Manual of Equine Anesthesia and Analgesia

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Preface

As in all areas of veterinary practice, equine anesthesia and analgesia have progressed rapidly over the last two decades with the introduction of new drugs, user-friendly monitoring devices and new methods of using drugs. Important knowledge has also been gained in identifying the risk factors for equine anesthesia. There is a growing awareness of the impact of anesthesia and analgesia on the surgical outcome, and a realization that equine anesthesia is not just a technical procedure aimed at producing immobilization for the sake of operator comfort.

This handbook is intended to be a useful clinical guide. The layout has been planned so that the information will be easily accessible, and an attempt has been made to impose some order on the confusion of facts which confront students and clinicians. We hope that we have achieved that goal. Drugs such as chloroform and chloral hydrate, which are rarely used nowadays, have been omitted.

Undoubtedly, not everyone will agree with all the descriptions of how to perform clinical anesthesia as we each have our own preferences. For instance, some readers will not feel comfortable with the multimodal drug approach to general anesthesia. We have emphasized techniques which have, over the years, been found to be effective for the authors. However, we realize that there are other acceptable methods.

It is our sincere hope that this handbook will be a valuable source of information for all involved in equine anesthesia.

Tom Doherty
Alex Valverde
Acknowledgments

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<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Full Form</th>
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<tbody>
<tr>
<td>ACh</td>
<td>acetylcholine</td>
</tr>
<tr>
<td>ACT</td>
<td>activated clotting time</td>
</tr>
<tr>
<td>AEP</td>
<td>auditory evoked potential</td>
</tr>
<tr>
<td>AF</td>
<td>atrial fibrillation</td>
</tr>
<tr>
<td>ANS</td>
<td>autonomic nervous system</td>
</tr>
<tr>
<td>AP</td>
<td>action potential</td>
</tr>
<tr>
<td>aPTT</td>
<td>activated partial thromboplastin time</td>
</tr>
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<td>AST</td>
<td>aspartate aminotransferase</td>
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<td>ATIII</td>
<td>antithrombin III</td>
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<td>AV</td>
<td>arteriovenous</td>
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<td>AVP</td>
<td>arginine vasopressin</td>
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<td>BIS</td>
<td>bispectral index</td>
</tr>
<tr>
<td>BUN</td>
<td>blood urea nitrogen</td>
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<tr>
<td>CBIL</td>
<td>conjugated (direct) bilirubin</td>
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<tr>
<td>CHF</td>
<td>congestive heart failure</td>
</tr>
<tr>
<td>CK</td>
<td>creatine kinase</td>
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<tr>
<td>COX</td>
<td>cyclooxygenase</td>
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<tr>
<td>CPD</td>
<td>citrate-phosphate-dextrose</td>
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<tr>
<td>CPDA-1</td>
<td>citrate-phosphate-dextrose-adenine</td>
</tr>
<tr>
<td>CPK</td>
<td>creatinine phosphokinase</td>
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<tr>
<td>CRH</td>
<td>corticotropin releasing hormone</td>
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<tr>
<td>CRI</td>
<td>constant rate infusion</td>
</tr>
<tr>
<td>CSF</td>
<td>cerebrospinal fluid</td>
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<tr>
<td>CSLH</td>
<td>context-sensitive half-life</td>
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<td>CVP</td>
<td>central venous blood pressure</td>
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<td>CVS</td>
<td>cardiovascular system</td>
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<tr>
<td>DA</td>
<td>dopaminergic</td>
</tr>
<tr>
<td>DIC</td>
<td>disseminated intravascular coagulation</td>
</tr>
<tr>
<td>DMSO</td>
<td>dimethylsulphoxide</td>
</tr>
<tr>
<td>ECFV</td>
<td>extracellular fluid volume</td>
</tr>
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<td>ECG</td>
<td>electrocardiogram</td>
</tr>
<tr>
<td>ER</td>
<td>exertional rhabdomyolysis</td>
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<tr>
<td>ETCO₂</td>
<td>end-tidal carbon dioxide</td>
</tr>
<tr>
<td>FDP</td>
<td>fibrin/fibrinogen degradation product</td>
</tr>
<tr>
<td>Abbreviation</td>
<td>Definition</td>
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<tr>
<td>--------------</td>
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<tr>
<td>FFT</td>
<td>Fast Fourier transformation</td>
</tr>
<tr>
<td>FIO₂</td>
<td>inspired oxygen fraction</td>
</tr>
<tr>
<td>FRC</td>
<td>functional residual capacity</td>
</tr>
<tr>
<td>FSP</td>
<td>fibrin split product</td>
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<tr>
<td>GABA</td>
<td>gamma amino butyric acid</td>
</tr>
<tr>
<td>GFR</td>
<td>glomerular filtration (or flow) rate</td>
</tr>
<tr>
<td>GGT</td>
<td>gamma glutamyl transferase</td>
</tr>
<tr>
<td>GnRH</td>
<td>gonadotropin releasing hormone</td>
</tr>
<tr>
<td>GX</td>
<td>glycineylidine</td>
</tr>
<tr>
<td>HR</td>
<td>heart rate</td>
</tr>
<tr>
<td>HYPP</td>
<td>hyperkalemic periodic paralysis</td>
</tr>
<tr>
<td>ICFV</td>
<td>intracellular fluid volume</td>
</tr>
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<td>ICP</td>
<td>intracranial pressure</td>
</tr>
<tr>
<td>IDH</td>
<td>iditol dehydrogenase</td>
</tr>
<tr>
<td>IFV</td>
<td>interstitial fluid volume</td>
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<tr>
<td>IPPV</td>
<td>intermittent positive pressure ventilation</td>
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<tr>
<td>IVCT</td>
<td>in vitro contracture testing</td>
</tr>
<tr>
<td>LAL</td>
<td>large-animal vertical lift</td>
</tr>
<tr>
<td>LP</td>
<td>lipopolysaccharide</td>
</tr>
<tr>
<td>MAC</td>
<td>minimum alveolar concentration</td>
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<tr>
<td>MEGX</td>
<td>monoethylglycinexilidine</td>
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<tr>
<td>MH</td>
<td>malignant hyperthermia</td>
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<tr>
<td>MLAEP</td>
<td>middle latency auditory evoked potential</td>
</tr>
<tr>
<td>NE</td>
<td>norepinephrine</td>
</tr>
<tr>
<td>NMDA</td>
<td>N-methyl-D-aspartate</td>
</tr>
<tr>
<td>NSAID</td>
<td>nonsteroidal anti-inflammatory drug</td>
</tr>
<tr>
<td>PAB</td>
<td>premature atrial beats</td>
</tr>
<tr>
<td>PCV</td>
<td>packed cell volume</td>
</tr>
<tr>
<td>PDA</td>
<td>patent ductus arteriosus</td>
</tr>
<tr>
<td>PDN</td>
<td>palmar digital nerve</td>
</tr>
<tr>
<td>PEEP</td>
<td>positive end-expiratory pressure</td>
</tr>
<tr>
<td>PIVA</td>
<td>partial intravenous anesthesia</td>
</tr>
<tr>
<td>PLA₂</td>
<td>phospholipase A₂</td>
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<tr>
<td>PNS</td>
<td>peripheral nervous system</td>
</tr>
<tr>
<td>PP</td>
<td>perfusion pressure</td>
</tr>
<tr>
<td>PPV</td>
<td>positive pressure ventilation</td>
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<tr>
<td>PSSM</td>
<td>polysaccharide storage myopathy</td>
</tr>
<tr>
<td>PT</td>
<td>prothrombin time</td>
</tr>
<tr>
<td>PV</td>
<td>plasma volume</td>
</tr>
<tr>
<td>PVC</td>
<td>premature ventricular contraction</td>
</tr>
<tr>
<td>PVR</td>
<td>peripheral vascular resistance</td>
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<tr>
<td>Abbreviation</td>
<td>Full Form</td>
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<td>--------------</td>
<td>-----------</td>
</tr>
<tr>
<td>RAO</td>
<td>recurrent airway obstruction</td>
</tr>
<tr>
<td>RR</td>
<td>respiratory rate</td>
</tr>
<tr>
<td>SA</td>
<td>sinoatrial</td>
</tr>
<tr>
<td>SBA</td>
<td>serum bile acids</td>
</tr>
<tr>
<td>SCh</td>
<td>succinylcholine</td>
</tr>
<tr>
<td>SDH</td>
<td>sorbitol dehydrogenase</td>
</tr>
<tr>
<td>SGOT</td>
<td>serum glutamic oxaloacetic transaminase</td>
</tr>
<tr>
<td>SID</td>
<td>strong ion difference</td>
</tr>
<tr>
<td>SNS</td>
<td>sympathetic nervous system</td>
</tr>
<tr>
<td>SV</td>
<td>stroke volume</td>
</tr>
<tr>
<td>TBIL</td>
<td>total bilirubin</td>
</tr>
<tr>
<td>TBW</td>
<td>total body water</td>
</tr>
<tr>
<td>TFPI</td>
<td>tissue factor pathway inhibitor</td>
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<tr>
<td>TIVA</td>
<td>total intravenous anesthesia</td>
</tr>
<tr>
<td>TNF$_{\alpha}$</td>
<td>tumor necrosis factor-alpha</td>
</tr>
<tr>
<td>TOF</td>
<td>train-of-four</td>
</tr>
<tr>
<td>TP</td>
<td>total protein</td>
</tr>
<tr>
<td>tPA</td>
<td>tissue plasminogen activator</td>
</tr>
<tr>
<td>TRH</td>
<td>thyrotropin releasing hormone</td>
</tr>
<tr>
<td>TSH</td>
<td>thyroid stimulating hormone</td>
</tr>
<tr>
<td>UBIL</td>
<td>unconjugated (indirect) bilirubin</td>
</tr>
<tr>
<td>uPA</td>
<td>urokinase plasminogen activator</td>
</tr>
<tr>
<td>USG</td>
<td>urine specific gravity</td>
</tr>
<tr>
<td>vWD</td>
<td>von Willebrand’s disease</td>
</tr>
</tbody>
</table>
1 Preoperative evaluation

The risk of equine anesthesia

Tanya Duke

Most of what is known about the risk of equine anesthesia comes from information gathered in a worldwide, multicenter study, and the following information is based, in large part, on these findings.

I. Risk of equine anesthesia

- Data from single clinics have cited the mortality rate in healthy horses to be between 0.63% and 1.8%.
- Data from multicenter studies cite the death rate for healthy horses undergoing anesthesia at around 0.9% (approximately 1:100).
- The overall death rate, when sick horses undergoing emergency ‘colic’ surgery are included, is around 1.9%.
- Surveys of feline and canine anesthesia have documented risk of mortality in healthy patients to be 1:2065 and 1:1483, respectively.
- Clearly, the risk of fatality during anesthesia of healthy horses is greater than for small animals.

II. Risk factors

A. Age
- The risk increases with age, and horses aged 14 years or older are at an increased risk of mortality.
- Older horses may be more prone to fracture of a long bone in the recovery period, resulting in euthanasia.
- Foals have an increased risk of dying and this is speculated to be associated with unfamiliarity with neonatal anesthesia, and presence of systemic illness.

B. Type of surgery
- In otherwise healthy horses, the risk following fracture repair is highest.
- This increased risk probably arises from re-fracture and other problems during the recovery period resulting in euthanasia.
• However, long periods of anesthesia typical of fracture surgery repair have also been associated with increased mortality, and horses presented for fracture repair may be dehydrated and stressed.
• Emergency surgery (non-colic) carries a 4.25 times higher risk of mortality compared with elective surgery, and for colics the risk of fatality is 19.5%.

C. Time of day
• Performing anesthesia outside of normal working hours carries an increased risk for horses. This increase in risk is separate from the fact that most of these cases are emergency in nature.
• Surgeries performed between midnight and 6 a.m. carry the highest risk of mortality. This may be due to the nature of the emergency, as well as to staff shortages and tiredness.

D. Body position
• This has not been found to increase risk after including operation type in the analysis, since most ‘colic’ surgeries are performed with the horse in dorsal.

E. Drug choice
• Using total inhalational anesthesia regime in foals (<12 months of age) without premedication carries the highest risk.
• Halothane, which sensitizes the myocardium to circulating catecholamines, may have a higher risk than newer volatile anesthetics.
• Not using any premedication is associated with the highest risk, probably owing to increased circulating catecholamines from stress.
  – It may be prudent to premedicate foals before induction of anesthesia.
• Acepromazine lowers the risk of mortality, when it is used on its own as a premedicant. This may be due to acepromazine’s stabilizing effect on the heart, making it less susceptible to ventricular arrhythmias.
• No particular injectable induction regime is associated with greater risk when used with inhalational anesthesia.
• Total intravenous anesthesia (TIVA) is associated with the lowest risk of all, but this may be due to the fact that TIVA is used for shorter procedures.

F. Duration of anesthesia
• Long periods of anesthesia with volatile anesthetics are often associated with cardiovascular depression and poor tissue perfusion leading to problems such as cardiac arrest or post-anesthetic myopathy.
Preoperative evaluation and patient preparation

I. Risk management

- Those of us involved in equine anesthesia are in the risk management business.
- Anesthesia of the horse is never without risk.
- The risks range from the less serious (e.g. skin wounds) to the more serious (e.g. myopathies and peripheral neuropathies) and to death in some cases.
- There is also a risk to personnel and this should never be taken lightly.
- The goal of the anesthesiologist is to minimize the adverse effects of these risks (ideally at minimum cost) by:
  - Identifying and defining the risk(s).
  - Selecting the best strategy for controlling or minimizing the risk(s).

II. Classification of physical status

- Classification of health status is generally based on the American Society of Anesthesiologists (ASA) system.
- This system uses information from the history, physical examination and laboratory findings to place patients into one of five categories.
- The classification allows for standardization of physical status only.
- The ASA system does not classify risk.
- These classifications are not as useful for equine patients; nevertheless, the system serves as a guide.

<table>
<thead>
<tr>
<th>ASA 1</th>
<th>A healthy horse.</th>
</tr>
</thead>
<tbody>
<tr>
<td>ASA 2</td>
<td>Horse with mild systemic disease (e.g. mild anemia, mild recurrent airway obstruction).</td>
</tr>
<tr>
<td>ASA 3</td>
<td>Horse with severe systemic disease (e.g. severe recurrent airway obstruction).</td>
</tr>
<tr>
<td>ASA 4</td>
<td>A horse with severe systemic disease that is a constant threat to life (e.g. ruptured urinary bladder, intestinal accident).</td>
</tr>
<tr>
<td>ASA 5</td>
<td>A moribund horse not expected to survive longer than 24 hours (e.g. foal with a uroperitoneum with severe metabolic derangements).</td>
</tr>
<tr>
<td>E</td>
<td>The letter E is added to each classification for emergency procedures.</td>
</tr>
</tbody>
</table>

III. Patient preparation

A. Evaluation

- The horse should be evaluated in light of its history and physical findings.
- Many emergency cases, especially intestinal emergencies, are in cardiovascular shock and must be resuscitated prior to induction of anesthesia.
- If deemed necessary, laboratory data are important in order to determine suitability for anesthesia and to determine the risk.
B. Laboratory tests

- In normal horses undergoing elective surgery, there is generally no value in performing laboratory tests.
- In emergency cases, performing laboratory tests may be vital to the management of the case (e.g. a foal with urinary bladder rupture).

C. Physical examination

- During the examination, particular attention should be directed to the cardiovascular and respiratory systems.
- Musculoskeletal problems, which may affect recovery, should be considered, and a plan should be made to assist recovery if deemed necessary.

D. History

- May reveal information that affects case management.
- A recent history of coughing may indicate a viral infection of the airway, in which case elective surgeries should be postponed until one month following resolution of clinical signs.
- Owners often report that the horse has previously had a ‘bad’ or ‘over’ reaction to some anesthetic drug. In most cases these are misunderstandings on the part of the owner, but they should nevertheless be noted.

E. Fasting

- Fasting (~ 12 h) was previously advised because of the potential benefits for lung function and the reduced risk of stomach rupture from trauma at induction or recovery.
- Some clinicians question this reasoning and many equine hospitals do not fast horses prior to elective surgery.
  - There is also a concern that fasting may increase the risk of postoperative ileus, although there is no evidence to support this.

F. Medications

- It is best to administer all ancillary drugs (e.g. antimicrobials, anti-inflammatory) prior to sedation.

G. Jugular catheter

- An intravenous catheter should always be placed prior to anesthesia.
- This reduces the likelihood of perivascular injection and provides ready access to the vein, for medication administration, in emergency situations.

H. Flushing the oral cavity

- It is important to flush food debris from the oral cavity, especially if the airway is going to be intubated.
I. Removal of shoes

- Removal of shoes is sometimes practiced to prevent damage to the horse and hospital flooring.
  - However, removal of shoes is not popular with owners. An alternative is to apply bandage material or tape to improve grip and cover metal points.
- Certainly, loose shoes and nails should be removed.

Serum chemistry testing prior to anesthesia

Nicholas Frank

- Ideally, routine serum chemistry results should be examined before general anesthesia is induced.
  - These values are particularly useful for assessing problems that cannot be recognized by physical examination.
  - For instance, a horse suffering from acute renal failure may appear healthy upon physical examination, but disease is revealed when serum blood urea nitrogen and creatinine concentrations are examined.
- This discussion focuses upon four body systems (muscle, liver, kidneys and plasma proteins) that should be assessed prior to anesthesia by examining serum chemistry values.
- Reference ranges are provided for each of the variables discussed, but clinicians are advised to use reference ranges provided by their laboratory.

I. Muscle

A. Creatine kinase (CK)

- Also called creatinine phosphokinase (CPK).
- Specific indicator of muscle damage.
  - Leakage enzyme released when myocytes rupture.
  - CK has a short half-life (hours), so serum concentrations fall quickly after an episode (indicates acute, ongoing muscle damage).
- This enzyme catalyzes the transfer of high-energy phosphate groups from ATP to creatine during exercise, and then the reverse reaction occurs during rest.
  - Mildly increased (< 1000 U/liter):
    - If the horse is recumbent or rolling.
    - Can also be detected after recent exercise or if the horse has just arrived by trailer.
    - If the previous conditions do not apply, then mild exertional rhabdomyolysis (ER) and/or polysaccharide storage myopathy (PSSM) should be suspected.
  - Moderately (> 1000 U/liter) to severely increased (> 10 000 U/liter):
    - If the horse is currently suffering from ER:
      - Urine should be checked for myoglobin.
      - Intravenous fluids should be administered to promote diuresis.
B. Aspartate aminotransferase (AST)
- Previously called serum glutamic oxaloacetic transaminase (SGOT).
- Indicator of muscle damage or liver damage.
  - Leakage enzyme released when myocytes or hepatocytes rupture.
  - Long half-life (days), so serum concentrations fall slowly after an episode.
- This enzyme is involved in amino acid degradation.
- Muscle damage affects AST and CK if the disease process is ongoing.
  - However, serum AST activity will remain increased after CK activity has returned to normal.
- Increased AST activity indicates a previous ER episode or suggests the presence of PSSM.

II. Liver

- Chronic liver diseases such as pyrrolizidine alkaloid toxicosis or cholelithiasis can go undetected unless serum chemistry values are examined.
  - This is particularly true in horses because the finding of icterus is often discounted as a consequence of reduced food intake.
- Presence of one of these diseases may significantly alter the overall prognosis for the patient and should be discussed with the client prior to anesthesia.
- Hepatic dysfunction must be recognized prior to anesthesia because this condition may alter the metabolism of certain anesthetic agents.

A. Gamma glutamyl transferase (GGT)
- Specific indicator of liver damage.
  - Inducible enzyme released when cells become stressed.
  - Bile accumulation (cholestasis) and certain drugs (e.g. phenobarbital) increase serum GGT activity.
  - GGT has a long half-life (days), so serum concentrations fall slowly after an episode.
- This enzyme is found within the membranes of hepatocytes and is most abundant within the biliary epithelial cells.
- It is involved in glutathione metabolism.
- Reference range: 6–32 U/liter.
  - The normal range for burros, donkeys and asses may be 2–3 times higher.
- Cholestasis can result from intra- and extrahepatic causes.
  - Intrahepatic cholestasis accompanies chronic liver diseases such as pyrrolizidine alkaloid toxicosis and cholelithiasis. Acute and subacute liver diseases also cause intrahepatic cholestasis when hepatocytes swell and compress bile ductules.
  - Extrahepatic cholestasis occurs when the common bile duct is occluded by choledoliths, or when bile flow is impaired by inflammation of the bile duct papilla within the duodenum.
- Horses that are accumulating gastric reflux as a result of enteritis may also have increased GGT activities and hyperbilirubinemia because bile is not being transported away by the ingesta.
■ Cholestasis sometimes accompanies displacement of the large colon because the common bile duct courses through the duodenocolic ligament, which becomes stretched.

B. Sorbitol dehydrogenase (SDH)

- Also called iditol dehydrogenase (IDH).
- Requires special handling.
  - SDH is not offered on most routine serum chemistry panels, but can be easily requested.
- Specific indicator of liver damage.
  - Leakage enzyme released when hepatocytes rupture.
  - SDH has a short half-life (hours), so serum concentrations fall quickly.
- This enzyme is found within the cytosol of hepatocytes and plays a role in a glucose metabolism pathway that bypasses glycolysis.
- Reference range: 1–8 U/liter.
- Increased activity indicates ongoing hepatocellular injury because SDH concentrations fall quickly as the disease resolves.

C. Aspartate aminotransferase (AST)

- Found on most serum chemistry panels, so can be evaluated if SDH is not available.
- Indicator of muscle damage or liver damage.
  - Leakage enzyme released when myocytes or hepatocytes rupture.

D. Total bilirubin (TBIL)

- Indicator of hepatic dysfunction, hemolysis, or reduced feed intake.
- Waste product of heme. Aged or defective erythrocytes are removed from circulation by the spleen and heme is catabolized to bilirubin within macrophages.
  - Unconjugated (indirect) bilirubin (UBIL) is released, which circulates in the blood bound to albumin.
  - Circulating UBIL is removed from the blood by the liver and conjugated with glucuronic acid to improve water solubility.
  - Conjugated (direct) bilirubin (CBIL) is excreted in the bile.
- TBIL concentration is commonly reported, but this value may be subdivided into UBIL and CBIL fractions.
- Reference range (TBIL): 0–3.2 mg/dl (0–54.7 μmol/liter).
- Hepatic dysfunction causes UBIL and CBIL concentrations to rise.
- Biliary obstruction (e.g. cholelithiasis) raises the CBIL to UBIL ratio.
- Hemolysis raises the serum UBIL concentration because erythrocytes are either lysed in circulation (intravascular hemolysis), or cleared more rapidly from the blood (extravascular hemolysis). Free hemoglobin is metabolized by hepatocytes.
- Reduced food intake also raises the serum UBIL concentration, but this time as a result of slowed clearance of bilirubin from the blood instead of overproduction. Free fatty acids, released in greater quantities in response to negative energy balance, are thought to compete with UBIL for carrier proteins that facilitate entry into hepatocytes.
E. Serum bile acids (SBA)

- Requires special handling.
- Indicator of hepatic dysfunction.
- Reference range: 0–20 μmol/liter.
- Bile acids are synthesized and secreted by the liver, so it at first seems logical to assume that SBA concentrations decrease as hepatic function declines. However, this is not the case because greater than 90% of bile acids excreted via the bile into the duodenum are subsequently reabsorbed by the intestine and used again by the liver (enterohepatic circulation). Bile acids are removed from the portal blood by hepatocytes, so SBA concentrations increase as hepatic function decreases.
- Only a single blood sample is required instead of pre- and post-feeding samples because the horse does not have a gallbladder and releases bile continuously.

III. Kidneys

- Detection of pre-renal azotemia or renal failure prior to anesthesia alerts the clinician to the need for intravenous fluids and blood pressure support during the procedure.
  - Renal failure affects the prognosis for the patient and should therefore be discussed with the client.

A. Blood urea nitrogen (BUN)

- Indicates that the horse suffers from pre-renal, renal, or post-renal azotemia (this term is also commonly used when serum creatinine concentrations are increased).
- Reference range: 10–25 mg/dl (3.6–8.9 mmol/l).
- BUN is synthesized by the liver and excreted via the kidneys.
- It is a waste product of amino acid catabolism.

Pre-renal azotemia

- Occurs when the glomerular filtration rate (GFR) has been decreased by a reduction in renal perfusion.
- Dehydration and circulatory shock are the most common causes of pre-renal azotemia.
- Prolonged renal hypoperfusion can lead to renal failure, so this problem should be addressed expeditiously.
- When renal function is adequate (pre-renal), azotemia is accompanied by a urine specific gravity (USG) > 1.025 g/ml (i.e. the urine is concentrated).
- Uroperitoneum secondary to bladder rupture in foals also causes pre-renal azotemia.

Renal azotemia

- Occurs when the GFR is low as a result of acute or chronic renal failure.
- Renal azotemia is diagnosed by concurrently measuring the urine specific gravity.
- Renal failure is defined by the presence of azotemia in a patient that cannot concentrate its urine (USG < 1.025 g/ml).
Post-renal azotemia

- Is associated with mechanical (e.g. uroliths) or functional (e.g. neurogenic bladder dysfunction) obstruction of the urinary tract.

B. Creatinine

- Usually examined with BUN (pre-renal, renal, or post-renal azotemia).
- Reference range: 0.4–2.2 mg/dl (35.4–194.5 μmol/liter).
- Creatinine is synthesized from creatine (found in muscle) by a nonenzymatic irreversible reaction at a constant rate and then excreted via the kidneys.
- Is freely filtered by the glomerulus.
  - In contrast with urea nitrogen, creatinine is not reabsorbed within the tubules, so serum creatinine concentrations provide a more accurate measurement of GFR.

IV. Plasma proteins

- Hypoproteinemia cannot be detected upon physical examination of the horse unless subcutaneous edema is observed, or wheezes consistent with pulmonary edema are auscultated.
  - These abnormalities are unlikely to be present when hypoproteinemia is first developing and may only become apparent when intravenous fluids are administered to correct dehydration.
- It is therefore imperative that, at least, the patient’s plasma total protein concentration be examined.
- A refractometer can be used to measure total solids, but it is preferable to examine serum total protein (TP), albumin, and globulin concentrations provided on a serum chemistry panel.
- Albumin and globulin concentrations should be examined individually because hyperglobulinemia can accompany chronic disease in horses and prevent hypoalbuminemia from being detected when only a serum total protein concentration is examined.

A. Total protein

- Reference range (serum): 5.6–7.6 g/dl (56–76 g/l).
- Reference range (plasma): 6.0–8.5 g/dl (60–85 g/l).
- In the author’s experience, most horses fall within a range of 6.0–7.0 g/dl.

B. Albumin

- This protein is synthesized by the liver and has a plasma half-life of 19 days.
- Reference range: 2.6–4.1 g/dl (26–41 g/liter).
- Albumin accounts for 75% of oncotic activity within the plasma.
- Edema develops as consequence of hypoalbuminemia, and the rate of progression depends upon the degree of hypoalbuminemia and how quickly it developed.
  - Generally, plasma or whole blood transfusion is considered when serum or plasma albumin concentrations approach 1.5 g/dl.
Four general causes of hypoalbuminemia

- Low dietary protein intake.
- Reduced synthesis by the liver.
- Excessive catabolism (as occurs with starvation).
- Increased loss from the blood.
  - The most common cause.
  - Examples include:
    - Loss into the lumen of the gastrointestinal tract with bacterial colitis or strangulation of the bowel.
    - Loss into the abdomen with peritonitis.
    - Loss into the thoracic cavity with pleuropneumonia.
    - Loss into subcutaneous tissues as a result of vasculitis.
    - Loss through the glomerulus when damage occurs at this site.

C. Globulins

- Include α, β, and γ globulins.
- These proteins are larger in size than albumin and are synthesized by various cells including hepatocytes (haptoglobin) and plasma cells (IgG).
- Reference range: 2.6–4.0 g/dl (26–40 g/liter).
- Hyperglobulinemia is associated with chronic disease, and should alert the clinician to the presence of a nidus of inflammation such as a tumor or abscess. This finding is unlikely to impact anesthesia, but may affect the overall outcome of the case.
2 The cardiovascular system

Physiology of the cardiovascular system

Tamara Grubb

• The cardiovascular system consists of three components (heart, vessels, blood) whose ultimate goal is to deliver oxygen to the working cells.
• Tissue oxygen delivery (DO₂) is determined by the amount of blood pumped to the cells (cardiac output or ‘Q’) and the oxygen content of the blood (CaO₂).
• Anesthesia can drastically alter cardiovascular function and have a global impact on organ function via decreased DO₂. Thus, a working knowledge of normal cardiovascular function is important.

I. Anatomy

A. Chambers

  o The equine heart is a typical mammalian heart with four chambers: two atria and two ventricles.

Atria

  o Primary function is to receive and store blood that will empty into the ventricles during early diastole.
  o Oxygen-depleted blood from the body is delivered to the right atrium via the cranial and caudal vena cavae and from the myocardium via the coronary sinus and cardiac veins.
  o Oxygen-rich blood from the lungs is delivered to the left atrium via pulmonary veins.

Ventricles

  o Primary function is to pump blood into the high-pressure systemic (left ventricle) and low-pressure pulmonary (right ventricle) circulations.
  o As described by the Law of Laplace, the thick-walled, conical left ventricle is better suited for high-pressure pumping than the thin-walled, flattened, right ventricle.

Atrioventricular valves

  o Connect atria and ventricles.
  o Tricuspid valve between right atrium and ventricle.
  o Mitral valve between left atrium and ventricle.
Semilunar valves
- Connect ventricles to outflow tracts.
- Aortic valve between left ventricle and aorta.
- Pulmonary valve between right ventricle and pulmonary artery.

B. Structural or ‘skeletal’ components of the heart
- Myocardium – muscle layer (striated muscle) of atria and ventricles.
- Endocardium – internal lining of the heart chambers, valves and blood vessels.
- Epicardium – external lining of the myocardium, continuous with pericardium; secretes pericardial fluid.

C. Neural input to the heart
- Atria are highly innervated by sympathetic and parasympathetic fibers.
  - Parasympathetic – decrease rate and contractility.
  - Sympathetic – increase rate and contractility.
- Ventricles are primarily innervated by sympathetic fibers.
  - Continually discharge to maintain a strength of ventricular contraction 20–25% greater than what would occur with no sympathetic input.

II. Cardiac contractions

A. Initiation
- Unlike most systems in the body, neither the autonomic nor motor neurons are necessary for initiating cardiac contractions.
- The heart can continue beating in the absence of outside neural control because the cells of the specialized electrical conducting system of the heart are capable of automatic rhythmical depolarization or ‘self-excitation’. This is due to:
  - Cell membranes that are ‘leaky’ or permeable to sodium.
    ■ Increased permeability to potassium and calcium ions also plays a role in the spontaneous depolarization of the pacemaker cells.
  - A resting cell membrane potential that is not negative enough to keep sodium channels closed.
    ■ The resting membrane potential of cardiac conducting cells is −60 to −70 millivolts (mV) and that of the sinoatrial node is −55 to −60 mV (compared with −90 mV for normal muscle cell membranes).

B. Components of the specialized electrical conducting system
- Sinoatrial (SA) node:
  - Has the fastest rate of spontaneous depolarization and is the pacemaker.
  - Located at the junction of the cranial vena cava and the right auricle.
- Atrioventricular (AV) node:
  - Slows the rate of impulse transmission as it conducts impulses from the atria to the ventricles.
o Internodal pathways:
  – Conduct impulses through the atria to the AV node.
o Right and left bundle branches and His–Purkinje system.
  – Conduct impulses throughout ventricles and ventricular septum.

### III. Unique features of the equine heart

- Large SA node.
  - A wandering pacemaker is common.
    - Seen as *variably shaped P waves* on electrocardiogram (ECG).
- Large atria that may depolarize slightly asynchronously.
  - Result in *biphasic P waves* on the ECG.
- Deeply penetrating His–Purkinje system.
  - Facilitates movement of electrical impulses throughout the large ventricular muscle.
    - Often called Type II Purkinje system.
- Ossa cordi.
  - In all species there is a connective tissue ‘skeleton’ that separates the atria from the ventricles.
  - In cattle and in older horses, these structures may ossify and create two bones, the ‘ossa cordi’.

### IV. Circulatory systems

- The *systemic* (high pressure) and *pulmonary* (low pressure) circulatory systems are separate but coupled (in series) and interdependent so that dysfunction of one will lead to dysfunction of the other.
- The circulatory systems are not mere conduits; through *dilation* and *constriction*, they control distribution of blood throughout the body and in localized tissue beds.
- The lymphatic system is often included as a component of the circulatory system.

#### A. Components of the systemic circulation

| Aorta → Arteries → Arterioles → Capillaries → Venules → Great veins → Right atrium |

- The elastic wall of the *aorta* recoils following ventricular contraction, creating a force that maintains blood flow throughout both systole and diastole.
- *Arterioles* provide the greatest resistance to circulation and, via dilation or constriction, control blood flow to each tissue capillary bed.
- *Capillaries* are the site of exchange of nutrients and waste products.
- The majority of the circulating blood volume (approximately 80%) is generally ‘stored’ in the *venules* and *great veins*. 
B. Components of the pulmonary circulation

- The pulmonary artery is the only artery in the body that carries deoxygenated blood, and the pulmonary vein is the only vein that carries oxygenated blood.
- Although the pulmonary circulation receives the same cardiac output as the systemic circulation, the pulmonary system remains a low-pressure system due to the:
  - Tremendous distensibility of the thin-walled vessels.
  - Large number of vessels that aren’t normally perfused but which can be recruited in times of increased output.
- Distribution of pulmonary blood vessels is an important component of ventilation/perfusion (V/Q), distensibility and gas exchange.
- Unlike most tissues in the body, pulmonary tissues constrict when hypoxic (hypoxic pulmonary vasoconstriction) in an attempt to divert blood away from poorly ventilated alveoli.
  - This phenomenon can contribute to V/Q mismatch, especially during anesthesia.
- The lung also receives blood flow through the bronchial circulation, a branch of the systemic circulation that perfuses the tissues of the respiratory system.

C. Blood

- Consists of plasma and cellular components.
- Normal equine hematocrit or packed cell volume (PCV) is approximately 35–45% and normal hemoglobin is approximately 15 g/dl.
  - Most oxygen is transported bound to hemoglobin. (See section on DO₂.)
  - When saturated, equine hemoglobin binds 1.36–1.39 ml of oxygen per gram of hemoglobin.

V. Cardiovascular physiology

- The cardiac cycle can be described as a period of ventricular contraction (systole) followed by ventricular relaxation (diastole).
- The electrical, mechanical and audible events that occur during the cardiac cycle are depicted in the Wiggers diagram (see Fig. 2.1) and described below.

A. Events occurring during late diastole

- The cardiac cycle begins with the spontaneous discharge of the pacemaker, the SA node.
- Discharge is followed quickly by electrical activation of the right atrial muscle and then the left atrial muscle.
  - This results in the P wave on the ECG.
  - Passive filling of the ventricles occurs during this period.
  - Because electrical activation always precedes mechanical activity (termed the electromechanical delay), the actual atrial contraction occurs shortly after the P wave is generated.