummond, and Philip Kosch, and to all our patients and dedicated veterinarians caring for them over the generations since.

Pam A. Wilkins

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Foreword

Anne Koterba, DVM, PhD

It has been quite a few years since Willa Drummond, Philip Kosch, and I published *Equine Clinical Neonatology*—a few decades, in fact! Back then, few hospitals or clinics had space or personnel dedicated to the care of critically ill equine neonates, and there was little advanced training available for clinicians interested in caring for these small patients. But horse owners increasingly demanded improved care and outcomes given economic factors and other considerations, such as the advances already made in human neonatology. When the book was published in 1990, it represented over 10 years of intensive clinical and basic research on the equine neonate. Our "green book" was designed to share what we had learned so that others—including clinicians, students, veterinary nurses, and horse owners—could provide better veterinary care for compromised neonatal foals.

Looking back at that time, I feel our productivity during that decade in terms of advancing the field of equine neonatology was the result of the collaboration of a large team that worked amazingly well together because we were all focused on the goal of improving outcomes for sick foals. These partners included equine organizations that provided important research funding, as well as veterinary clinicians, residents, and basic scientists who worked together in both university and private practice settings to better understand the physiology, pharmacology, and disorders of the equine neonate. A large group of medical specialists, including human neonatologists, respiratory and physical therapists, and nutritionists, volunteered their time and provided important advice and perspectives on accurate identification of disease and treatment of neonates. Veterinary technicians, veterinary students, horse lovers, and community volunteers formed "foal teams" and provided essential around-the-clock intensive nursing care. I still feel very fortunate to have been a part of this innovative collaborative effort.

At the time, our book reflected the state of the art in equine neonatal intensive care. But in the years since, the number of veterinarians, students, and nurses caring for equine neonates has increased greatly. These individuals have expanded our knowledge of the topics we covered in our original book, and this increased understanding of the adaptive physiology from fetus through neonates—along with significant advances in diagnostics, treatments, and nursing care—has greatly improved our neonatal patients' outcomes. Much of this work has contributed to the material covered by this new book.

I want to thank and congratulate David and Pam for taking our concept of the original book and running with it, updating older information and introducing new knowledge. There are now many hospitals and clinicians with space, equipment, and personnel both interested in and capable of delivering high-quality care to equine neonates. This book, like our original, is for the dedicated veterinarians, interested students, and nurses who support them and deliver this care.

Preface

In the year 1990, Drs. Anne Koterba, Willa Drumond, and Philip Kosch edited and published the first and only edition of *Equine Clinical Neonatology*. Many veterinarians refer to this small little green book as *the* reference text for equine neonatal care and without a doubt, over the years, this book has served as an invaluable reference and resource for countless clinicians in their quest to provide veterinary care to neonatal foals. Over the past 30 years, veterinarians and researchers have greatly advanced our understanding of physiology, disease processes, and therapeutic options as it pertains to equine neonatal medicine and critical care. Although many aspects of the foal remain to be studied, the goal of this book is to relay new discoveries and provide clear and comprehensive information on disease processes and their treatment in neonatal foals. This information on equine neonatal care is targeted to veterinary students, practicing veterinarians, and equine veterinary specialists so that we can better serve our patients and their owners.

This textbook is authored by experts in the field of equine perinatology and is organized by body systems; within each system, subtopics such as embryology and anatomy, clinical physiology, diagnostic procedures, congenital disorders, and specific disease processes and treatment are discussed. Furthermore, one section is dedicated to general treatment principles for the equine neonate.

We are incredibly grateful for the expertise and generosity shared by the various contributors to this textbook. Their willingness to spare their time in authoring these chapters and share their individual depth of knowledge and clinical experience form the foundation of knowledge for this textbook and allows us to expand on the information provided by the original neonatal textbook published by Drs. Koterba, Drumond, and Kosch. It is our sincere hope that this text, *Equine Neonatal Medicine*, will prove to be a valuable resource to all those involved in the care of the neonatal foal.

David M. Wong
Pamela A. Wilkins

Part I

The Newborn Foal

Chapter 1 Postpartum Adaptation of the Newborn Foal

Section I Fetal Heart Rate and Fetal ECG

David Wong

Throughout the course of gestation, the maternal and fetal heart rates (FHR) are monitored as a general reflection of health. In the healthy mare, the mean maternal heart rate slowly increases, from 150 days gestation to term [1]. Maternal heart rate in late gestation is relatively constant during the last 15 days of pregnancy with a mean ± SD heart rate of 53 ± 1 beats/min prior to parturition. After parturition, heart rate significantly decreases to 42 ± 1 beats/min (Figure 1.I.1) [2]. The fetal heart is not as easily monitored as the mare but can be measured using fetal electrocardiography (ECG) or ultrasound. Determining the FHR via ECG is a simple parameter used to monitor fetal well-being in the pregnant mare, but this technique has been largely supplanted by determination of FHR through transabdominal ultrasonographic visualization of the fetal heart beat and rate. Acquisition and interpretation of the fetal ECG can be hampered by the larger amplitude of the maternal ECG signal, movement of the mare, small amplitude of the fetal ECG signal, and fetal position. Despite these limitations, most clinicians familiar with fetal ECGs have little difficulty acquiring cardiac activity of the equine fetus [3]. In addition, fetal ECG can be used in a continuous fashion via telemetry, allowing detection of fetal arrhythmias as well as more prolonged monitoring of FHR trends, while the mare is maintained in a quiet stall environment. Fetal ECG can also be used through stage I of labor.

Acquisition of a maternal ECG is initiated first, using a base-apex lead configuration, to differentiate the maternal heart rate and rhythm and confirm the maternal cardiac activity as compared to the fetal cardiac activity obtained via fetal ECG lead placement. A guide to electrode placement for fetal ECG includes the following, keeping in mind that electrode placement may have to be modified based on fetal position: *left arm lead* on the dorsal midline of the mare in the mid-lumbar region; *right arm lead* on the ventral midline 10–15 cm in front of udder; and *neutral lead* anywhere on the trunk of the horse (i.e. left croup) [3]. In late-pregnant mares, the right arm lead may have to be positioned on the right ventral abdomen at a level approximately at the height of the stifle [3]. Electrodes with alligator clips can be used, but electrodes with adhesive pads, reinforced with rapid-drying adhesive glue, can be used for longer or repeated use and is better tolerated by the mare. Conventional and telemetric methods can be used to acquire the ECG tracing. If conventional methods are used, recordings are performed over 10- to 20-minute periods, several times a day. Telemetry allows for continuous monitoring, with paper tracings recorded at approximately 2-hour intervals [4]. Both the maternal and fetal cardiac activity will appear blunted as compared to a maternal base apex lead (Figure 1.I.2) and the tracing can be lost in artifact or background interference. The maximum amplitude of the fetal ECG ranges from 0.05 to 0.1 mV [4].

Volumes of information are available to the obstetrician in regard to monitoring fetal health and viability in human fetuses. In comparison, a trivial amount of published information is available to the equine clinician. However, some of the pertinent information in regard to the fetal response to hypoxia and fetal ECG changes associated with fetal distress and hypoxia in the human fetus can be extrapolated to the equine species, with the disclosed fact that there may be unknown differences between species. Of note, much of the published information in reference to the human fetus's response to hypoxia has been established via experiments performed in fetal sheep.

The clinician should recognize that transient accelerations and decelerations in FHR are present in the healthy fetus. In fact, the presence of intermittent FHR accelerations associated with fetal movement is believed to suggest adequate oxygenation to maintain normal fetal autonomic nervous system function [5]. However, persistent tachycardia or bradycardia can signal fetal distress. One must first recognize that despite the fact that the fetus has twice the

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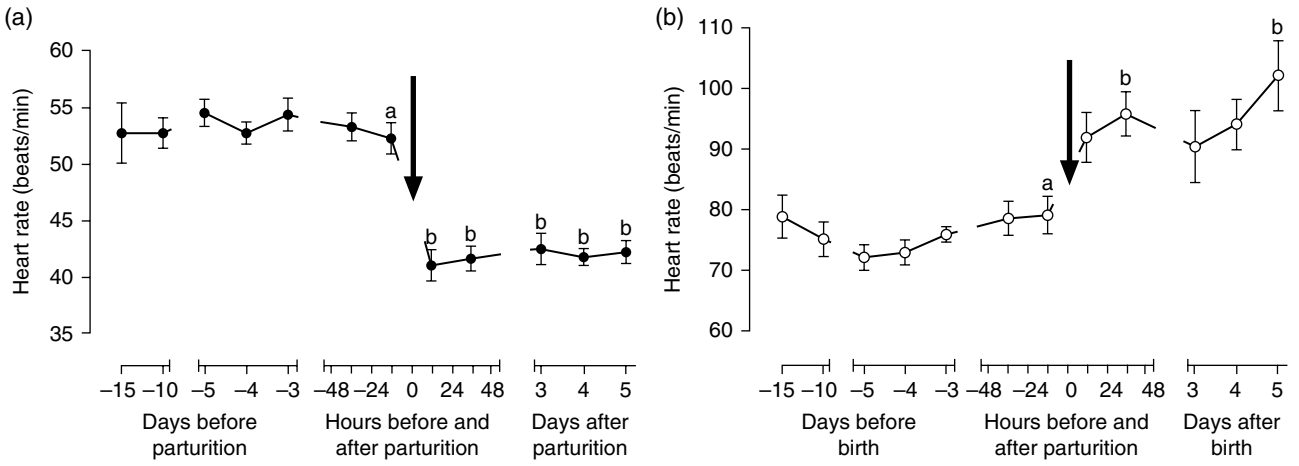

Figure 1.I.1 Maternal (a), fetal and newborn (b) peri-parturient heart rates in the horse. Arrow indicates time of parturition [2].

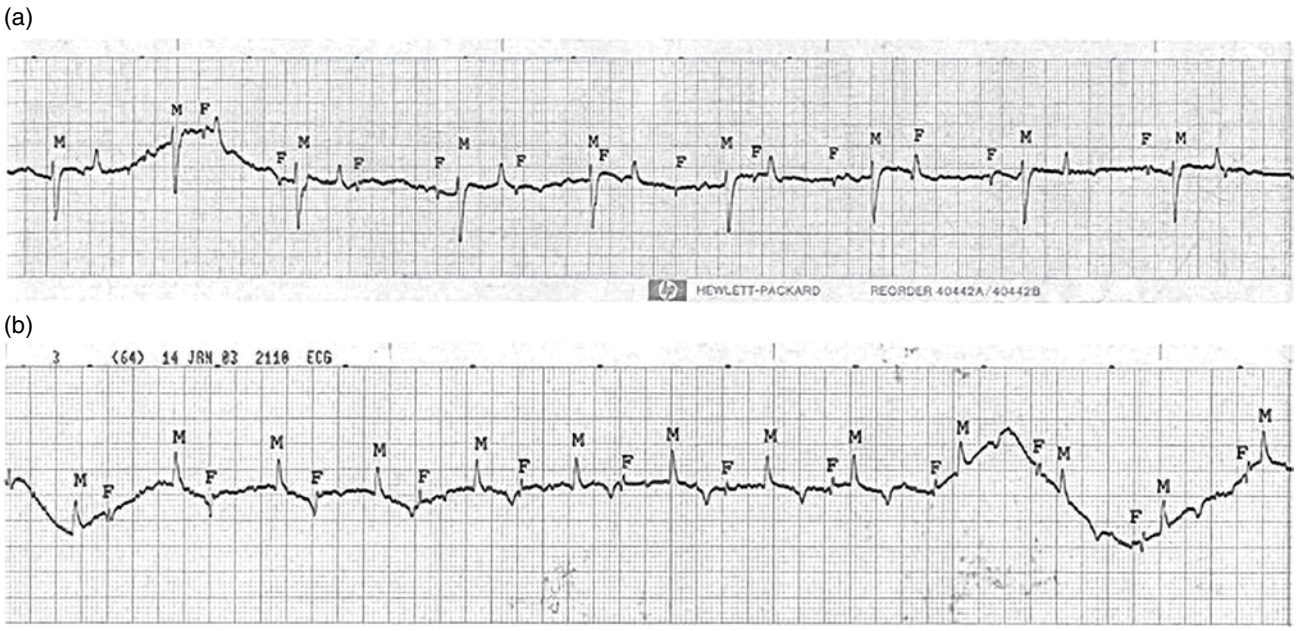

Figure 1.I.2 Fetal and maternal ECG (a). Maternal "M" and fetal "F" signal. Fetal and maternal rates are 48 and 83 beats/min, respectively. In (b), fetal and maternal heart rates are more similar, emphasizing need for careful inspection [4].

oxygen demand as the adult, and yet far lower partial pressures of oxygen and hemoglobin saturations, the fetus has a remarkable ability to compensate for sub-normal provision of oxygen that is necessary to maintain health in the adult [6–8]. In reality, under normal conditions, the fetus has a surplus of oxygen available and is able to survive profound hypoxia for extraordinary periods of time, often without injury [6–8]. This is possible because of the combination of notable fetal anaerobic tolerance and the fetal capacity to establish a coordinated cardiovascular and metabolic defense to hypoxia [8–10]. During episodes of acute hypoxia, peripheral chemoreceptors are stimulated, resulting in a coordinated cardiovascular response designed to sustain perfusion [6–8]. This chemoreflex response balances the severity of the hypoxic insult with the cellular tolerance of the individual [11]. In addition, once initiated, the response is titrated to the hypoxic insult, producing varied responses that are manifested clinically as variable decelerations in FHR [12].

The initial response in the near-term fetus to acute hypoxic challenge is mediated by parasympathetic pathways that result in rapid deceleration in FHR, with the degree of deceleration broadly related to the severity of hypoxia [13–15]. Shallow decelerations in FHR are associated with modest reduction in uteroplacental flow whereas more profound decelerations in FHR suggest near total or

total reduction in uteroplacental flow [15, 16]. Fetal bradycardia in response to hypoxic challenge reduces cardiac workload and is likely a defense mechanism present to preserve cardiac glycogen and reduce cardiac stress [17]. Slowing of the FHR may also allow increased exposure of the fetal blood to maternal blood, thus increasing the time for equilibration of dissolved gas from the placenta and improving oxygen content of fetal blood [4, 18, 19]. Regardless of the reduced FHR and concomitant decrease in cardiac output, the fetus maintains blood pressure and normal oxygen delivery to vital organs during moderate hypoxia by peripheral vasoconstriction [6–8, 11, 20]. If mild to moderate hypoxia is sustained, bradycardia is followed by tachycardia, a response that is mediated by increases in circulating catecholamines [21]. Tachycardia allows for ventricular output to contribute to maintaining of blood pressure while allowing peripheral vasoconstriction to abate, consequently permitting more perfusion to peripheral organs to occur [7]. This increase in heart rate does not occur if the hypoxic insult is more severe, in which case bradycardia becomes progressively more profound and is likely related to direct effects of hypoxia on the heart itself [11]. Interestingly, in fetal sheep, age-related differences in the FHR response to hypoxia were observed in that a similar hypoxic challenge that elicited bradycardia followed by tachycardia in near-term fetuses was not observed earlier in gestation; this lack of response might be related to immaturity of neurohormonal regulators and chemoreceptor function [7, 22, 23].

In horses, transcutaneous fetal ECG can be used to evaluate fetal well-being and viability in pregnant mares from approximately 150 days of gestation to parturition. As a general trend, the FHR decreases gradually from 150 days gestation to term (Table 1.I.1). Sporadic episodes of tachycardia are typically observed throughout gestation in the healthy equine fetus and may be associated with fetal movements (Table 1.I.1). Other underlying causes of fetal tachycardia include maternal medications, maternal medical disorders, obstetric bleeding, and fetal tachyarrhythmia [5]. In contrast, fetal bradycardia is not typically observed during normal gestation and, in people, has been associated with maternal hypotension, umbilical cord prolapse, rapid fetal descent, excessive frequent uterine contractions and uterine rupture or fetal congenital heart defects [1, 5]. Thus, substantial and/or prolonged increases or decreases in fetal heart can indicate fetal distress.

The definition of fetal bradycardia and tachycardia remains obscure in the horse with normal FHR ranging from 65 to 115 beats/min in the last months of gestation [4]. During the last weeks of gestation, the baseline FHR ranges from 60 to 75 beats/min with the lower range being 40 to 75 beats/min. In one study, the lowest measured FHR was <70 beats/min in 80% of fetuses evaluated, <60 beats/min in 55%, and <50 beats/min in 14%. In contrast, the highest FHR measured were in the range of 83–250 beats/min, with 86% of fetuses having a FHR >100 beats/min, >120 beats/min in 50% and >200 beats/min in 20% [24]. Thus, if the FHR is <60 or >120 beats/min over a prolonged period of time, repeated evaluation is indicated. One source suggests persistent fetal tachycardia as a heart rate over 200 beats/min at 120–220 days gestation and greater than 110 beats/min from 280 days gestation to parturition; bradycardia is defined as a FHR <60 beats/min at any stage of gestation [25].

Persistent fetal bradycardia usually coincides with late gestation asphyxia, whereas persistent tachycardia may represent early effects of hypoxia, maternal pyrexia, or a prolonged acceleration pattern in a healthy fetus [26]. The fetus is dependent on the placenta for oxygen supply, and a reduction in fetal cardiac activity is the only means to reduce cardiac oxygen consumption. Thus, the primary response to fetal hypoxia is a reduction in heart rate and absence of episodic heart rate increases. Subsequently, persistent tachycardia followed by bradycardia and cardiac

Table 1.I.1 Heart rate, beat-to-beat (RR) interval, and number and duration of accelerations and decelerations in heart rate acquired via equine fetal ECG evaluation at various stages of gestation [3].

Gestational age (days)	170–180	181–220	221–240	280	320	340	1 day prior to parturition
Heart rate (beats/min)	126 ± 2	117 ± 4	101 ± 5	105 ± 4	83 ± 3	79 ± 3	79 ± 1
R-R interval (ms)	481 ± 10	518 ± 15	601 ± 30	578 ± 20	762 ± 41	772 ± 34	764 ± 12
Heart rate accelerations							
Number (number/h)	23 ± 7				22 ± 2		25 ± 3
Duration (heartbeats)	29 ± 11				42 ± 6		35 ± 5
Hear rate decelerations							
Number (number/h)	23 ± 6				27 ± 3		30 ± 3
Duration (heartbeats)	31 ± 12				32 ± 5		23 ± 2

arrest occur as fetal decompensation occurs through the loss of central nervous system control mechanisms [3]. In one report, fetal bradycardia was recorded in an equine fetus 48 hours prior to abortion and reached a nadir of 38 beats/min at 10 minutes prior to abortion [1].

During the last 10–14 days prior to parturition, FHR remains fairly stable, although one report noted increases in FHR 5 days prior to parturition to 5 days after birth [2, 3]. Similar to other studies, the mean ± SD FHR within 24 hours of parturition was 79 ± 3 beats/min and was significantly lower than the heart rate at 48 (96 ± 3 beats/min) and 120 hours of age (102 ± 6 beats/min) in healthy neonatal foals [2]. Unfortunately, FHR and R-R interval do not allow prediction of impending parturition in the horse [3]. In a review of FHR during stage I of labor, a decrease in FHR was observed prior to rupture of the chorioallantois [27]. After rupture of the chorioallantois, 82% (37/45 fetal ECGs) of fetuses demonstrated a gradual decrease or maintenance of the same FHR, whereas 18% had an increase in FHR during stage II [1, 3, 27, 28]. Decelerations in FHR have been associated with brief periods of decreased uteroplacental blood flow during uterine contractions associated with labor in people [5]; however, this has not been directly examined in the horse. Additionally, fetal arrhythmias were detected during stage I of labor in a small number (4/39 fetuses, 10%) of fetal ECGs and included sinus arrhythmia and atrial premature contraction. One fetus with atrial premature contractions was later observed to have atrial fibrillation as a neonatal foal; however, fetal ECG could not be used to predict the presence of neonatal arrhythmias.

Beat-to-beat (R-R interval) variability is another parameter that can be measured when evaluating the fetal ECG. The R-R interval ranges from 0.5 to 4 mm in the equine fetus, with most in the range of 1 mm [4]. The R-R interval variability arises from an intact fetal central nervous system and appropriate functioning of the parasympathetic and sympathetic nervous systems. If there is an absence of variation in R-R interval, loss of the function of the fetal CNS may be present and indicates close observation. Of note, the R-R interval should be measured when the FHR is not accelerating or decelerating and the clinician should be cognizant of the fact that maternal drugs (i.e. sedation) can affect the RR interval [4].

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Section II Fetal Circulation and Cardiorespiratory Transition

Cristobal Navas de Solis

Fetal Circulation

Fetal circulation is notably different from what is present in the neonate and adult horse in order to maximize efficiency of blood flow and oxygen delivery during fetal development. Understanding fetal circulation is important to comprehending hemodynamics in utero as well as congenital diseases, but for the clinician, this knowledge is also important in the evaluation of severely ill or premature foals that fail to make the normal transition to extrauterine life. In the fetus, vascular shunts such as the ductus venosus and foramen ovale exists to deliver oxygenated blood from the placenta to the left atrium, while another shunt brings deoxygenated blood to the descending aorta (ductus arteriosus). The critical characteristic of fetal circulation is that gas exchange occurs at the placental interface and not the fetal lungs [1]. Compared to other veterinary species, the difference in partial pressure of oxygen (PO_2) between the uterine vein and umbilical vein is low (0–4 mmHg), resulting in a high umbilical vein oxygen content (48–54 mmHg) [2, 3]. Likewise, the gradients for carbon dioxide (PCO_2) are very low (0–1 mmHg) in the horse, reflecting the greater diffusability of CO_2 compared to O_2 [3]. Umbilical vein oxygenation in the equine fetus is more sensitive to changes in maternal arterial pressure of oxygen (P_aO_2) than in other species. This has been postulated to account for the frequent incidence of perinatal asphyxia in the foal. On the other hand, when maternal P_aO_2 is increased with inhaled oxygen, oxygen concentration in the umbilical vein increases more than in the uterine vein; this characteristic can be used therapeutically in fetal foals with suspected hypoxemia [2].

In other species, a portion of oxygenated blood in the umbilical vein travels to the right atrium through the ductus venosus and caudal vena cava allowing approximately 50% of blood in the umbilical vein to bypass the liver [1]. One theory in regard to the purpose of the ductus venosus is to direct highly oxygenated blood derived from the ductus venosus in such a way that it passes preferentially through the foramen ovale, therefore allowing distribution to the upper body (heart and brain) [4]. A unique feature of the equine fetus is that this species lacks the ductus venosus [3, 5]. The cause for this is uncertain, but one theory put forth is that equine fetal arterial blood has relatively high oxygen tensions and that preferential distribution of umbilical blood contributing to enhanced supply of oxygen to the heart and brain is not necessary [4]. Regardless of the reason, oxygenated blood is preferentially shunted to the left atrium via the valve of the foramen ovale (septum primum). Blood is shunted as higher pressure in the right atrium (due to high volume venous return) keeps the valve over the foramen ovale open, thus allowing blood to flow from right to left atrium [1]. A small amount of blood from the pulmonary veins joins the blood shunted through the foramen ovale; the blood then enters the left ventricle and is ejected and into the aorta to the systemic circulation [6].

Deoxygenated blood that returns to the right atrium from the systemic circulation flows through the tricuspid valve into the right ventricle and is ejected into the pulmonary artery. Due to the high pulmonary vascular resistance of the fetal lungs, most of the right ventricular output is shunted into the descending aorta through the ductus arteriosus. A small part (10–25%) perfuses the lungs to support metabolic needs. The ductus arteriosus connects the pulmonary artery and aorta at the level of the origin of the left subclavian artery and the pulmonary artery bifurcation. For this reason, the brain, heart, and cranial portion of the body receive blood with higher oxygen content during fetal life. For this same reason, selective cyanosis of the caudal extremities is seen in neonates with a patent ductus arteriosus (PDA) and Eisenmenger syndrome. In this situation, the left-to-right shunt causes pulmonary hypertension as blood travels through the ductus arteriosus, eventually reversing the blood flow right-to-left, thereby resulting in

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