

Chapter 20 Anesthesia and Analgesia for Foals

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Foals are born after a gestation period of approximately 11 months (335 to 342 days)¹ and birth takes place quickly, consistent with the status of a horse as a prey and flight animal. Unlike many other species, the foal is developmentally much more mature at the time of birth, reaching the status of a juvenile physiologically within 6 to 8 weeks of life.² Based on physiological parameters, anesthesiologists may classify foals from birth to 1 month of age as neonates and as pediatric and then juvenile animals when they are 1 to 3 and 3 to 4 months old, respectively. They may be treated anesthesiologically like young adults when they have acquired mature cardiopulmonary function and metabolic pathways at 4 to 5 months of age.^{2,3}

Foals may require deep sedation; local, regional, or general anesthesia; and analgesia care for a variety of reasons, most commonly for abdominal, urogenital, traumatic, orthopedic, endoscopic, and diagnostic imaging procedures. In 1995, the overall perioperative mortality rate for equine patients under 1 year of age was reported as high as 1.9%, which was higher than the rate reported for the general horse population.⁴ However, recent data indicate that the perianesthetic mortality rate can be reduced to 0.2% or less, similar to that reported in adult horses,⁵ provided anesthetic techniques and analgesic regimens applied are tailored to the developmental stage and the specific needs of the individual foal.

Physiological and Pharmacological Considerations As They Relate to Anesthesia in the Neonatal and Maturing Foal

In the first days and weeks of life, the newborn foal undergoes major physiological changes that will affect almost all organ systems and functions, including circulation, respiration, oxygen (O₂) and nutrient delivery and consumption, central and peripheral neuronal activity, cell and organ metabolism, thermoregulation, and immune system activity. Administering safe anesthesia in the foal requires a thorough understanding of those changes, which are summarized in [Table 20-1](#).

Cardiovascular System

Transition from Fetal to Neonatal Circulation

In mammals, the most dramatic change in cardiovascular function occurs at birth with the transition from fetal to neonatal circulation.⁶ The primary function of the circulatory system of both the fetus and newborn is to deliver O₂ and nutrients to metabolizing organs and return deoxygenated blood to the gas exchange organ to replenish the O₂ and eliminate waste products including carbon dioxide (CO₂). In the fetus, the gas exchange organ is the placenta, and its vascular connections are in a parallel arrangement with the other systemic organs, remote from the pulmonary circulation. To supply deoxygenated blood to the placenta and return oxygenated blood to systemic organs, a series of extracardiac shunts (ductus venosus, patent ductus arteriosus) and an intracardiac communication (foramen ovale) are necessary. At birth, the function of gas exchange is transferred from the placenta to the lungs, and therefore from the systemic circulation to the pulmonary circulation. The venous and arterial circulations are now separated, and not only are the fetal shunts unnecessary, but their persistence may compromise circulatory functions. Therefore, transition from fetal to neonatal circulation includes elimination of the placental circulation; lung expansion and increase in pulmonary blood flow; and closure of the foramen ovale,

right-to-left shunting may continue and murmurs consistent with a patent ductus arteriosus may be auscultated in normal foals during the first 3 to 5 days of life,⁷ with partial reopening possible up to the moment of complete fibrosis of those pathways, which occurs within 2 to 3 weeks.⁸ As part of the transition from fetal to neonatal circulation, the left ventricular wall increases in thickness in parallel with a rise in systemic vascular resistance, reflecting the shift from the physiological right ventricular hypertrophy during fetal life to the physiological left ventricular hypertrophy in postnatal life.⁹ An understanding of fetal hemodynamics and the acute and chronic changes that occur with transition to the newborn circulation are important for the care of normal newborns and are crucial to the recognition and management of a newborn with significant congenital heart disease or transient hemodynamic changes that may occur during general anesthesia and trigger a reversal to conditions of fetal circulation.

TABLE 20-1 Most Relevant Aspects of Foal Physiology that Affect Anesthetic Management

System	Neonate (1 Month or Younger)	Pediatric/Juvenile Foal (1-4 Months)
Cardiovascular	Transition from fetal to neonatal circulation	
	Risk of return to fetal circulation	More SV-, less HR-dependent cardiac output
	HR-, not SV-dependent cardiac output	Increasing systemic vascular resistance
	Low systemic vascular resistance	
Respiratory	Maturation of pulmonary microanatomy, neuromuscular control, compliance, surfactant production	Respiratory function
	High RR-dependent V_{\min} , low V_T	Higher V_{\min} and RR with normal V_T
	High O_2 consumption but low PaO_2	Close to normal PaO_2
Nervous	Immature central, autonomic, and peripheral nervous system function	Matured central, autonomic, and peripheral nervous system function
	Higher BBB permeability	Close to adult BBB permeability
Metabolism and tissue composition	High ECF compartment, CBV, CPV	Higher ECF but close to adult CBV and CPV
	Low glycogen reserves	Larger glycogen reserves
	No fiber intake	Increasingly more fiber intake
	High body surface area (heat loss)	

System	Neonate (1 Month or Younger)	Pediatric/Juvenile Foal (1-4 Months)
Hepatic	Maturing liver function in first 3-4 wks	Overall close to mature
Renal	Immature	Overall mature
	Reduced concentrating ability	
Hematology and biochemistry	Physiologic anemia	Normalizing PCV
	Gradual increase in WBC	Adult WBC
	Elevated serum enzyme activities	Elevated serum enzyme activities

BBB, Blood-brain barrier; *CBV*, circulating blood volume; *CPV*, circulating plasma volume; *ECF*, extracellular fluid volume; *HR*, heart rate; *PaO₂*, arterial oxygen tension; *PCV*, packed cell volume; *RR*, respiratory rate; *SV*, stroke volume; *V_{min}*, minute ventilation volume; *V_T*, tidal volume; *WBC*, total white blood cell count.

Hemodynamic Function

Cardiac output (CO) is defined as the amount of blood ejected by the heart per minute and is calculated as the product of heart rate (beats per minute) and stroke volume (mL). It is the most appropriate index of overall cardiovascular function and, when normalized to body weight, is referred to as cardiac index (CI, mL/min/kg). Fullfilling the needs of metabolically highly active organs and tissues during the early postnatal life, CI in resting foals up to 2 to 3 months of age is markedly higher when compared to adults and primarily rate-dependent (Table 20-2).¹⁰⁻¹⁴ If CO is adjusted for metabolic size (0.75/kg),¹¹ the average CI in foals is approximately twice that of adults but the average stroke volume index 30% less.^{7,15} Therefore, the normal heart rate of a resting equine neonate is significantly higher to maintain higher CO (see Table 20-2).^{16,17} It is in this early period of life that any drug with heart rate-decreasing properties like α_2 -adrenoceptor agonists may compromise hemodynamic function to an extent that the neonate cannot tolerate.^{3,7} From 4 months of age onward, heart rates reach close to adult values and remain relatively stable throughout the remainder of the first year.¹⁸

Mean systemic arterial blood pressure is substantially lower in the early days of life but pulse pressure amplitude is higher in the neonate compared to the adult owing to a lower vasomotor tone and hence systemic vascular resistance (see Table 20-2).^{7,10, 11, 13, 14} By 1 month of age, foals tend to have a lower CI and heart rate (see Table 20-2) but a larger stroke volume, and their mean arterial pressure increases during this period because of a marked increase in vascular resistance indicative of the maturing sympathetic branch of the autonomic nervous system.⁷

Respiratory System

It is pertinent for every anesthetist to appreciate that any impairment of respiratory function, whether caused by sedative, analgesic or anesthetic drugs, recumbency and positioning, or surgical/diagnostic interventions, may severely compromise vital functions of the newborn. At birth, neither neuromuscular control of ventilation nor the lung itself is fully developed in foals.^{7,15, 19-21} Pony lungs are microanatomically more mature at birth than horses' lungs,²¹ but still sufficient surfactant production is lacking and gas exchange occurs across terminal air spaces and more primitive alveoli.⁸ Compliance of the chest wall is large in the neonate but lung elasticity is decreased.⁸ Therefore, functional residual capacity (FRC), which is the gas volume left in the lung after a normal expiration, and tidal volumes are markedly smaller than in the adult (see Table 20-2). Thus, in the immediate postnatal period foals are hypoxemic, with PaO₂ values being significantly lower than during adult life, whereas PaCO₂ values being similar. Still, because O₂ needs of the rapidly developing organism are much higher than in the adult, especially in the first week postpartum, O₂ consumption (6-8 mL/kg/min) exceeds that of the adult horse by two- to threefold,¹⁵ requiring increased respiratory minute ventilation. To compensate for the smaller FRC and tidal volume, newborn foals typically breathe up to 60 to 80 times per minute, which in the fourth to sixth week declines to 30 to 40 breaths per minute for the remainder of the first 3 months of life before gradually approaching adult values. In addition, neonates close the upper airway during end expiration and therefore do not allow the lung to collapse easily; however, this protective mechanism (often referred to as "auto-PEEP") is often lost during anesthesia. This in conjunction with a lower sensitivity of the respiratory center to changes in PaO₂ and PaCO₂, most prominent after sedation with α₂-adenergic drugs, which particularly predisposes neonatal foals to hypoxemia and hypercarbia.

TABLE 20-2 Hemodynamic, Respiratory, and Acid-Base Parameters in Normal Awake Foals Compared to Adults*

Parameter	Age				
	1-3 days	1 week	2 weeks	4-6 weeks	Adult
HR (beats/min)	118 ± 10	110 ± 30	103 ± 21	84 ± 11	39 ± 4
SAP (mm Hg)	137 ± 31	—	—	—	142 ± 12
DAP (mm Hg)	62 ± 7	—	—	—	99 ± 11
MAP (mm Hg)	87 ± 10	100 ± 20	100 ± 11	115 ± 14	114 ± 11
CI (mL/kg/min)	271 ± 3	225 ± 56	229 ± 74	167 ± 40	69 ± 17
RR (breaths/min)	44 ± 19	42 ± 11	38 ± 11	36 ± 9	16 ± 6
V _T (mL/kg)	6 ± 0.5	8 ± 1.2	14 ± 2	13 ± 2	14 ± 2

There are numerous age-dependent changes in the autonomic nervous system responsiveness of myocardial contractile and conducting tissue and vasomotor tone reported in laboratory animal species,²⁶ but little is known in the horse. Those studies suggest that at birth parasympathetic nervous activity dominates while sympathetic innervation of heart and vasculature is still immature, which may in part explain the low systemic vascular resistance and mean systemic blood pressure as well as higher rate of bradyarrhythmias observed in the newborn foal subject to hypoxemia and/or hypothermia.

Body Metabolism, Biotransformation, and Excretion

Body Water Content and Body Tissue Composition

Unlike in neonates of most other species, in the newborn foal total body water content is around 72%²⁷ or 74.4% \pm 2.4%²⁸ of total body mass and hence is relatively low compared to puppies and kittens; and it does not change much over the first 5 months of life.²⁹ It also is close to the 67% of body weight measured in the adult horse.³⁰ The extracellular fluid (ECF) compartment is on a per kilograms of body weight basis about one third larger in foals than in adults, as are the blood and plasma compartments (Table 20-3),^{27,31, 32} which must be accounted for during perianesthetic fluid therapy and intravascular volume substitution. The higher ECF volume implies a larger apparent volume of distribution for many drugs, which must be taken into account for appropriate drug dosing and for predicting of drug uptake and distribution in the body.²⁵ Furthermore, because of the presumably higher capillary permeability in the neonate yet increasing systemic arterial blood pressures postpartum, intravascular water rapidly redistributes into the interstitial space, where it accumulates.³³ As a result, no sustained increase in intravascular volume occurs, which in the adult animal triggers diuresis by modulating release of vasopressin, renin, and atrial natriuretic peptide.³³ Consequently, neonates, especially ill neonates, retain administered fluid over a much longer time and thus do not handle large fluid loads well. At the same time, the expanded interstitial space in the neonate serves as a reservoir for fluid and can be rapidly mobilized in situations of acute hemorrhage or hypovolemia, restoring total blood volume much faster than in an adult.³³ As a result, the neonate can tolerate a greater blood loss before any significant decrease in blood pressure and tissue hypoperfusion is noted.

Glycogen reserves in liver and muscle are smaller in the newborn foal than in neonates of other species and last only for a few hours, making the foal more susceptible to hypoglycemia and energy deficits if the foal does not nurse.²⁷ However, from 2 to 4 weeks of age onward the foal's diet changes gradually to solid food with high-quality grains and forage increasingly covering the foal's dietary requirements, and by 4 months of age the mare's

Thermoregulation

Rectal temperature of foals ranges from 37.2 to 38.6° C (99 to 101.5° F). The much higher ratio of body surface area to weight, thin skin, and scarce subcutaneous fat tissue (poor insulation) increase environmental heat loss in the neonate compared to the adult horse.^{8,37} Conduction, convection, radiation, and evaporation all play a role and can expose the newborn to rapid heat loss. In addition, mature equine neonates have the ability to generate heat through shivering, but they can respond with non-shivering (cellular) thermogenesis and behavioral actions

as well.³⁸ Anesthetic drugs and commonly used sedatives will interfere with thermoregulation and therefore promote extended periods of hypothermia.

Hepatic Function and Development

The liver is the principal site of drug metabolism. The microsomal cytochrome P450 enzyme system is primarily responsible for transforming lipophilic compounds to polar and pharmacologically less-active or inactive substances (phase I reactions), whereas glucuronidation and other conjugation processes (phase II reactions) render the metabolites more hydrophilic, facilitating renal elimination. Functional maturity of the liver is incomplete at birth and thus the capacity to metabolize endogenous substances such as bilirubin or drugs is markedly lower in newborn foals than in the adult horse.^{17,25} As a result, metabolism and half-lives of organic waste products (e.g., bilirubin) are expected to be prolonged causing higher plasma concentrations to persist in the newborn foal (see Table 20-3). Likewise drugs have longer plasma half-lives and may accumulate on repeated dosing, thereby extending effects and slowing elimination from the body.³⁹ As blood flow to the liver increases after birth, enzyme induction begins with exposure to various endogenous and exogenous substances. In the horse, metabolic pathways seem to mature more rapidly than in other species. In particular, microsomal enzyme activity increases rapidly during the first 3 to 4 weeks of life, while conjugation processes approach activity levels similar to those measured in the adult more gradually.^{25,39} Nevertheless, by 6 to 12 weeks postpartum most hepatic metabolic pathways are completely functioning.

Renal Function and Development

In horses, renal development, in terms of glomerular number, is complete by 30 to 40 weeks of gestation, although the kidney volume continues to grow until 50 to 90 weeks of postnatal life.²¹ As a result, on a per kilogram of body weight basis, glomerular filtration rate and effective renal plasma flow of the full-term newborn foal is already comparable with that of the adult.^{40,41} Foals have a relatively greater renal tubular internal surface area available for reabsorption but reduced renal concentrating ability in the postpartum period as compared to adult animals. Normal urine output in neonatal foals is reported to be approximately 6 mL/kg/hr but then decreases gradually over the subsequent 12 weeks of life.^{21,32} Reflecting a high water intake and urine excretion, normal urine specific gravity in newborn foals, after the first 24 hours of life, is usually hyposthenuric (1.008 or less) and is reported to range from 1.001 to 1.027.^{32,42} When compared with values reported in adults, excretion,

Preanesthetic Examination and Preparation

A thorough history and physical examination of the foal in the presence of the mare, involving assessment of mental status and temperament, cardiopulmonary functions (heart rate and rhythm, pulse pressure, capillary refill time, mucous membrane color and moisture, respiratory rate and rhythm), hydration status, and body temperature are essential before any suitable protocol for sedation, anesthesia, and/or analgesia can be formulated. The need for ancillary tests (e.g., chest radiographs, ultrasound, electrocardiogram) and laboratory analyses (e.g., complete blood cell count, clinical chemistry profile, blood gas analysis, urinalysis) is largely dependent on the physical status of the foal, the presenting complaint, and the intended surgical or diagnostic procedure and should take into account age-dependent differences in vital, hematologic, and biochemical parameters between foals and adult horses (see [Tables 20-1](#) and [20-2](#)). As a minimum, packed cell volume, white blood cell count and differential, total plasma protein content, and blood urea and glucose concentrations should be determined in any foal undergoing prolonged sedation or general anesthesia. If the foal is a newborn, the assessment should include a detailed history of the perinatal period and a test of the adequacy of passive antibody transfer; if the foal is more mature, a complete medical history may be all that is necessary.¹⁷

Nursing foals up to 2 months of age have little fiber intake and should not be muzzled prior to anesthesia but should have free access to their mother. Suckling helps maintain adequate blood glucose levels, liver glycogen reserves, and hydration status. Older foals with increased solid food intake may be muzzled and held off feed for 3 to 6 hours prior to anesthesia. These older foals, particularly when hypovolemic, may profit from antiulcer medication (ranitidine [Zantac] 1.5 mg/kg IV every 8 hr, famotidine [Pepcid AC] 0.3 mg/kg IV every 12 h, omeprazol [GastroGard] 2 to 4 mg/kg PO every 24 h).⁴⁷ In foals of any age, the mouth should be rinsed out with water close to the time of induction of anesthesia to prevent feed or bedding material that may be present in the pharynx from being pushed into the airway during the process of endotracheal intubation.

In preparation for long-term sedation or general anesthesia and to ensure safe fluid and/or drug administration, a 16-gauge (18-gauge in minihorse or small pony foals) jugular venous catheter should be placed in the equine neonate using aseptic technique. Catheter placement is facilitated by infiltration of the subcutaneous tissue with local anesthetic (e.g., 2% lidocaine [Lidocaine HCl USP]) at the site of skin and blood vessel puncture. In the healthy neonate, mild sedation ([Table 20-4](#)) may be necessary to facilitate aseptic placement of an IV catheter. If anesthesia is being induced using an inhalant anesthetic technique, IV catheter placement may be postponed to the moment following induction of anesthesia. If antibody titers indicate inadequacy of passive immune transfer, the neonate should receive either colostrum or plasma, as appropriate, and antibiotics because newborns are highly susceptible to serious infections when stressed by injury, metabolic disease, anesthesia, or surgery.

Sedation of the Mare

In most instances, it is desirable to have the mare present when handling an awake or mildly sedated foal because separation from the mother may trigger anxiety, excitement, and stress. To facilitate preparation of the foal for

anesthesia, sedation of the mare is highly desirable because it prevents her from becoming agitated or even aggressive toward personnel handling the foal.

A physical examination of the mare should precede any administration of sedatives or tranquilizers. Ideally the mare should be tranquilized while still in the stall with her foal. Sedative agents or a combination of drugs with relatively long duration of action are preferred. Depending on the temperament of the mare and the anticipated length of separation of mother from foal, acepromazine alone (PromAce, 0.02 to 0.05 mg/kg IV/IM) or in combination with α_2 -adrenoceptor agonists (xylazine [Rompun] 0.2 to 0.3 mg/kg IV, detomidine [Domosedan] 5 to 10 μ g/kg IV/IM, or romifidine [Sedivet] 0.02 to 0.04 mg/kg IV/IM) will provide adequate and long-lasting sedation.^{3,7, 8}

Anesthetic Management of the Neonate (1-Month-Old or Younger)

The immaturity of its central nervous, cardiopulmonary, hepatic, renal, and metabolic systems described earlier in this chapter must be kept in mind when designing the anesthetic plan for a neonate so as not to expose the foal to an increased risk of perianesthetic complications. Sedation and anesthetic drug regimens that are the least likely to impair vital functions and to cause prolonged central nervous depression are preferred.

Sedation

Foals up to 14 to 21 days of age usually do not require any chemical restraint or tranquilization to be handled and instrumented prior to induction of general anesthesia or locoregional anesthesia for brief and less-invasive surgical or diagnostic procedures.^{2,3, 7, 8, 37} If, however, sedation is required or the animal is older than 2 to 3 weeks, a benzodiazepine derivative is the preferred choice because it has limited adverse cardiopulmonary effects.^{3,7, 37} All benzodiazepines listed in [Table 20-4](#) provide sufficient sedation and muscle relaxation, thereby facilitating minor interventions such as radiographic or ultrasonographic examinations, cast application and changes, synovial or cerebrospinal fluid aspiration, rhinolaryngoscopy, intravenous catheterization, or short surgical procedures under local anesthesia or induction of general anesthesia.^{2,3, 7, 8, 37} If infusion or repeated drug dosing is anticipated to maintain sedation, midazolam (Versed) may be the better choice because the propylene glycol vehicle in other benzodiazepine preparations (diazepam [Valium], lorazepam [Ativan], clonazepam [Klonopin]) can cause metabolic acidosis and nephrotoxicity.⁴⁸ In the more mature neonate (older than 2 to 3 weeks), benzodiazepines may be supplemented with one of the opioids listed in [Table 20-4](#) and/or a low dose of xylazine (0.05 to 0.1 mg/kg) to enhance sedation and provide some analgesia.^{3,8, 37} If desired, the benzodiazepine effect can be countered at the end of the procedure using flumazenil (Romazicon; 0.025 to 0.1 mg/kg IV) or sarmazenil (Sarmasol; 0.025 to 0.1 mg/kg IV). The opioid can be antagonized with with naloxone (10 to 15 μ g/kg IV) or levallorphan (Lorfan; 22 μ g/kg IV),^{49,50} and xylazine can be reversed with yohimbine (Yocon; 0.1 to 0.2 mg/kg IM).

TABLE 20-4 Anesthetic Management of the Systemically Healthy Foal

Neonate (1 Month or Younger)

Pediatric/Juvenile Foal (1-4 Months)

	None ($\leq 2-3$ wk)	Benzodiazepines (4-8 wk)
	Benzodiazepines ($\geq 2-3$ wk)	Midazolam 0.05-0.1 mg/kg
	Midazolam 0.05-0.1 mg/kg	Diazepam 0.1-0.25 mg/kg
	Diazepam 0.1-0.25 mg/kg	Lorazepam 0.02-0.05 mg/kg
	Lorazepam 0.02-0.05 mg/kg	Climazolam 0.1-0.2 mg/kg
	Climazolam 0.1-0.2 mg/kg	α_2 -Agonists (> 8 wk)
Sedation (IV)	α_2 -Agonists (not preferred)	Xylazine 0.2-0.5 mg/kg
	Xylazine 0.2-0.5 mg/kg	Detomidine 0.005-0.01 mg/kg
	Supplementation with:	Romifidine 0.02-0.04 mg/kg
	Morphine 0.03-0.06 mg/kg	Phenothiazines
	l-Methadone 0.05 mg/kg	Acepromazine 0.03-0.05 mg/kg
	Butorphanol 0.05-0.1 mg/kg	Supplementation with:
		Morphine 0.03-0.06 mg/kg
		l-Methadone 0.05 mg/kg
		Butorphanol 0.02-0.1 mg/kg
	Pre-oxygenation (2.5-5 L/min) via mask or nasotracheal tube	
	Inhalant anesthetic in O ₂	
	Isoflurane	Injectable anesthetics (in combination with benzodiazepine listed above or guaifenesin 20-50 mg/kg IV)
Induction of anesthesia	Sevoflurane	Ketamine 2-2.5 mg/kg
	Desflurane	Propofol 1-3 mg/kg
	Injectable anesthetics (after sedation)	Ketamine 1.5 mg/kg + propofol 0.5 mg/kg
	Ketamine 2-2.5 mg/kg	Thiopental 4-6 mg/kg
	Propofol 2-2.5 mg/kg	

		Inhalant anesthetic in gas mixture ($FiO_2 > 0.3$)
		Isoflurane
	Inhalant anesthetic in O_2	Sevoflurane
	Isoflurane	Desflurane
	Sevoflurane	Supplementation* with
Maintenance of anesthesia	Desflurane	Lidocaine CRI
	Total intravenous anesthesia (TIVA)	Ketamine + propofol CRI
	Propofol 0.2-0.4 mg/kg/min	Dexmedetomidine CRI
		Total intravenous anesthesia (TIVA)*
		Triple drip CRI
		Propofol 0.1-0.3 mg/kg/min

CRI, Constant rate infusion; FiO_2 , inspired fraction of oxygen.

*

See text for more details.

Induction and Maintenance of Anesthesia

Induction and maintenance of general anesthesia can be achieved with one of the currently approved volatile anesthetics (isoflurane [Isoflo], sevoflurane [Sevoflo], or desflurane [Suprane] in O_2) or an injectable agent such as ketamine (Ketaset) or propofol (Propoflo) (see [Table 20-4](#)). Use of only a volatile anesthetic offers several advantages in neonates: (1) rapid uptake and elimination of the anesthetic via the lungs aided by the usually high minute ventilation and CO_2 ; (2) easy and rapid adjustment of anesthetic depth if untoward cardiovascular or respiratory depression or arrhythmias occur; (3) elimination of the anesthetic independent of hepatic and renal function. While the previous multicenter study⁴ indicated a 4.5 times higher risk of perioperative mortality in neonatal foals that had received an inhalant anesthetic (halothane) versus ketamine for induction of anesthesia, this finding does not coincide with a clinical investigation of the safety of two inhalant anesthetics (halothane and isoflurane) for induction and maintenance of anesthesia in foals.⁵¹ Also personal experiences do not corroborate a higher risk associated with using inhalant anesthesia in foals. Of the 153 neonatal foals anesthetized over the past 10-year period approximately 43% received one of the inhalant anesthetics (predominantly isoflurane) for induction and 57% an injectable anesthetic, yet only one animal with a perforated esophagus in which anesthesia was induced with ketamine suffered a fatal outcome because of an airway obstruction in the recovery period.

Considering the high O₂ consumption and predisposition of neonates to develop hypoxemia when being deeply sedated or anesthetized, it is recommended to have them breathe O₂

Among the injectable anesthetics, ketamine is currently the most commonly used agent for induction of anesthesia in the equine neonate, typically following sedation with a benzodiazepine derivative alone or in combination with an opioid and/or low-dose xylazine. It will induce anesthesia lasting 10 to 20 minutes (see [Table 20-4](#)).^{3,8, 37} Alternatively, either with or without benzodiazepine sedation, propofol may be administered slowly (over 45 to 60 seconds) to effect (to avoid severe respiratory depression and apnea).^{3,8, 37, 53, 54} Induction of anesthesia with thiopental or other barbiturates should be avoided in neonates because of the prolonged recovery period.

In most neonates, anesthesia is maintained with one of the volatile agents to avoid drug accumulation and slow awakening from anesthesia if injectable agents (ketamine or barbiturates) are being infused or repeatedly administered. For isoflurane, an average anesthetic vaporizer concentration setting of $2.8 \pm 0.1\%$ has been reported,⁵¹ which accords well with my own observations of an average dial setting of $2.2 \pm 0.7\%$ and end-tidal isoflurane concentration of $1.5 \pm 0.4\%$ recorded in 152 neonate anesthetics. In foals undergoing major trauma surgery, a balanced anesthesia regimen involving intermittent (every 1 to 2 hours) subcutaneous administration of a low dose of medetomidine (Dormitor, 1 to 2 $\mu\text{g}/\text{kg}$) or dexmedetomidine (Dexdormitor, 0.5 to 1 $\mu\text{g}/\text{kg}$) has advantages over maintaining anesthesia only with an inhalant anesthetic. While providing analgesia, these drugs reduce the need for high doses of volatile anesthetic that typically cause severe hypotension. Recovery (commonly assisted) to standing position usually occurs quickly, within 15 ± 1 minutes after 86 ± 4 minutes of isoflurane anesthesia⁵¹ and within 27 ± 18 minutes after 133 ± 66 minutes of isoflurane anesthesia. If higher doses of a benzodiazepine or xylazine have been used for preanesthetic sedation or if anesthesia was relatively short, reversal of the premedication agents with appropriate antagonists (e.g., flumazenil, yohimbine) should be considered to speed up recovery.

Using propofol in the neonate allows maintenance of anesthesia without risk of untoward drug accumulation and prolonged recovery. It facilitates safe anesthesia administration over extended periods of time when inhalant anesthesia may not be a feasible option, for example, for MR imaging when compatible anesthesia equipment is not available. An anesthetic technique considered suitable under those circumstances in neonates (3 to 6 days of age) includes xylazine (0.5 mg/kg IV) premedication followed 5 minutes later by a bolus (2 to 2.5 mg/kg IV) and

subsequent infusion of propofol (0.2 to 0.4 mg/kg/min).^{53,54} However, the hemodynamic effects of α_2 -agonists at such high doses cannot be ignored in this age group. A study of xylazine sedation in healthy 10- and 28-day-old foals indicated a decrease in heart rate by 20% to 30%, yet without causing second-degree atrioventricular block that is typically seen in adult horses.⁵⁵ In addition, a biphasic (initial increase followed by a decrease) change in blood pressure, similar to that in adult horses, occurred, but mean arterial pressure did not fall below 60 mm Hg.⁵⁵ Therefore, one should still exercise caution when using α_2 -agonists in the very young or sick neonate and keep doses at a minimum. In one study, recovery time after constant rate infusion of propofol (0.30 \pm 0.07 mg/kg/min) for 60 to 122 minutes ranged from 15 to 32 min (mean, 27 min), and foals suckled within 10 minutes of standing.⁵⁴

Anesthetic Management of the Pediatric/Juvenile Foal (1 to 4 Months Old)

Beyond 1 month of age, the normally developing foal of common breeds (i.e., Thoroughbreds, Standardbreds, Arabians, Quarter Horses, Warmblood horses, and Paint horses) has arrived at a stage of maturation when anesthetic techniques used in the adult can be applied with some modifications. In parallel with the maturation process, the risk for fatal perianesthetic complications seems to decrease markedly.⁴

Sedation

Systemically healthy foals 4 to 8 weeks of age (more than 120 to 150 kg body weight) or older are more difficult to physically restrain and therefore frequently require adequate tranquilization for preanesthetic catheter placement or other minor procedures. In younger pediatric foals, sedation with one of the benzodiazepine derivatives listed in [Table 20-4](#) again offers the advantage of little adverse cardiovascular and respiratory effects yet profound calming and immobilization.^{3,7, 37}

In fractious individuals or foals older than 2 months, benzodiazepine administration often causes inadequate sedation and muscle relaxation or even excitement similar to what is described in the adult horse.⁴⁹ In these foals α_2 -adrenoceptor agonists such as xylazine (0.2 to 0.5 mg/kg IV, 0.5 to 1 mg/kg IM), which is the most widely used drug in this group, as well as detomidine or romifidine, provide more reliable sedation and muscle relaxation, and in addition profound analgesia (see [Table 20-4](#) for dosages).^{2,3, 7, 8, 37} Overall, hemodynamic and respiratory side effects observed after α_2 -agonist administration in foals up to 2 to 3 months old are similar to those noted in adults with maybe the exception of atrioventricular blocks occurring rarely in younger foals.^{3,7, 37, 55, 56} Of note, xylazine has been shown to cause hypothermia in foals.⁵⁷ Unlike in adult horses, α_2 -agonists do not seem to produce hypoinsulinemia and hyperglycemia in 4-week old foals, indicating differences in pancreatic responses to α_2 -agonists in early life and further emphasizing the need to monitor blood glucose levels during prolonged sedation and anesthesia.⁵⁷ Lower dosages of xylazine (0.2 to 0.3 mg/kg IV) usually provide adequate sedation for 15 to 30 minutes and are associated with minimal cardiovascular and respiratory changes,⁷ making this drug the agent of choice for use in foals of that age group. In contrast, detomidine and romifidine have a longer duration of action and also carry a higher risk for untoward effects, including arrhythmias.^{3,56} When combined with one of the opioids listed in [Table 20-4](#), either the foal will lie down or it can be placed into lateral recumbency, allowing performance of minor surgical (in combination with local and regional anesthesia) or diagnostic procedures of short duration.^{2,7} If desired or necessary, the α_2 -agonistic effects can be antagonized at the end of the procedure using atipamezol (Antisedan, 0.05 to 0.1 mg/kg IV/IM) or yohimbine (0.1 to 0.2 mg/kg IV/IM).⁴⁹

Acepromazine in clinically common dosages produces overall mild but long-lasting sedation.^{3,37} to enhance and prolong sedation with xylazine. Clinically relevant hypotension secondary to vasodilation is a rare observation in normovolemic foals and therefore is not a concern.³

Induction and Maintenance of Anesthesia

Liver and kidney functions are significantly more mature in foals older than 1 month of age, and physical restraint becomes increasingly more difficult as the foal matures. Therefore, an IV technique is often considered the preferred method of induction of anesthesia (see [Table 20-4](#)). Ketamine is currently the most commonly used agent for induction of anesthesia in pediatric and juvenile foals and, to obtain good muscle relaxation, it is commonly combined with a benzodiazepine, unless this type of drug had been already administered for purposes of sedation. Alternatively and preferably in foals older than 3 to 4 months of age, ketamine may be coadministered with the centrally acting muscle relaxant guaifenesin (5%; Geolate), which is administered IV to effect (dropping of head, general muscle relaxation and calmness, fetlock knuckling) at a rate of 2 to 3 mL/kg/min.^{3,7, 37} To avoid inadvertent guaifenesin toxicity, the infusion container (bag, syringe, or bottle) should only contain up to the calculated maximum dosage for the individual foal, about 50 mg/kg. Following xylazine administration (0.25 mg/kg), ketamine in combination with diazepam produces anesthesia in 4- to 6-week-old foals typically of 10 minutes' duration.⁷ Ketamine may be replaced by propofol (see [Table 20-4](#)) for induction of anesthesia, but respiratory depression is likely to occur even in the more mature foal, and anesthesia may last only 5 minutes.^{3,8, 37, 54} Alternatively, ketamine and propofol may be combined for induction of anesthesia (see [Table 20-4](#)).³⁷ Thiopental in conjunction with a benzodiazepine or guaifenesin is suitable for induction of anesthesia in the more mature foal and under certain circumstances (e.g., foals with seizures or brain trauma) it is the preferred technique (see [Table 20-4](#)).^{7,37}

The NSAIDs flunixin meglumine (Banamine),^{72,73} phenylbutazone (Phenylbutazone USP),^{74,75} ketoprofen (Ketofen),⁷⁶ and ibuprofen (Caldolor)⁷⁷ have been studied in neonatal foals. Data from those studies indicate that clearance of these drugs is significantly slower and volume of distribution larger in the neonate than older foals and adult horses, causing prolonged half-lives. As a result, NSAIDs often need to be administered differently in neonatal foals, compared with adults. Under similar clinical circumstances, flunixin meglumine doses administered in neonatal foals during the first 24 hours of their life may be increased by as much as 1.5 times to induce comparable therapeutic concentrations, but in general, dosage intervals should be increased to avoid drug toxicity, including gingival and gastrointestinal ulceration, hypoproteinemia, colitis, nephrotoxicity, and platelet dysfunction, especially in sick foals.

TABLE 20-5 Systemic Analgesics for Perioperative Pain Management

Neonate (1 Month or Younger)	Pediatric/Juvenile Foal (1-4 Months)
Flunixin meglumine IV/IM q 24-36 hr 1.4 mg/kg (foal <24 hr) 0.5-1 mg/kg (foal 1-4 wk)	Flunixin meglumine 1.1 mg/kg IV/IM q 24-36 hr
NSAIDs Phenylbutazone 2.2 mg/kg IV/PO q 12-24 hr	Phenylbutazone 2.2 mg/kg IV/PO q 12-24 hr
Meloxicam 0.5-0.6 mg/kg IV q 8-12 hr	Meloxicam 0.5-0.6 mg/kg IV q 8-12 hr
Ketoprofen 1-2 mg/kg IV q 24 hr	Ketoprofen 1-2 mg/kg IV q 24 hr
Ibuprofen 10-20 mg/kg IV/PO q 8 hr	Ibuprofen 10-20 mg/kg IV/PO q 8 hr
Butorphanol 0.01-0.04 mg/kg IV	Butorphanol 0.01-0.4 mg/kg IV, IM
Butorphanol 0.02-0.08 mg/kg IM	Morphine 0.1-0.2 mg/kg IV, IM
Opioids Morphine 0.1-0.2 mg/kg IV, IM	L-Methadone 0.05-0.1 mg/kg IV, IM
L-Methadone 0.05-0.1 mg/kg IV, IM	Transdermal fentanyl (one or two 100-µg/hr patches)
Transdermal fentanyl (100-µg/hr patch)	

Neonate (1 Month or Younger)

Pediatric/Juvenile Foal (1-4 Months)

α_2 - Agonists	Xylazine 0.1-0.5 mg/kg IV (use sparingly in foal colic because of adverse effects on cardiovascular and respiratory systems and GI motility)	Xylazine 0.1-0.5 mg/kg IV/IM
	Medetomidine 1-2 μ g/kg SC	Detomidine 2-5 μ g/kg IV/IM
	Dexmedetomidine 0.5-1 μ g/kg SC	Medetomidine 2-5 μ g/kg IM/SC
Other		Dexmedetomidine 1-3 μ g/kg IM/SC
	Lidocaine 50 μ g/kg/min following 1.3-1.5 mg/kg IV loading dose	Lidocaine 50 μ g/kg/min following 1.3-1.5 mg/kg IV loading dose

GI, Gastrointestinal; *NSAIDs*, non-steroidal antiinflammatory drugs.

See text for more details.

The opioid agonist-antagonist butorphanol (0.05 mg/kg IV/IM) has been tested in newborn foals.⁷⁸ In animals up to 3 weeks of age, the elimination half-life was 2.1 hours after IV injection (about twice as long) and bioavailability was 66% \pm 12% (twice as high as in adults).⁷⁹ In neonates, butorphanol has minimal effects on vital signs but makes the animals more sedate and even mildly ataxic compared to older foals and adults, in which higher doses commonly cause excitement. Morphine or l-methadone (l-Polamivet) at doses similar to those used in adults have been used,³⁷ but there are no reports on the pharmacokinetics or pharmacodynamics of these drugs in foals. Application of fentanyl patches (Duragesic) has been tested in neonatal foals, but not for analgesic efficacy.⁸⁰ After placement of one 100- μ g/hr fentanyl patch on the skin above the jugular vein, fentanyl was detected as early as 20 minutes after patch placement, and plasma concentrations peaked after 14 \pm 8 hours and returned to baseline concentrations 12 hours after patch removal.⁸⁰ All foals satisfactorily tolerated the patch application and showed no significant adverse effects.

Foals undergoing soft tissue surgery and especially those with abdominal pain respond well to lidocaine infusion (50 μ g/kg/min following a loading dose of 1.3 mg/kg) and seem to tolerate such an infusion as well as adult animals.^{81,82}

Anesthetic Considerations for the Critically Ill Neonate and Maturing Foal

Foal with Uroperitoneum

The clinical presentation as well as medical and surgical management of uroperitoneum in newborn foals (up to 3 weeks of age) has been summarized.⁸⁷⁻⁸⁹ Reports in the past suggested a sex predilection for males and emphasized the presence of characteristic electrolyte abnormalities including hyponatremia followed by hyperkalemia and then hypochloremia, in conjunction with azotemia and metabolic acidosis. However, more recent retrospective analyses challenge this traditional view.^{88,89} A sex predilection was not observed, nor were the classic electrolyte abnormalities present in more than half of the affected foals. The clinical symptomatology appeared to be different when uroperitoneum was not the primary presenting complaint but rather developed as a secondary complaint during hospitalization. Foals having received fluids for other reasons were more likely to be septic despite having normal serum electrolyte concentrations. However, serum creatinine concentrations were always greatly elevated in foals with uroperitoneum. Of importance for the anesthetist is the fact that most foals develop respiratory distress with increased respiratory rate and respiratory effort as a result of significant abdominal distention, and lung auscultation often reveals wheezes and harsh lung sounds. Arterial O₂ saturation and tensions are frequently reduced. The heart rate is commonly and greatly increased with an irregular rhythm because of abdominal pain, hypovolemia, and hypoxemia, and a grade II systolic murmur commonly can be auscultated.

Urinary tract defects, located in the bladder wall or the urachus, require surgical repair as the treatment of choice, and thus emphasis must be placed on preanesthetic stabilization of the patient. This should include O₂ supplementation via nasal insufflation or mask delivery, restoration of circulating blood volume, correction of electrolyte and acid-base abnormalities (primarily hyperkalemia and metabolic acidosis) and slow drainage of the peritoneal fluid by abdominocentesis or by peritoneal dialysis to prevent the development of hypovolemic shock. Continuous recording of heart rate and rhythm via ECG and noninvasive measurement of blood pressures aids in monitoring the progress achieved with treatments. After initial blood volume restoration, with physiological saline or isotonic crystalloid solutions low in K⁺ content (5 mEq/L or less), hypertonic saline may be infused to correct the Na⁺ (and Cl⁻) deficit. At Na⁺ concentrations less than 110 mEq/L, seizures commonly occur.⁶⁸ The Na⁺ deficit in mEq can be calculated as: normal serum Na⁺ in mEq/L: measured serum Na⁺ in mEq/L × 0.4 × body weight in kg. The Cl⁻ deficit is of similar magnitude. If hyponatremia prevails over several days, the brain slowly adapts by altering the osmolality of its cells through loss of intracellular potassium and organic solutes. Therefore, hyponatremia should be corrected slowly (i.e., 0.5 mEq/kg/hr or less) to avoid central pontine myelinosis.⁶⁹ Serum K⁺ can be effectively decreased to clinically acceptable levels by giving regular insulin at a dose of 0.1 to 0.2 IU/kg slowly IV in 2.5% to 5% dextrose over 30 to 45 minutes prior to induction of anesthesia. The management of metabolic acidosis (pH less than 7.2) may necessitate administration of sodium bicarbonate

($\text{Na}^+\text{HCO}_3^-$). The required dose of $\text{Na}^+\text{HCO}_3^-$ (in mEq) to be administered can be determined based on the base deficit (-BE): $-\text{BE}$ in mEq/L \times 0.4 \times body weight in kg. One half of the calculated bicarbonate dose should be administered first over 20 to 30 minutes and then a blood gas analysis repeated to assess the effect before the second half of the dose is administered.

Sedation is rarely needed in sick foals with uroperitoneum, but premedication with a low dose of a benzodiazepine may be considered in less-compromised animals. Use of α_2 -agonists (e.g., xylazine) should be avoided because of their respiratory depressant and proarrhythmic properties. Induction of anesthesia with any of the modern inhalant agents (isoflurane, sevoflurane, or desflurane in O_2) is rapid and very smooth followed by the use of those agents to maintain anesthesia. Alternatively, a combination of ketamine (2 mg/kg) and diazepam or midazolam (0.2 mg/kg) may be used for induction of anesthesia, especially in the older foal, followed by inhalant anesthesia for maintenance.

The most common life-threatening arrhythmia observed in foals with uremia and hyperkalemia is a third-degree AV-block, which may be precipitated by surgical stimulation. Discontinuation of surgical stimulation and administration of atropine (20 to 40 $\mu\text{g}/\text{kg}$ IV) and/or ephedrine (25 to 50 $\mu\text{g}/\text{kg}$ IV) may resolve the arrhythmia. If these are not effective, epinephrine (10 to 20 $\mu\text{g}/\text{kg}$ IV) and closed chest massage must be initiated.