Chapter 47
Neonatal and Geriatric Patients

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Introduction

In the practice of veterinary anesthesia, neonatal and geriatric animals are a significant proportion of the patient population. Attention to the unique physiology and particular requirements of individuals within either of these age groups will contribute to the provision of safe, effective anesthesia and analgesia.

Physiology of Neonatal and Pediatric Animals

Neonatal and pediatric veterinary patients may be presented for either elective or emergency anesthesia. Much of our knowledge of the delivery of anesthesia to neonatal animals is based on our experience with young to middle-aged animals or is extrapolated from information obtained from human neonates.

Although the process of maturation and aging varies greatly between individual animals, species, and breeds, the neonatal and pediatric phases of life can be roughly defined. In dogs and cats, the neonatal period extends for the first 6 weeks of life and the pediatric period for the first 12 weeks.[1–3] Foals and calves are generally considered to be physiologically mature by 4 to 6 weeks of age.[4]

Compared with young and middle-aged adults, neonatal and pediatric patients have a limited organ reserve, a decreased ability to respond to a physiological challenge or change, and increased "sensitivity" to anesthetic drugs that is manifest as exaggerated or prolonged effects after the administration of drug dosages that are appropriate for young adults. This results in an increased risk of perianesthetic complications in neonates, necessitating judicious administration of anesthetics and vigilant monitoring.

The unique physiology of neonates results in differences in pharmacokinetics and pharmacodynamics that contribute to altered sensitivity to drugs. These differences include the following:

1. Hypoalbuminemia, which results in a greater free, active portion of protein-bound drugs. This may have a profound impact on the activity of highly protein-bound drugs like barbiturates, ketamine, etomidate, and the nonsteroidal anti-inflammatory drugs (NSAIDs).

2. Increased permeability of the neonatal blood-brain barrier, which enables a larger percentage of drug to reach the brain.

3. An increased percentage of body-water content, which alters the volume of distribution. In foals, extracellular fluid volume is 43% of body weight, compared with 22% body weight in adult horses.[5] The larger extracellular fluid volume results in a greater apparent volume of distribution of drugs that are highly ionized in plasma or relatively polar (e.g., NSAIDs).

4. A circulating fluid volume that is fixed and relatively centralized, making the neonatal patient more susceptible to hypovolemia. This reduced circulating volume also allows for greater delivery of anesthetic agents to the highly perfused tissues, including the brain.

5. Low body-fat percentage, resulting in a smaller adipose tissue compartment for drug redistribution.[6] In foals, for example, total body fat is 2% to 3%, compared with 5% in adult horses.[7]

6. Immature hepatic metabolism for the first 3 to 4 weeks (perhaps up to 12 weeks) of life.[8–10] Drug metabolism may be prolonged, extending the duration of effect of drugs or their active metabolites.

7. Immature glomerular filtration rate (GFR). In small animals, GFR does not mature until 2 to 3 weeks of age. Tubular secretion does not mature until 4 to 8 weeks of age.[8,11,12] As a result of this immaturity in renal function, the effects of drugs dependent on renal excretion for termination of activity are prolonged. For example, the half-life of diazepam is increased in neonates due to decreased renal excretion.[9] Although the kidneys mature more rapidly in foals and calves (the GFR is mature by 2 to 4 days of life and tubular secretion by 2 weeks of life),[4,8] immature renal function may be prolonged in unhealthy animals.

8. A high metabolic rate with a concomitant higher rate of oxygen consumption. This necessitates a need for a rate of alveolar ventilation that is much greater than that of adults. Because of this increase in alveolar ventilation, anesthetic induction with inhalant anesthetics may occur more rapidly.

In addition to physiological differences that result in alterations in the pharmacokinetics and pharmacodynamics of anesthetics, neonatal physiology alone can contribute to increased anesthetic risk (Table 47.1). These physiological differences include:

Table 47.1. Physiological characteristics of neonates.

Cardiovascular system
Low myocardial contractile mass
Low ventricular compliance
High cardiac index
Low cardiac reserve
Cardiac output rate dependent
Poor vasomotor control
Respiratory system
High oxygen consumption
High minute volume
Nervous system
Immature sympathetic nervous system
Poor vasomotor control
Renal and hepatic systems
Immature hepatic microsomal enzyme system
Immature renal function
Body composition
Limited thermoregulation
Low body fat and muscle mass
Low protein binding of drugs
High body-water content
Large extracellular fluid compartment

1. Compared with the adult heart, the neonatal heart has less contractile tissue per gram of myocardial tissue, and ventricular compliance is limited.[13] Stroke volume and cardiac reserve are limited in pediatric patients, and cardiac output is dependent on heart rate. Furthermore, the resting cardiac index is much higher in neonates than adults and is very close to maximal cardiac output, making the cardiac reserve minimal. An adult can increase cardiac output by 300%, whereas the neonate can only increase output by 30%.[14]

2. The sympathetic nervous system is not fully developed in neonates, and sympathetic stimulation results in only minimal increases in rate and contractility, further impairing the ability to increase cardiac output.[12] Sympathetic immaturity also manifests itself in poor vasomotor control and an incomplete or inadequate hypotension-induced baroresponse.

3. Fetal circulation may persist in some patients. For example, normal healthy foals have a right-to-left shunt for the first 3 days, and the duration of the shunt is often extended in unhealthy foals.[15]

4. In the first 1 to 3 days (foals and calves) or 1 to 2 weeks of life (puppies and kittens), neonatal kidneys may be less efficient than adult kidneys at eliminating fluid loads and regulating electrolytes, so judicious use of appropriate intravenous fluids is necessary.[6,8] Rapid or excessive fluid administration may result in pulmonary edema.

5. Pulmonary reserve is minimal, increasing the possibility of hypoxia during apnea or airway obstruction. The neonatal rib cage is compliant, resulting in less efficient ventilation and greater work of breathing. This predisposes young patients to hypoxia and ventilatory fatigue, especially in the event of airway obstruction (e.g., endotracheal tube plugged with mucus) or respiratory disease.

6. Minute ventilation is high in neonates, raising the alveolar ventilation to a functional residual capacity ratio above that of adults. Closing volume is high in the neonate and occurs within the lower range of the tidal volume.

7. In small animals, the hematocrit decreases by more than a third in the first 28 days of life;[16,17] thus, even minor hemorrhage can greatly affect oxygen delivery to tissues.

8. Neonates are more susceptible to hypothermia because of their immature thermoregulatory system, high body surface to mass ratio, and limited ability to vasoconstrict to conserve heat.

Physiology of Geriatric Animals

The effect of age per se on perioperative morbidity and mortality is related to the decreased physiological reserve of the various organ systems that occur with aging. Aging is a progressive physiological process that results in unavoidable alterations in organ system function. Within organ systems, reductions in functional reserve manifest as a decreased capacity for adaptation, a predisposition to the failure of homeostasis, and a reduced ability to respond to external stress. The effects of disease, stress, lack of exercise, genetics, malnutrition, and environment may hasten changes associated with aging. The time course of the aging process varies between organ systems within the same individual and between individuals. Assessment of the influence of aging on anatomical and physiological function in animals is further complicated by the marked variations in life span and life expectancy within and between species. There is little correlation between chronological and physiological age. For the purposes of this discussion, geriatric animals are considered to be those that have attained 75% of their expected life span.[18]

Pathophysiological changes associated with aging in organ systems influence anesthetic management (Table 47.2). Cardiovascular changes are multifactorial, reflecting not only age-related degeneration but also age-related disease. In the absence of a particular cardiovascular disease, the major anatomical changes in aging hearts are primarily an increase in the severity of myocardial fibrosis, valvular fibrocalcification, and ventricular wall thickening. Variable degrees of myocardial fiber atrophy result in decreased pump function and cardiac output. The heart rate may be affected if the pacemaker cells are involved. Fibrosis of the endocardium and valves leads to decreased compliance. Valvular incompetence may accompany valvular fibrocalcification. The vascular tree gradually loses elasticity, resulting in a decrease in distensibility, increased resistance to left ventricular output, and progressive hypertrophy of the ventricle. As ventricular hypertrophy and decreased chamber elasticity progress, the aging heart is more dependent on atrial contraction for diastolic ventricular filling. Thus, the atrial kick and normal sinus rhythm become more important in the maintenance of appropriate cardiac output.[19,20]

In geriatric animals, the maximal chronotropic response during physiological stress decreases. In addition, despite higher endogenous levels of norepinephrine, the response to stress is decreased. This appears to be due to receptor attrition and reduced affinity for

Table 47.2. Changes in the anatomy and function of major organ systems in geriatric animals.

Cardiovascular system
Decreased arterial compliance
Decreased myocardial compliance
Decreased maximal heart rate
Decreased maximal cardiac output
Blunted ß-receptor responsiveness
Pulmonary system
Reduced gas-exchange efficiency
Reduced vital capacity
Increased work of breathing
Decreased thoracic compliance
Decreased lung elasticity
Increased degree of airway closure
Nervous system
Increased sympathetic nervous system activity
Downregulation of ß receptors
Decreased parasympathetic nervous system activity
Reduced central neurotransmitter activity
Increased sympathetic nervous system outflow
Renal and hepatic systems
Decreased drug clearance
Decreased glomerular filtration rate
Difficulty handling water-and-salt loads
    Decreased urine concentrating ability and decreased ability to
      conserve sodium
Decreased perfusion and system blood flow
Decreased tissue mass
Body composition
Decreased skeletal muscle mass
Increased lipid fraction

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agonist molecules. Whereas young adults increase cardiac output primarily through increased heart rate, geriatric animals increase cardiac output by increasing stroke volume in association with an increase in end-diastolic volume. Thus, geriatric animals depend more on preload than do younger animals and are not as tolerant of volume depletion in the perianesthetic period. This said, fit individuals maintain high levels of cardiac output and oxygen consumption, and reductions in cardiac index occur in direct proportion to reductions in skeletal muscle mass and metabolic rate associated with reductions in lean-tissue mass.[19-21]

Pulmonary changes associated with aging include a decrease in ventilatory volumes and a reduction in the efficiency of gas exchange. Vital capacity, total lung capacity, and maximum breathing capacity decrease as the intercostal and diaphragmatic muscle masses reduce and the thorax becomes more rigid and less compliant. Functional alveoli and elasticity progressively decrease. As pulmonary elasticity decreases as result of decreases in lung elastin, the ratio of residual volume and of functional residual capacity to total lung capacity increase. Closing volume is increased, resulting in air trapping and an increase in ventilation-perfusion mismatch. As a result, PaO_2 decreases with age.[22]

In the central nervous system, aging is associated with a reduction in brain size that occurs with the loss of neurons. Cerebro-spinal fluid volume increases to maintain normal intracranial pressure. Despite this loss in brain tissue, functional and anatomical redundancy within the nervous system provides for the maintenance of functioning at levels that approximate those observed at somatic maturity. With the loss of brain tissue, cerebral blood flow decreases, but cerebral autoregulation of blood flow is well maintained. In addition to the loss of functional neurons in aging individuals, generalized depletions of dopamine, norepinephrine, tyrosine, and serotonin occur. Receptor affinity for neurotransmitters may be reduced. Compared with the neuronal plasticity observed in the young, this process is slower and less complete in geriatric individuals. As a result of these functional and anatomical changes in the central nervous system, geriatric individuals have a decreased requirement for anesthetic agents. The minimum alveolar concentration for inhalant anesthetics decreases linearly with age, and the requirement for local anesthetics, opioids, barbiturates, benzodiazepines, and other intravenous drugs is likely similarly reduced.[19,23,24]

With aging, there is a primary loss of cortical kidney mass and functional nephron units. Total renal blood flow decreases with age, with the majority of the loss occurring in the renal cortex. In humans, one-half of the glomeruli present in the young adult atrophy or are nonfunctional by the age of 80. Glomerular filtration rate decreases, partly in response to a reduction in renal plasma flow. Geriatric individuals are less responsive to antidiuretic hormone and have an impaired ability to conserve sodium or concentrate urine. A reduction in renal blood flow makes the geriatric animal more susceptible to renal failure in the face of renal ischemia. Since geriatric patients cannot maximally retain sodium or water under conditions of volume depletion, the ability to correct fluid, electrolyte, and acid-base disturbances or to tolerate hemodynamic insults is reduced. Because geriatric patients have difficulty excreting a salt-and-water load, vigorous fluid and electrolyte therapy may result in excessive intravascular and extravascular volume, with the possible sequelae of congestive heart failure and peripheral edema. Those anesthetic drugs eliminated primarily by renal excretion have a greater elimination half-time in geriatric animals, necessitating a reduction in doses when these drugs are administered.[25,26]

Hepatic clearance of drugs decreases with age as the mass of the liver decreases. In geriatric people, the liver mass, and consequently hepatic blood flow, may be decreased by 40% to 50%. Microsomal and nonmicrosomal enzyme function appears to be well maintained, although the reduction in hepatic mass significantly impairs overall hepatic function. Consequently, the metabolism of lipid-soluble drugs, particularly anesthetics, is decreased. Combined with decreased glomerular filtration and renal excretory capacity, the reduction in hepatic clearance of drugs results in an increase in the half-life and duration of effect of drugs that depend on these routes of elimination.[19,25-27]

Aging results in changes in body composition that include a decrease in skeletal muscle, an increase in body fat as a percentage of total body weight, and a loss of intracellular water. A loss in total body water occurs as a result of decreased intracellular water and a reduction in plasma volume, although fit geriatric individuals maintain plasma volume well. Intravenous injection of

anesthetic drugs into a contracted volume of distribution results in an increased plasma concentration that may be responsible for the observation that geriatric animals are more sensitive to anesthetics. Increased adipose tissue is associated with an increase in the fraction of a single dose of a lipid-soluble drug redistributed to adipose tissue, further delaying elimination from the body.

Reductions in serum albumin concentrations in association with aging can lead to reduced protein binding of drugs. In addition, structural changes in the serum protein that occur with aging may decrease binding to the available protein. Theoretically, the administration of highly protein-bound drugs to animals with reduced serum proteins may lead to an exaggerated clinical effect.[28]

A decrease in basal resting metabolic rate with age results in a reduction in the production of body heat. Consequently, geriatric individuals are less able to maintain core body temperature. This is particularly important in anesthetized animals placed in cold environments. Anesthetized geriatric animals tend to be more hypothermic than younger animals in the operative and postoperative periods, and rewarming occurs at a slower rate. Because shivering during recovery increases oxygen consumption by 200% to 300%, perianesthetic hypothermia alone may place severe demands on the cardiopulmonary system. If these demands are not met, arterial hypoxemia may ensue.[19]

Anesthesia

Preparation for Anesthesia

A thorough physical examination, including careful auscultation of the heart, is an essential component of preanesthetic assessment. In geriatric people, exercise tolerance is one of the most important predictors of perioperative outcome, and this is likely an important predictor of outcome in the anesthesia of geriatric animals. Significant, preexisting abnormalities should be corrected prior to the induction of anesthesia. Left untreated, these abnormalities are likely to be exacerbated by anesthesia. Neonatal, pediatric, or geriatric animals with limited physiological reserves are less able to respond to altered homeostasis. Hydration status should be assessed and fluid requirements carefully calculated. Fluid deficits should be corrected prior to anesthesia.

In neonates, standard preanesthetic blood work should include a minimum of hematocrit, total and fractionated protein, and blood glucose. Other blood work should be performed as indicated. In geriatrics, the preanesthetic assessment should include a complete blood count, serum chemistry profile, electrocardiogram, and urinalysis.

Neonates that are still suckling should not be held off food prior to anesthesia. Pediatric animals that are eating solid food should be denied food for only 3 to 4 h prior to anesthesia and should not be denied water at any time.

Premedication and Pain Management

Because of the immaturity of the neonatal nervous system, it has been a commonly held thesis that neonates are incapable of experiencing pain. However, we now know that neonatal humans and animals do indeed experience pain.[29,30] In addition, evidence suggests that pain experienced at an extremely young age may

lead to dynamic changes in the nociceptive pathway, resulting in chronic pain conditions later in life.[31] Like many pathologies, pain is easier to prevent than to treat. Regardless of age, every patient anesthetized for a painful procedure should receive appropriate analgesic therapy. As with other drugs, dosages of the analgesic drugs should be conservative, and patients should be closely monitored for signs of adverse side effects. Opioids are potent analgesics whose effects are reversible, making them an excellent choice for analgesia in neonates with immature metabolism or geriatrics with reduced hepatic function. The addition of local anesthetic techniques to anesthetic protocols for neonatal or geriatric animals is particularly appropriate. The inclusion of such techniques provides for additional analgesia and an associated reduction in the requirement for general anesthetics that may have more profound side effects. NSAIDs may be an option in older pediatric patients, but this class of drugs should be reserved for patients with mature, competent renal function.

The routine use of anticholinergics is not necessary, but, because neonates depend on adequate heart rate for maintenance of cardiac output, anticholinergics may be used to treat sinus bradycardia. Anticholinergics should also be considered in breeds with high vagal tone (e.g., brachycephalic breeds) and for surgical procedures that are likely to stimulate a vagal reflex (e.g., many ocular procedures). Geriatric animals may be less able to compensate for a slow heart rate than younger adult animals.

Sedation may not be necessary in quiet or debilitated patients. However, sedatives alleviate stress in anxious patients and decrease the dosage of drugs needed for induction and maintenance of anesthesia. The side effects of sedatives should be weighed against the side effects of having to use a larger dose of anesthetic drug. The opioids often provide adequate sedation and have the added advantage of providing analgesia. μ-Agonist opioids such as morphine, hydromorphone, and oxymorphone provide the most profound analgesia but also may cause greater cardiovascular and respiratory depression.[32] Partial agonists (buprenorphine) and agonist-antagonists (butorphanol) provide only mild to moderate analgesia but also cause minimal cardiovascular and respiratory depression. Selection of an appropriate opioid is best made based on the particular analgesic requirements and the health status of the patient and the intended procedure.

Benzodiazepines are also useful sedatives/anxiolytics in neonatal or geriatric animals. Although they do not provide analgesia, they are reversible and produce little to no cardiovascular and respiratory depression. Benzodiazepines do not provide consistent or deep sedation and may need to be combined with other sedatives in very active or anxious neonatal or geriatric animals. The judicious use of low doses of α_2-adrenergic agonists may also be considered for use in neonatal or geriatric animals. However, drugs in this class produce profound cardiovascular side effects (bradycardia, atrioventricular conduction block, and increased peripheral vascular resistance), and their use in either neonatal or geriatric animals should be confined to those cases where the benefit of their use outweighs the negative side effects associated with their administration. Low dosages of xylazine have been shown to be safe and effective in foals as young as 10 days of age.[33] Acepromazine may also be used for sedation of

neonates or geriatrics, but the cardiovascular effects, including hypotension, may be poorly tolerated in both geriatric and neonatal animals. The vasodilation caused by acepromazine can also contribute to the development of hypothermia. Acepromazine is not reversible and alone does not provide analgesia.

Induction

Anesthesia can be induced by using a variety of injectable anesthetic drugs or by mask delivery of inhaled anesthetics, when necessary. Etomidate or propofol may be excellent choices in neonates and geriatrics. Both agents are rapidly eliminated from the body by a variety of routes so that termination of activity does not depend on the functioning of a single organ system.[34] Propofol does produce respiratory and cardiovascular depression and should be titrated to achieve the desired depth of anesthesia. The administration of oxygen by face mask for approximately 5 min prior to the induction of anesthesia with propofol will reduce the risk of complications associated with propofol-induced apnea in either neonatal or geriatric animals. Preinduction sedation of animals, particularly with a sedative/analgesic drug, will reduce the dose of etomidate or propofol needed for induction. Ketamine, in combination with a benzodiazepine, is also a choice for the induction of anesthesia in either neonates or geriatric animals. Ketamine causes only mild respiratory depression and may actually improve cardiovascular function through stimulation of the sympathetic nervous system.[35] Although of benefit to geriatric animals, this response may be less in neonates because of their immature sympathetic nervous system. Ketamine requires either hepatic metabolism or renal clearance for termination of activity; thus, the effects of ketamine may be prolonged in patients with either immature or failing hepatic and renal systems.[35] Barbiturates may be used in low dosages for the induction of anesthesia in neonatal, pediatric, or geriatric animals, keeping in mind that drugs of this class are highly protein bound, and their termination of activity depends on both redistribution and hepatic metabolism. The response to barbiturates can be both pronounced and prolonged in either neonates or geriatrics with reduced plasma protein concentrations and/or immature or failing hepatic or renal function.

Inhaled anesthetics may be used for induction, as well as maintenance of anesthesia. This method is recommended only in sedated or debilitated patients because the prolonged excitement phase that occurs during induction can be more physiologically detrimental than a judicious dose of an injectable anesthetic. Environmental pollution and personnel exposure are also concerns during mask inductions with inhaled anesthetics. Induction in foals and calves with an inhaled anesthetic following nasotracheal intubation often results in rapid, excitement-free anesthesia simply because bypassing the nasal passages eliminates the ability to smell anesthetic gas.[36,37] Because anesthesia can be induced with an inhaled anesthetic very rapidly in depressed neonatal and geriatric animals, excessive anesthetic depth may be reached very rapidly. Careful monitoring is a must.

Maintenance

Inhaled anesthetics that are minimally metabolized and easily cleared in animals with either immature or reduced hepatic or renal function should be used for maintenance of anesthesia. However, it should be remembered that inhaled anesthetics cause dose-dependent hypotension, hypoventilation, impaired cardiac contractility, and hypothermia. Because of these side effects, inhaled anesthetics must be very carefully titrated, and vigilant monitoring should be employed to avoid excessive anesthetic depth. The concurrent administration of analgesics and sedatives will reduce the inhaled anesthetic requirement while decreasing the magnitude of their unwanted side effects.

Support

Along with a carefully chosen anesthetic protocol and conservative drug dosing, meticulous physiological support and vigilant monitoring are imperative during the anesthesia of neonatal, pediatric, or geriatric animals. Compared with adults, fluid requirements are greater in neonates (60 to 180 mL/kg/day)[38] because of their higher body-surface area, immature renal function (decreased ability to concentrate urine), higher percentage of body water, and higher respiratory rates leading to greater fluid losses.[9] Conversely, overhydration should be avoided, because renal clearance may be limited and excessive dilution of serum protein can occur more readily in animals with preexisting hypoalbuminemia. Neonates have minimal stores of hepatic glycogen and are prone to hypoglycemia, so the use of dextrose-containing fluids should be considered.

In geriatric patients that have difficulty excreting a salt-and-water load, aggressive fluid and electrolyte therapy may result in excessive intravascular and extravascular volume, with the possible sequelae of congestive heart failure and peripheral edema. Thus, fluid therapy in the perianesthetic period of geriatric animals should be targeted at correcting specific deficits and maintaining adequate perfusion and oxygen delivery without delivering excessive electrolyte loads or fluid volumes.

Both neonatal and geriatric animals are highly susceptible to hypothermia, so every effort should be made to maintain normal body temperature. Hypothermia increases the incidence of adverse myocardial outcomes in high-risk patients, increases the incidence of surgical wound infection, adversely affects antibody- and cell-mediated immune defenses, changes the kinetics and action of various anesthetic and paralyzing agents, increases thermal discomfort, and is associated with delayed postanesthetic recovery.[39] The attempt of the body to rewarm itself is not benign, because shivering may cause a tremendous increase in oxygen consumption (200% to 300%), and this increased oxygen demand may not be met by an increase in oxygen delivery, particularly if anesthetic-induced hypoventilation occurs.

Diligent monitoring is crucial during the entire anesthetic period and well into recovery. Some commonly monitored indices are different in neonates than in adults, and anesthetists should be familiar with the normal physiological indices for each species and age group of individuals that are being anesthetized. Generally, neonatal and pediatric animals have a higher heart rate but lower blood pressure than adults. The normal heart rate in conscious neonatal dogs and cats is approximately 200 beats per minute, and the respiratory rate is approximately 15 to 35 breaths per minute. Mean arterial blood pressure in 1-month-old puppies

Table 47.3. Drugs commonly used in geriatric small animals.[a]

	Dose (mg/kg)	
Drug	Dog	Cat
Anticholinergics		
Atropine[b]	0.01–0.02	0.01–0.02
Glycopyrrolate[b]	0.005–0.01	0.005–0.01
Sedatives and analgesics		
Midazolam[b]	0.1–0.3	0.1–0.3
Diazepam[c]	0.2–0.4	0.2–0.4
Oxymorphone	0.1–0.2	0.1–0.2
Hydromorphone	0.1–0.2	0.1–0.2
Butorphanol	0.2–0.4	0.2–0.4
Buprenorphine	0.005–0.02	0.005–0.02
Induction		
Propofol	4–6	4–6
Ketamine-midazolam	3–5 and 0.1–0.2	3–5 and 0.1–0.2
Hydromorphone-diazepam	0.1 and 0.2	0.1 and 0.2
Etomidate	0.5–1.5	0.5–1.5

[a]Endeavor always to use the minimum effective dose of all drugs to achieve surgical or diagnostic objectives.
[b]Intramuscular or intravenous administration is appropriate.
[c]Diazepam is not recommended for intramuscular use and should be given slowly when administered intravenously.

is only 49 mm Hg.[40] The average heart rate in foals 1 to 2 days of age ranges from 70 to 90[41] beats per minute, and the normal respiratory rate is 30 to 40 breaths per minute.[42]

Summary of Protocols

Suggested protocols for neonatal animals include an opioid premedicant with an additional sedative or tranquilizer, if required. Anesthesia can be induced with the intravenous administration of propofol or ketamine-benzodiazepine combinations. In severely compromised animals, anesthesia may be induced with an inhaled anesthetic delivered by face mask.[43,44] For neonatal horses and cattle, suggested protocols include sedation with low-dose xylazine plus butorphanol followed either by intravenous administration of propofol or valium-ketamine or by nasal intubation and subsequent induction with an inhalant anesthesic.[4,37] For all species, anesthesia is maintained with an inhalant and local blockade employed when possible to augment analgesia.

No one ideal anesthetic protocol exists for all geriatric patients. An understanding of the pathophysiological changes and the alterations in pharmacodynamics and pharmacokinetics that arise in conjunction with aging is necessary when choosing an anesthetic protocol for any geriatric animal. Particular attention to decreased dosage requirements and the titration of anesthetics to achieve the central nervous system depression necessary for a specific surgical procedure is advocated (Table 47.3). Whenever possible, local and regional anesthetic techniques should be employed to reduce the dosage of concomitantly administered inhaled or injectable anesthetics. Appropriate anesthetic management of geriatric animals is centered around thorough evaluation and assessment; preoperative correction of identified abnormalities; vigilant, aggressive perianesthetic monitoring; careful titration of anesthetic drugs; and appropriate perianesthetic support.

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