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Cardiovascular Emergencies

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Dysrhythmias – Diagnosis and Treatment

Emergency Triage Steps

Recognition of Dysrhythmias – Bradycardias, Tachycardias (How to Read ECGs)

Assessment of the cardiovascular system is an essential part of the physical examination in a critically ill patient. Frequently patients present with tachycardia or tachyarrhythmia while occasionally we see patients with pathological bradycardia. It is important to be able to differentiate a sinus rhythm from an atrial, junctional, or ventricular rhythm (Figures 12.1a–c, 12.2 and 12.3a,b). Recording an electrocardiogram (ECG) is key to determining if the rhythm is normal or abnormal which will then guide treatment. The process of how to record an ECG is covered in Chapter 2. Table 12.1 presents the author's approach to analyzing an ECG (Mitchell 2019, 2020). A case example of a patient presenting with a tachyarrhythmia is found in Appendix 12.3 (online).

Treatment will vary depending on the origin of the rhythm (atrial, junctional, or ventricular) and the heart rate. In the emergency situation, treatment is focused on (1) **improving cardiac output**, (2) **controlling the ventricular rate,** and (3) **converting the rhythm back to a sinus rhythm** (Box 12.1).

It is also important to decide if there is underlying structural cardiac disease leading to the dysrhythmia, if the dysrhythmia is secondary to systemic disease (e.g., systemic inflammatory response syndrome [SIRS], hemorrhage or electrolyte derangements) or if the dysrhythmia is considered a "primary" problem. It will be difficult to control the rhythm if there is severe underlying structural disease while treatment of any systemic problems will play an important role in restoration of a sinus rhythm (van Loon 2019).

Patient Stabilization An intravenous (IV) catheter should be placed and IV fluids considered if there is evidence of poor cardiac output (high heart rate, azotemia, poor peripheral perfusion, abnormal mentation). Large volumes of IV fluids should be avoided until it can be determined if myocardial dysfunction or cardiac failure is present, but a **targeted approach to fluid therapy** with small boluses (e.g., **10 mL/kg**) can be helpful and repeated if necessary.

Diagnostic Plan An ECG is *essential* to accurately diagnose the rhythm present and for monitoring the response to therapy. Point-of-care blood work to assess hydration, perfusion, and monitor for cardiac injury is indicated (PCV/TP, lactate, creatinine, electrolytes, and cardiac troponins). A focused rapid assessment of the cardiovascular structures (EFATE, CRASH, or similar protocol) (Bevevino et al. 2023; Eberhardt and Schwarzwald 2022) to investigate for underlying structural disease and monitor cardiac function should be performed. Non-invasive blood pressure (NIBP) measurements from the tail can be useful to monitor and guide therapy.

Emergency Treatment Guidelines The indications for treatment of dysrhythmias are found in Table 12.2. Rate and rhythm control should be attempted if there is evidence of **hemodynamic instability**, **rapid ventricular rate (>100 bpm)**, **extreme bradycardia (<20 bpm)**, **polymorphic or multiform QRS complexes**, **evidence of R-on-T phenomena,**

Emergency Triage of the Horse, First Edition. Edited by Rolfe M. Radcliffe and Jamie J. Kopper.

Companion website: www.wiley.com/go/radcliffe/triage

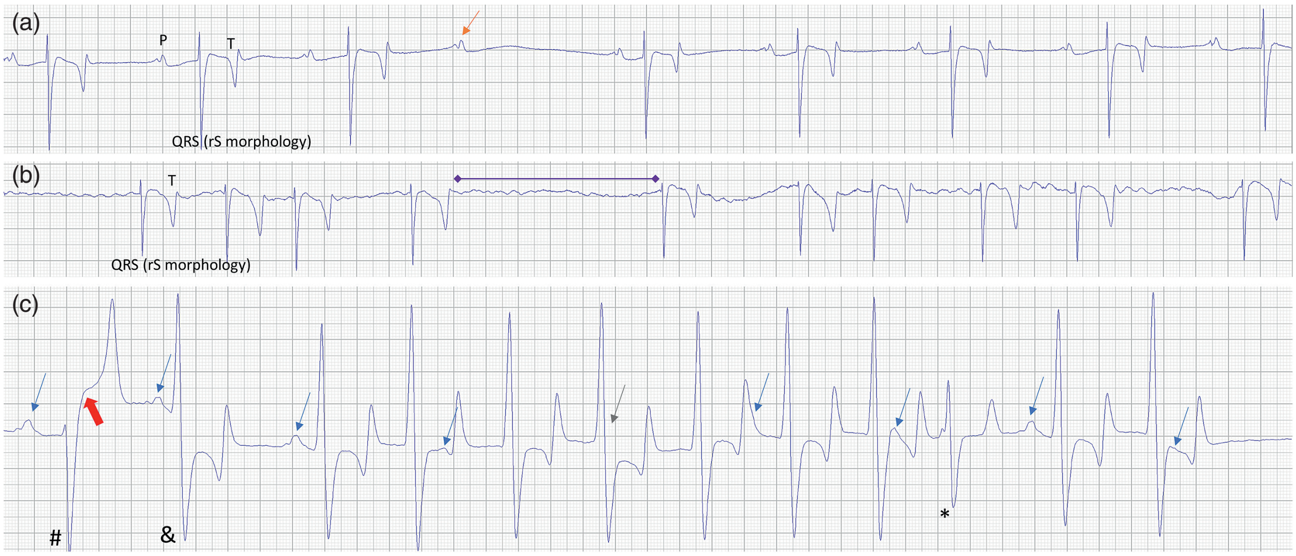

Figure 12.1 (a) A modified base–apex ECG recording from a horse in normal sinus rhythm (showing the typical equine short R, tall S [rS] QRS morphology). The baseline rhythm is regular until **a second-degree AV block is observed** (orange arrow), where there is a P wave but no subsequent QRS-T. Progressive lengthening of the PQ interval is also observed in the last 5 complexes. The atrial rate is 34/min while the ventricular rate is 30/min. Paper speed 25 mm/s, gain 10 mm/mV. (b) A modified base–apex ECG recording from a horse with an irregularly irregular rhythm. The RR interval is constantly changing, no P waves are observed, but the QRS-T morphology is very appropriate and similar between complexes (showing the typical equine rS morphology). An undulating baseline is present – where fibrillation (f) waves are observed (purple line). **This is atrial fibrillation (AF)**. The ventricular response rate is appropriate (approx. 38/min). Paper speed 25 mm/s, gain 10 mm/mV. (c) A modified base–apex ECG recording from a horse with a tachyarrhythmia. The RR interval is variable and the QRS-T morphology differs (three different QRS-T morphologies are present, marked #, &, *). P waves are present but there are variable PQ intervals and P waves are seen wandering in and out of the QRS-T complexes, indicating AV dissociation (observed P waves – blue arrows, presumed hidden in QRS – gray arrow). The ventricular rate is 90/min while the atrial rate is 68/min. **This is a multiform (or polymorphic) accelerated idioventricular rhythm (sometime called slow ventricular tachycardia)**. The red arrow indicates ST segment elevation, which can be a marker for myocardial ischemia in other species. Paper speed 50 mm/s, gain 10 mm/mV.

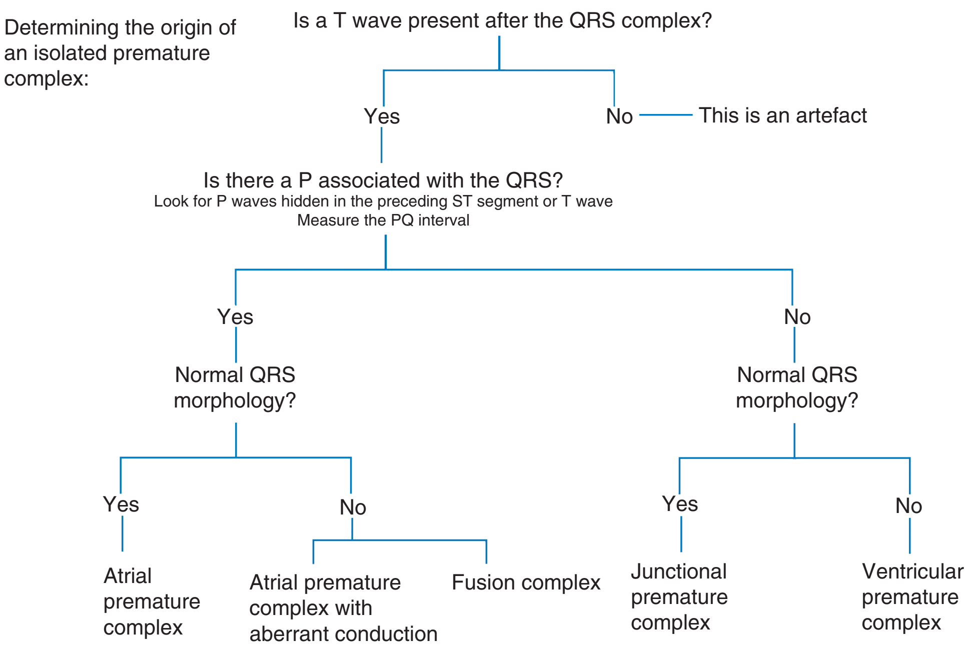

Figure 12.2 Decision tree algorithm to determine the origin of a premature complex identified on an ECG recording. A fusion complex is a normal complex fused with a complex of ventricular origin. Determining the origin of the rhythm is vital for implementing the most appropriate anti-arrhythmic therapy.

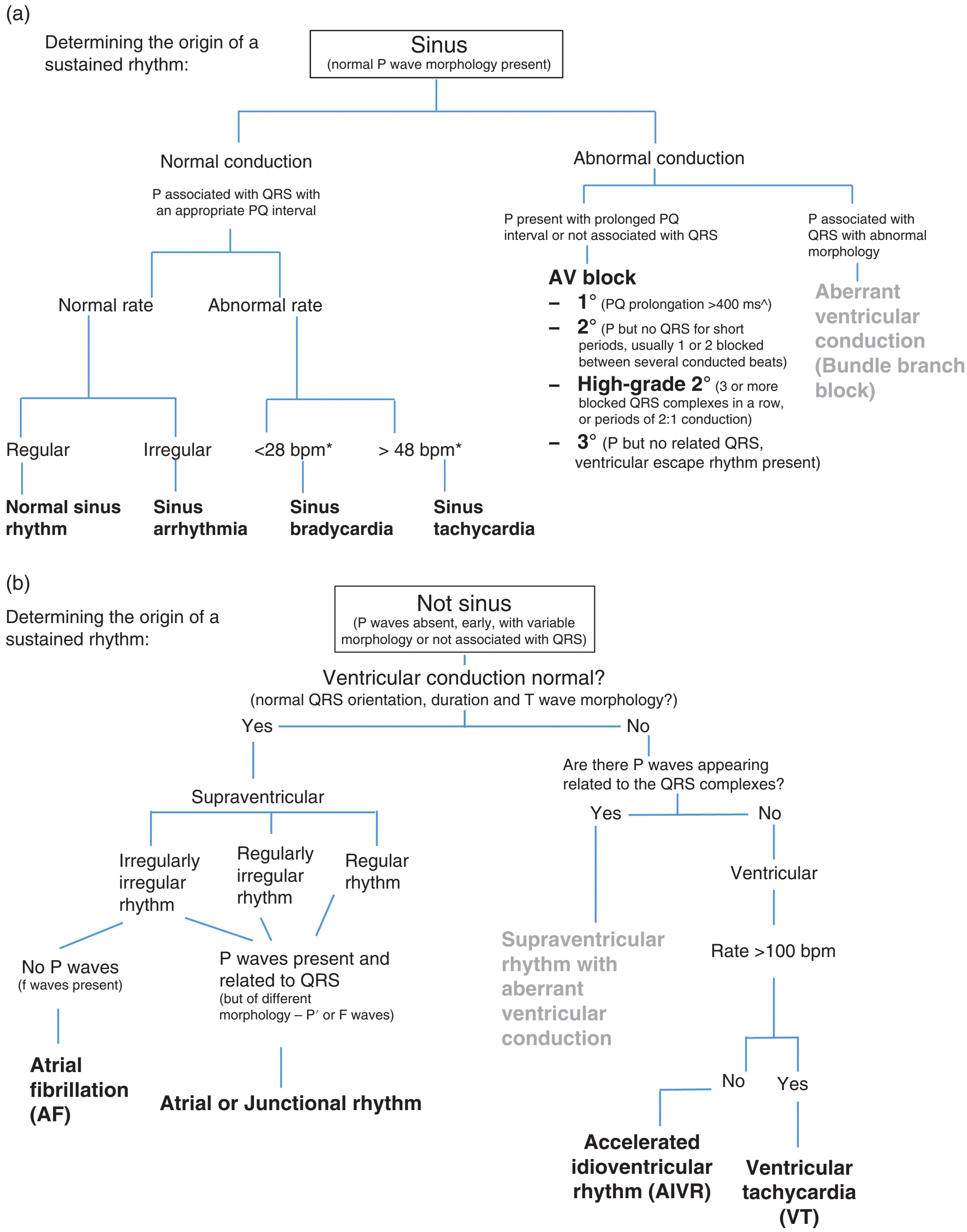

Figure 12.3 Decision tree algorithm to determine the origin of a sustained rhythm identified on an ECG recording. Determining the origin of the rhythm is vital for implementing the most appropriate anti-arrhythmic therapy. (a) If normal P and QRS-T wave morphology is present, the rhythm is considered likely originating from the sino-atrial node. ^, the PQ interval is dependent on size/body weight, a cut-off of 400 ms is appropriate for a 500 kg horse. *, the HR is dependent on size/bodyweight, these cut-offs apply to the average-sized 500 kg horse. (b) If the rhythm appears to originate from a non-sinoatrial node location, it may be supraventricular (atrial or junctional) or ventricular in origin. AV, atrioventricular; AF, atrial fibrillation; VT, ventricular tachycardia; AIVR, accelerated idioventricular rhythm; F, flutter; 1°, 1st degree; 2°, 2nd degree; 3°, 3rd degree; ms, milliseconds; bpm, beats per min.

Table 12.1 A step-by-step approach to analyze an ECG and recognize abnormalities.

Step 1.	Assess the overall quality of the recording. • The P-QRS-T complexes should be clearly visible and free of obvious artifacts. • Analysis should not be performed if the recording quality is poor, as misdiagnosis of the rhythm is possible.
Step 2.	Identify the paper speed (typically 25 or 50 mm/s) and calculate the overall heart rate. The heart rate can be divided into three broad categories, normal (28–48 bpm), bradycardic (<28 bpm), or tachycardic (>48 bpm). Most digital systems will perform an RR interval analysis and calculate the instantaneous heart rate automatically.
Step 3.	Assess if the rhythm is regular or irregular. • If the rhythm is irregular, is there any underlying pattern (regularly irregular) or is there no pattern (irregularly irregular)? • Are there pauses, premature complexes, or both?
Step 4.	Identify the most normal P-QRS-T complexes (if present) and then compare them to the abnormal complexes. • Is there a P for every QRS? • Is there a QRS for every P? • Do they appear related (similar PR intervals) or dissociated (varying PR intervals)? • Is the morphology of all P-QRS-T complexes similar or different? • Are the QRS complexes of similar polarity and duration (ms). With the modified base–apex lead placement (described in Chapter 4) normal horses should have an rS or S morphology. • Are all T waves the same or is there variation in T wave morphology that could be caused by secondary T wave changes (i.e., if the preceding QRS is abnormal, then likely the following T wave is abnormal as well) or superimposition of other waves (e.g., a P wave within the preceding T wave)? • Identify if any R-on-T phenomena present
Step 5.	Measure the PR, QRS, and QT intervals. • Are they similar or varying?
Step 6.	Define the rhythm. • Is this a disorder of abnormal impulse formation, abnormal impulse conduction, or both? • Is the rhythm primarily sinus in origin? If no, are the abnormal complexes of **atrial**, **junctional**, or **ventricular** origin? See Figures F1–3. • Is the rhythm sustained, paroxysmal, or intermittent? • What is the timing and frequency of occurrence of abnormal beats?
Step 7.	If unsure about the rhythm diagnosis or interpretation of the clinical relevance, seek a second opinion from a person experienced at equine ECG interpretation.

Box 12.1 Treatment goals for emergency stabilization

- Improving cardiac output.
- Controlling the ventricular rate.
- Converting the rhythm back to a sinus rhythm.

or ***torsade de pointes*** as these findings indicate a high risk for the rhythm becoming life-threatening. In most cases, treatment in the field is difficult to achieve due to the lack of availability of appropriate monitoring and medication. Therefore, referral to a hospital facility is frequently indicated in these cases.

Controlling the Ventricular Rate It can be difficult to determine if a rhythm is sinus or non-sinus in origin, particularly at high ventricular rates. In these cases, **attempting to slow the ventricular rate** can be helpful to determine the presence of P waves, associations between P and QRS complexes, and to measure the atrial rate. In the short term (and in the absence of fulminant heart failure), small doses of an **alpha 2 agonist** (e.g., xylazine 0.25 mg/kg IV) can facilitate slowing of the AV conduction for a short period of time, allowing a more accurate ECG diagnosis. Some horses with tachyarrhythmias will be

Table 12.2 When to treat a dysrhythmia – indications for treatment.

- Is there evidence of hemodynamic instability?
  - Poor peripheral perfusion (cold extremities, weak pulses).
  - Delayed jugular filling, jugular pulsation, jugular distension.
  - Prolonged capillary refill time.
  - Reduced urine output.
  - Dull mentation.
  - Weakness.
  - Collapse/syncope/recumbency.
  - Low systemic blood pressure.
  - Laboratory evidence of reduced perfusion (e.g., hyperlactatemia or azotemia).
- Ventricular rate is consistently high (>100 bpm) or extremely low (<20 bpm).
- Polymorphic or multiform ventricular complexes.
- Evidence of impaired cardiac function on echocardiography.
- *R-on-T phenomena*.
- *Torsade de pointes*.

quite anxious and this can contribute to their rapid ventricular rate, so small doses of **acepromazine** (0.005–0.01 mg/kg IV) can help reduce anxiety and sympathetic stimulation, therefore slowing ventricular conduction (measure NIBP first and don't use if profound hypotension already present). It the rhythm is determined to be supraventricular in origin, attempts can be made to slow AV node conduction for longer periods of time (e.g., **digoxin or sotalol**, see Table 12.3) to improve cardiac output and hemodynamic stability until rhythm control can be attempted.

In cases of bradycardia, attempts should be made to **increase the sinus rate** and **improve AV node conduction**. The use of medication is usually a temporary measure, with the definitive treatment being placement of a pacemaker if no underlying structural reason for the bradycardia is detected and the bradycardia does not resolve. The medications used to treat bradycardia are summarized in Table 12.4. In some cases, a temporary pacemaker lead may need to be placed intravenously, prior to permanent pacemaker implantation (van Loon et al. 2020).

Controlling the Rhythm Once the horse is stabilized and the origin of the rhythm determined, efforts can be made to convert the horse back into a sinus rhythm. While there are many anti-arrhythmic medications available, the practicalities and monetary considerations of treating horses often weighing in excess of 500 kg, make the list considerably shorter. It should be remembered that most anti-arrhythmic medications have the potential to be pro-arrhythmic. Continuous ECG monitoring during therapy is important to recognize adverse events and detect changes in the underlying rhythm.

For confirmed supraventricular dysrhythmias, there is also the possibility of transvenous electrical cardioversion (TVEC) in addition to pharmacological conversion. This can be performed at some referral institutions, and should only be considered once the patient has been stabilized and it has been determined that there are minimal underlying structural changes (Decloedt et al. 2021; Van Steenkiste et al. 2019).

Most ventricular tachyarrhythmias will require treatment, but some of the slower accelerated idioventricular rhythms can be monitored and the abnormal rhythms will resolve once the underlying systemic disease (e.g., hemorrhage, SIRS, endotoxemia, electrolyte derangements) have been treated (Mitchell 2017; Navas de Solis 2020; Williams and Mitchell 2023).

Important Decisions During Management

Decision for Hospital Referral

If the patient is hemodynamically unstable, then rapid referral is usually indicated, as these patients require intensive treatment and ongoing monitoring. Most referral facilities have access to a larger range of medication and the ability to provide continuous monitoring.

Table 12.3 Treatment options for horses with supraventricular or ventricular dysrhythmias.

Drug	Drug class	Dose recommendations	Dose for 500 kg bwt	Comments
First-line medication for short duration *rate* control if the rhythm is *supraventricular*				
Xylazine	α_2 agonist	0.25 mg/kg bwt slowly IV, repeat once if required	125 mg IV over 2–3 min	Adverse effects: muscle tremors, bradycardia with AV blocks, reduced respiratory rate, sweating, and ataxia. Contraindications: Preexisting cardiac dysfunction, hypotension or shock, pathological ventricular dysrhythmias, respiratory dysfunction, severe hepatic or renal insufficiency, and seizures.
Diltiazem	Class IV Ca^{2+} channel blocker	0.125 mg/kg over 2 min IV, repeated every 10 min to effect, up to 1.25 mg/kg total dose	62.5 mg IV over 2 min; total dose 625 mg	Titrate to effect. Use diltiazem doses >0.5–1.0 mg/kg with caution. Adverse effects: Hypotension, tachycardia, sinus arrhythmia, bradycardia, sinus arrest, high-grade AV block, negative inotropism, and exacerbation of heart failure (unless secondary to SVT or AF with rapid ventricular response). Contraindications: Hypotension, bradycardia, SA or AV block, ventricular systolic dysfunction, severe heart failure, cardiogenic shock, and β blocker.
Esmolol	Class II β_1 blocker	50–100 μg/kg bwt IV slowly over 3–5 min, up to 500 μg/kg total dose	25–50 mg IV slowly	Ultra-short acting, use incremental doses up to a total dose of 500 μg/kg. Adverse effects: hypotension, bradycardia, and collapse. Contraindications: weakness, bradycardia, AV block, hypotension, negative inotropism, and exacerbation of heart failure.
Metoprolol	Class II β_1 blocker	0.02–0.06 mg/kg bwt IV q4–6 h	10–30 mg IV slowly	Short acting (hours). Adverse effects and contraindications: see Esmolol.
First-line medication for longer duration *rate* control if the rhythm is *supraventricular*				
Digoxin	Digitalis glycosides	0.0022 mg/kg q12 h IV 0.011 mg/kg q12 h PO	1.1 mg IV 5.5 mg PO	TDM: peak (1–2 h) and trough (12 h) concentrations at steady state should fall within 0.8–2.0 ng/mL (1–2.6 nmol/L). Adverse effects: Depression, anorexia, colic, diarrhea, sinus bradycardia, AV block, and supraventricular and ventricular dysrhythmias (bigeminy). Contraindications: AV block, diastolic ventricular dysfunction, pre-existing digitalis toxicity, myocarditis, and ventricular dysrhythmias.
Sotalol	Class II/III K^+ channel and β blocker	1 mg/kg bwt PO q12 h for 1 d, continue at 2–3 mg/kg bwt PO q12 h	500 mg initial dose 1–1.5 g per maintenance dose	Good oral bioavailability. Generally well tolerated, even with chronic oral administration. Dosage should be gradually reduced before discontinuing medication. Adverse effects: QT prolongation, ventricular dysrhythmias, and sweating. Contraindications: Preexisting QT prolongation. Use with caution in patients with reduced-poor systolic function, uncorrected hypokalemia or hypomagnesemia.

Table 12.3 (Continued)

Drug	Drug class	Dose recommendations	Dose for 500 kg bwt	Comments
Atenolol	Class II β_1 blocker	0.12 mg/kg bwt PO q12 h initially increasing to 0.25 mg/kg bwt PO q12 h if well tolerated. Can increase up to 1 mg/kg bwt PO q12 h if needed.	60 mg per dose initially, then 125 mg per dose	Variable bioavailability. Dosage should be adjusted on an individual basis. Dosage should be gradually reduced before discontinuing medication. Adverse effects: Sweating, depression, lethargy, weakness, bradycardia, AV block, hypotension, negative inotropism, and exacerbation of heart failure. Contraindications: Bradycardia, high-degree AV block, and untreated heart failure.
Propranolol	Class II β_1/β_2 blocker	0.38–0.78 mg/kg bwt PO q8 h	190–390 mg per dose	Variable bioavailability. Dosage should be adjusted on an individual basis. Dosage should be gradually reduced before discontinuing medication. Adverse effects: Depression, lethargy, weakness, bradycardia, AV block, hypotension, negative inotropism, exacerbation of heart failure, and bronchoconstriction (aggravation of recurrent airway obstruction). Contraindications: Bradycardia, high-degree AV block, untreated heart failure, and bronchopulmonary disease.
First-line medication for *rhythm* control – atrial fibrillation – supraventricular tachycardia (confirmed)				
Quinidine sulfate	Class I_A Na^+ channel blocker	22 mg/kg bwt PO by NGT q2 h for up to four doses or until converted, adverse or toxic effects, or plasma quinidine concentration >4 µg/mL. Do not exceed six doses PO q2 h. Continue q6–8 h until converted, adverse or toxic effects, or total dose of 180 mg/kg. **Stop therapy if:** QRS duration exceeds more than 25% of its pretreatment value, plasma quinidine exceeds 5 µg/mL, significant adverse reaction or signs of toxicity are observed.	11 g per dose	*Use with caution* Adverse effects: Commonly, depression, diarrhea, colic, ataxia, and nasal mucosal swelling. Less commonly paraphimosis, urticarial, and laminitis. Accelerated AV conduction, tachycardia, QRS and QT prolongation, VT, *torsade de pointes*, hypotension, negative inotropism, exacerbation of heart failure, cardiovascular collapse, and sudden death. TDM: Therapeutic range 2–5 µg/mL [6.2–15.4 µmol/L] 1–2 h after PO administration. Contraindications: Ventricular tachyarrhythmias, *torsade de pointes*, untreated heart failure, preexisting QRS or QT interval prolongation, complete AV block, and digitalis intoxication. Use with caution in patients with hypokalemia, hypomagnesemia, hypoxia, or acid-base disorders. If therapy with quinidine sulfate is chosen, an IV catheter should be placed and the emergency drugs required to treat any adverse effects should be available immediately adjacent to the stall, with the appropriate drug doses calculated for the patients weight. Transvenous electrical cardioversion (TVEC) could be considered in cases where quinidine sulfate therapy is contraindicated, and there is no underlying relevant structural heart disease or heart failure.

(*Continued*)

Table 12.3 (Continued)

Drug	Drug class	Dose recommendations	Dose for 500 kg bwt	Comments
Second-line medication for *rhythm* control – atrial fibrillation – supraventricular tachycardia (confirmed)				
Quinidine gluconate	Class I_A Na^+ channel blocker	1–2.2 mg/kg bwt IV q10 min or 0.1–0.22 mg/kg bwt/min CRI up to 12 mg/kg bwt total dose	500 mg–1.1 g q10 min or 50–110 mg/min CRI; total dose 6 g	*Use with caution* See above. For use in acute cases only (<7 day duration). #Not available in all regions of the world.
Amiodarone	Class III K^+ channel blocker	5 mg/kg bwt/h IV for 1 h, followed by 0.83 mg/kg bwt/h for 23 h, subsequently 1.9 mg/kg bwt/h for 30 h or to effect	2.5 g for first hour, then 415 mg/h for the next 23 h, then 950 mg/h for additional 30 h	Also class I, II, and IV effects. Adverse reactions: Hind limb weakness, weight shifting, *torsade de pointes*, SA and AV nodal inhibition, bradycardia, and hypotension. Prolonged treatment may affect the lungs, liver, heart, thyroid gland, GI tract, eyes, skin, and nerves. Contraindications: Sino-atrial node dysfunction, bradycardia, AV block, and cardiogenic shock.
Procainamide	Class I_A Na^+ channel blocker	1 mg/kg bwt/min IV, up to 20 mg/kg bwt total dose	500 mg/min; total dose 10 g	For use in acute cases only (<7 day duration). Adverse events: Hypotension, QRS and QT prolongation, negative inotropism, dysrhythmia, GI, and neurologic disorders (similar to but generally less severe than quinidine). Contraindications: Untreated heart failure, prolonged QRS or QT interval, complete AV block, and digitalis intoxication. Use with caution in patients with hypokalemia, hypomagnesaemia, or acid-base disorders.
Propafenone	Class I_C Na^+ channel blocker	0.5–1.0 mg/kg bwt in 5% dextrose slowly IV over 5–10 min 2 mg/kg bwt PO q8 h	250–500 mg IV over 5–10 min 1 g PO q8 h	Not commonly available. Adverse effects: GI and neurologic disorders, bronchospasm, neg. inotropism, exacerbation of heart failure, AV block, QRS and QT prolongation, and dysrhythmias. Contraindications: Structural heart disease, heart failure, and SA or AV node dysfunction.
First-line medications (intravenous) for *rhythm* control if the rhythm is *ventricular*				
Lidocaine (lignocaine)	Class I_B Na^+ channel blocker	0.25–0.5 mg/kg bwt slow IV, repeat in 5–10 min to effect, up to 1.5 mg/kg bwt total dose; followed by 0.05 mg/kg bwt/min CRI	125–250 mg per bolus up to total dose of 750 mg; CRI 25 mg/min	Adverse effects: Uncommon at therapeutic doses. Overdose can lead to ataxia, muscle tremors, CNS excitement, dysrhythmias, and collapse. Contraindications: SA, AV or intraventricular block, and bradycardia. Caution with hypovolemia, liver disease, shock, and heart failure.
Magnesium sulfate	Physiologic Ca^{2+} and K^+ channel blocker. Activator of membrane Na^+/K^+ ATPase	2–6 mg/kg bwt/min IV to effect, up to 55–100 mg/kg bwt total dose	1–3 g/min; total dose 27–50 g	Adverse effects: Rare, but overdose may lead to CNS depressant effects, weakness, trembling, bradycardia, and hypotension. Very high doses cause neuromuscular blockade with respiratory depression and cardiac arrest. Contraindications: Bradycardia, SA and AV block, and renal failure.

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Table 12.3 (Continued)

Drug	Drug class	Dose recommendations	Dose for 500 kg bwt	Comments
Second-line medications (intravenous) for *rhythm* control if the rhythm is *ventricular*				
Procainamide	Class I_A Na^+ channel blocker	1 mg/kg bwt/min IV, up to 20 mg/kg bwt total dose	500 mg/min; total dose 10 g	As described above.
Amiodarone	Class III K^+ channel blocker	5 mg/kg bwt/h IV for 1 h, followed by 0.83 mg/kg bwt/h for 23 h, subsequently 1.9 mg/kg bwt/h for 30 h or to effect	2.5 g for first hour, then 415 mg/h for the next 23 h, then 950 mg/h for additional 30 h	As described above.
Propafenone	Class I_C Na^+ channel blocker	0.5–1.0 mg/kg bwt in 5% dextrose slowly IV over 5–10 min	250–500 mg IV over 5–10 min	As described above.
Metoprolol	Class II β_1 blocker	0.02–0.06 mg/kg bwt IV q4–6 h	10–30 mg per dose	As described above.
Quinidine gluconate	Class I_A Na^+ channel blocker	1–2.2 mg/kg bwt IV q10 min or 0.1–0.22 mg/kg bwt/min CRI up to 12 mg/kg bwt total dose	500 mg–1.1 g q10 min or 50–110 mg/min CRI; total dose 6 g	As described above.
Bretylium tosylate	Class III K^+ channel blocker	3–5 mg/kg bwt IV, repeat up to 10 mg/kg bwt total dose	1.5–2.5 g per dose; total dose 5 g	# Difficult to obtain. Use in cases of refractory life-threatening VT/VF. Also indirect anti-adrenergic effects. Adverse effects: Excitement, GI disorders, hypotension, tachycardia, and dysrhythmias. Contraindications: Preexisting hypotension.
Second-line medications (oral) – *rhythm* control if the rhythm is *ventricular*				
Sotalol	Class II/III K^+ channel and β blocker	1 mg/kg bwt PO q12 h for 1 d, continue at 2–3 mg/kg bwt PO q12 h	500 mg initial dose 1–1.5 g per maintenance dose	As described above.
Phenytoin	Class I_B Na^+ channel blocker	20 mg/kg bwt PO q12 h for three to four doses or until signs of sedation, followed by 10–15 mg/kg bwt PO q12 h maintenance dose	10 g loading dose 5–7.5 g per maintenance dose	Maintenance dose varies between horses. TDM: 5–10 μg/mL. Adverse effects: Sedation, lip and facial twitching, gait deficits, excitation, seizures, and dysrhythmias. Hepatotoxicity with chronic use. Contraindications: SA or AV block and sinus bradycardia.
Propafenone	Class I_C Na^+ channel blocker	2 mg/kg bwt PO q8 h	1 g per dose	As described above.
Propranolol	Class II β_1/β_2 blocker	0.38–0.78 mg/kg bwt PO q8 h	190–390 mg per dose	As described above.
Metoprolol	Class II β_1 blocker	2–4 mg/kg bwt PO q8–12 h	1–2 g per dose	As described above.
Quinidine sulfate	Class I_A Na^+ channel blocker	22 mg/kg bwt PO by NGT q2 h for up to four doses or until converted, adverse or toxic effects, or plasma quinidine concentration >4 μg/mL. Do not exceed six doses PO q2 h.	11 g per dose	*Use with extreme caution* As described above.

AF, atrial fibrillation; AV, atrio-ventricular; Bwt, bodyweight; CNS, central nervous system; CRI, continuous rate infusion; GI, gastrointestinal; IV, intravenous; kg, kilogram; NGT, nasogastric tube; PO, per oral; SA, sino-atrial; SVT, supraventricular tachycardia; TDM, therapeutic drug monitoring; VF, ventricular fibrillation; VT, ventricular tachycardia.

Table 12.4 Therapeutic interventions for bradycardia.

Drug	Drug class	Dose recommendations	Dose for 500 kg bwt	Comments
Bradyarrhythmias causing hypotension (sinus arrest, sinus bradycardia, high-grade or complete AV block). These medications should be used to provide confirmation of the dysrhythmia (based on a failure to respond appropriately to the medication) and to provide temporary therapy until a longer-term solution can be found (i.e., pacemaker implantation).				
Dobutamine	β_1-adrenergic, β_2- and α_1-adrenergic Preferred over dopamine	1–5 µg/kg bwt/min CRI, titrate to effect or adverse reaction	500–2500 µg/min CRI	Adverse effects: Tachycardia, ventricular dysrhythmias, vasoconstriction, and hypertension (at doses >4 µg/kg bwt/min). Contraindications: Ventricular dysrhythmias, tachycardia, and atrial fibrillation (risk of severe tachycardia due to accelerated atrio-ventricular conduction).
Dopamine	β_1 -adrenergic, dose dependent dopaminergic, and α1-adrenergic	1–5 µg/kg bwt/min CRI, titrate to effect or adverse reaction	500–2500 µg/min CRI	
N-butylscopolammonium bromide (or hyoscine N-butylbromide)	Anticholinergic (vagolytic)	0.05–0.2 mg/kg bwt IV	25–100 mg IV	Short duration of action (30 min). Adverse effects: Transient tachycardia, decreased GI motility, and mydriasis. Contraindications: Tachycardia, tachyarrhythmia, heart failure, and GI disease. Do not use in animals with glaucoma.
Atropine	Anticholinergic (vagolytic)	0.01–0.02 mg/kg bwt IV or IM	5–10 mg IV or IM	Adverse effects: Constipation, ileus, colic, bradycardia (at very low doses), tachycardia, dysrhythmias, and CNS effects (stimulation, drowsiness, ataxia, seizures, respiratory depression). Glycopyrrolate is slightly less arrhythmogenic and rarely results in CNS effects. Contraindications: Tachycardia, tachyarrhythmia, heart failure, GI disease, and colic.
Glycopyrrolate	Anticholinergic (vagolytic)	0.005–0.01 mg/kg bwt IV	2.5–5 mg IV	
Theophylline	Phosphodiesterase and adenosine receptor inhibitor	12 mg/kg bwt PO loading dose, then 5 mg/kg bwt PO q12 h Sustained release formulation: 20 mg/kg bwt PO loading dose, then 15 mg/kg bwt PO q24 h	6 g PO loading dose, then 2.5 g PO q12 h 10 g PO loading dose, then 7.5 g PO q24 h	Positive chronotropic and inotropic medication. Adverse effects: narrow therapeutic window; tachycardia, tachyarrhythmias, CNS excitation, and seizures. Contraindications: do not use with erythromycin, cimetidine, or fluoroquinolones.

AV, atrio-ventricular; Bwt, bodyweight; CNS, central nervous system; CRI, continuous rate infusion; GI, gastrointestinal; IV, intravenous; kg, kilogram.

Decision for Medical vs Surgical Treatment

Cardiac dysrhythmias are treated medically, although they may occur in some surgical patients, particularly while undergoing anesthesia. Recognition and appropriate treatment of cardiac dysrhythmias in these surgical patients is critical to improving their morbidity and mortality while undergoing anesthesia or during recovery.

How to Manage Important Complications

Complications of dysrhythmias result from decreased cardiac output or deterioration of the rhythm to a life-threatening and untreatable dysrhythmia (e.g., ventricular fibrillation). It is important to **maximize cardiac output** by **optimizing preload**, **normalizing the heart rate,** and **improving systolic function** while monitoring for dangerous rhythms (e.g., short-coupling intervals, R-on-T phenomena, QT prolongation).

Estimating Prognosis and Decision for Euthanasia

If the rhythm deteriorates rapidly, sudden death is possible. If there is evidence of severe underlying structural cardiac disease the prognosis for long-term survival is considered guarded and treatment is not recommended in most situations. It is possible that attempted conversion to a sinus rhythm in cases that have substantially impaired cardiac function will precipitate the development of congestive heart failure (CHF). Euthanasia should be considered in patients with signs of congestive heart failure that are refractory to emergency treatment or when the clinical condition of the horse becomes dangerous to itself and those handling it (e.g., frequent episodes of collapse).

What Can Be Done on Farm?

Most important is recognition of a cardiovascular abnormality on clinical examination. If there is the possibility to quickly record an ECG of good quality to confirm the diagnosis this is a bonus. In most of these cases, a comprehensive evaluation of the cardiovascular system is necessary to decide about the most appropriate therapy (including echocardiography, telemetric continuous ECG monitoring, measurement of NIBP, blood work including cardiac troponins, etc.); therefore, referral is frequently necessary.

What Can Be Done at a Referral Hospital?

A thorough evaluation of the cardiovascular system is necessary to identify any underlying pathology and decide on the most appropriate therapy. This will include echocardiography, telemetric continuous ECG monitoring, measurement of NIBP, blood work including cardiac troponins and electrolytes. In addition, most anti-arrhythmic therapy is also pro-arrhythmic, so close monitoring of the cardiac rhythm is required during therapy, to identify any worsening of the dysrhythmias. There are situations where the cardiac dysrhythmias are secondary to underlying structural heart disease, and this will alter the prognosis and likelihood of response to therapy.

How to Decide the Most Appropriate Course of Action?

If the horse has **clinical signs** that the dysrhythmia is **hemodynamically relevant** (see Table 12.2) then **immediate treatment or referral** for appropriate treatment is indicated. If the horse appears hemodynamically stable (e.g., heart rate is fairly appropriate, normal mentation, and good peripheral perfusion) then you can consider referral for evaluation as a less emergent situation.

Congestive Heart Failure

CHF is the clinical scenario in which the heart is unable to pump enough blood to meet the requirements of tissue perfusion and oxygen delivery or can only do so from an elevated filling (venous) pressure. The problem can arise from abnormal filling or ejection. Left- or right-sided heart failure caused by valvular insufficiency is the most common cause of CHF, and filling problems are rare in the horse.

The most common **signs** (Box 12.2) in horses presenting in CHF are tachycardia, tachypnea, murmurs that are often (but not always) grade $\geq 3/6$, abnormal lung sounds (crackles), jugular distention or pulses, ventral edema, or poor body condition score. Murmurs are more common in the left side of the chest and systolic. A discussion on common murmurs and how to differentiate them is found in Appendix 12.1 (online). Muffled heart sounds; abnormal mucous membranes or fever are less commonly described signs.

Box 12.2 Clinical signs of congestive heart failure (CHF)

- Tachycardia.
- Tachypnea.
- Murmurs that are often grade $\geq 3/6$.
- Abnormal lung sounds (crackles).
- Jugular distention or pulses.
- Ventral edema.
- Poor body condition.

Emergency Triage Steps

Patient Stabilization

Urgent medical therapy is needed to stabilize horses in CHF. The initial approach to the most common presentations of CHF includes **diuretics** to decrease preload, angiotensin-converting enzyme **(ACE) inhibitors** to reduce preload and afterload and **inotropes** to improve systolic function. It is common that horses in CHF will present with atrial fibrillation (AF) and an increased heart rate. The medications commonly used in treatment of CHF are described in Table 12.5.

Table 12.5 Therapeutic treatment options for congestive heart failure.

Drug	Drug class	Dose recommendations	Dose for 500 kg bwt	Comments
Diuretics				
Furosemide	Loop diuretic	1–2 mg/kg IV initially, repeat as indicated 0.12 mg/kg/min CRI if further diuresis is indicated after initial bolus (Johansson et al. 2003) Long term management: 1–2 mg/kg IV or IM q 6–12 h	500–1,000 mg 60 mg/min CRI 500–1,000 mg	Induces rapid diuresis (within 15 mins) after administration. Titrate dose based on clinical progression. Oral absorption is poor. If longer-term management is required then intramuscular administration is advised. Adverse effects: hypovolemia, hypokalemia, hyponatremia, hypomagnesemia, metabolic acidosis, azotemia. Contraindications: Pericardial effusion causing cardiac tamponade. Caution in patients with pre-existing renal disease.
Torsemide	Loop diuretic	0.5–1 mg/kg PO q 12 h	250–500 mg	Advantage of higher oral bioavailability than furosemide, with less hypokalemia and longer half-life. Adverse effects: hypovolemia, hypokalemia, hyponatremia, hypomagnesemia, metabolic acidosis, azotemia. Doses of 4 mg/kg are reported to cause excessive diuresis and dehydration. Contraindications: Pericardial effusion causing cardiac tamponade. Caution in patients with pre-existing renal disease.
ACE inhibitors				
Benazepril	ACE inhibitor	0.5–1 mg/kg PO q 12 h	250–500 mg	Preload and afterload reduction through effects on the RAAS. When used in conjunction with diuretics (e.g., furosemide) can reduce clinical signs of heart failure. Enalapril is not absorbed in horses. Adverse effects: azotemia, cough, bronchoconstriction. Monitor for hypotension when used in conjunction with diuretics. NSAIDs may reduce clinical efficacy of ACE inhibitors. Contraindications: Hyponatremia. Do not use in pregnant animals. Caution in patients with pre-existing renal disease.

Table 12.5 (Continued)

Drug	Drug class	Dose recommendations	Dose for 500 kg bwt	Comments
Quinapril	ACE inhibitor	0.25 mg/kg PO q 24 h	125 mg	Preload and afterload reduction through effects on the RAAS. When used in conjunction with diuretics (e.g., furosemide) can reduce clinical signs of heart failure. Lower ACE inhibition reported compared to benazepril but anecdotally there is still perceived clinical improvement with its use. Adverse effects: azotemia, cough, bronchoconstriction. Monitor for hypotension when used in conjunction with diuretics. NSAIDs may reduce clinical efficacy of ACE inhibitors. Contraindications: Hyponatremia. Do not use in pregnant animals. Caution in patients with pre-existing renal disease.
Inotropes				
Dobutamine	β_1 – adrenergic, β_2 – and α_1 adrenergic **Preferred over dopamine**	1–5 µg/kg bwt/min CRI, titrate to effect or adverse reaction	500–2,500 µg/min CRI	Long-term therapy not effective due to tolerance. Continuous ECG monitoring recommended. Decrease dose if tachycardia or tachyarrhythmias develop. Adverse effects: Tachycardia, ventricular dysrhythmias, vasoconstriction and hypertension (at doses >4 µg/kg bwt/min) Contraindications: Ventricular dysrhythmias, tachycardia, atrial fibrillation (risk of severe tachycardia due to accelerated atrio-ventricular conduction)
Digoxin	Digitalis glycosides	0.0022 mg/kg q 12 h IV (Sweeney et al. 1993) 0.011 mg/kg q 12 h PO	1.1 mg IV 5.5 mg PO	TDM: peak (1–2 h) and trough (12 h) concentrations at steady state should fall within 0.8–2.0 ng/mL (1–2.6 nmol/L) Adverse effects: Depression, anorexia, colic, diarrhea, sinus bradycardia, AV block, supraventricular and ventricular dysrhythmias (bigeminy) Contraindications: AV block, diastolic ventricular dysfunction, pre-existing digitalis toxicity, myocarditis, ventricular dysrhythmias.
Pimobendan	Phosphodiesterase-3 inhibitor Inodilator	0.25 mg/kg PO q 12 h	125 mg	No clinical data on its use in horses with CHF. Concurrent use with calcium channel blockers or beta blockers may attenuate effects of pimobendan. Adverse effects: Dysrhythmias. Contraindications: Hypertrophic cardiomyopathies, valvular stenosis.

Bwt, bodyweight; Kg, kilogram; CNS, central nervous system; AV, atrio-ventricular; CRI, continuous rate infusion; GI, gastrointestinal; IV, intravenous; ACE, angiotensin converting enzyme; RAAS, renin angiotensin aldosterone system; TDM, therapeutic drug monitoring; CHF, congestive heart failure.

Emergency Treatment Guidelines

Diuretics

Decreasing preload is often the most important component of CHF treatment in the horse. Diuretics decrease congestion and slow the progression of the disease by decreasing ventricular filling pressures (Redpath and Bowen 2019).

Furosemide, a loop diuretic, given intravenously achieves rapid diuresis (within 15 minutes) and is short acting (approximately two hours) (Hinchcliff and Muir 3rd 1991).

Torsemide is another loop diuretic that has the main advantage of a high bioavailability after oral administration (Agne et al. 2018). Other potential advantages are less potassium loss, a half-life that allows dosing every 12 h and less renin–angiotensin–aldosterone system activation than more frequent doses of furosemide.

ACE Inhibitors

Benazepril is given mainly for its preload- and afterload-reducing properties and overall benefits of blunting the renin–angiotensin–aldosterone system. Effects on echocardiographic measurements have been shown in horse (Afonso et al. 2017, 2013). Other ACE inhibitors like quinapril have been used in the horses with perceived clinical benefit despite a lower reported percentage of ACE inhibition in experimental conditions. Enalapril is not absorbed in the horse. For many, benazepril is the ACE inhibitor of choice in most clinical scenarios.

Inotropes

Digoxin is a cardiac glycoside that traditionally has been used for its positive inotropic and rate-controlling effects. One of its main uses is to slow the ventricular response rate in AF, a common scenario in the horse with CHF. Digoxin has arrhythmogenic effects and can cause anorexia and dull mentation.

Dobutamine is the most widely used positive inotrope with weak positive chronotropic and vasoconstrictive effects. Due its short half-life a constant rate infusion (CRI) is needed.

Pimobendan is an inodilator very frequently used in small animals with valvular disease or CHF. Information about the efficacy in the equine clinical setting is lacking, but pimobendan has been shown to have positive chronotropic and inotropic effects in healthy horses after IV administration (Afonso et al. 2016).

Other therapies less commonly used are:

Sedation - Sedatives such as acepromazine (0.005–0.01 mg/kg IV or IM) or butorphanol (0.01–0.1 mg/kg IV or IM) can be given to treat anxiety associated with dyspnea. Blood pressure, heart rate, and respiratory rate should be monitored. Acepromazine induces vasodilation and blood pressure should be monitored to prevent severe hypotension and lower doses should be used initially.

Vasodilators - Calcium channel blockers such as amlodipine and the indirectly acting vasodilators like prazosin and isoxsuprine have not been evaluated for the management of equine cardiovascular disease and are used anecdotally following doses adapted from small animals (Redpath and Bowen 2019). Hydralazine (0.5–1.5 mg/kg PO q 12 h) and milrinone (0.5 µg/kg bolus then 0.5–5 µg/kg/min CRI) have been rarely reported in the horse.

Oxygen supplementation can be administrated via nasal cannulas at 5–10 L/min when tissue oxygenation is assessed as poor. Pleural or peritoneal drainage may be needed if effusions can contribute to respiratory distress or poor ventilation. These scenarios are rare in the horse.

Sildenafil, spironolactone, nitroglycerin (glyceryl trinitrate), bronchodilators, or beta blockers could play a role in some cases of CHF, but their use is **not common** in equine CHF.

Diagnostic Plan

History: Assessing the clinical **history** is a key component. Exercise intolerance, murmurs, increase in respiratory rate or effort, and tachycardia are commonly found in the history of horses with CHF. Occasionally the first sign of heart failure that owners detect is severe pulmonary edema and foamy nasal discharge.

Clinical Pathology: Clinicopathological findings are often absent or non-specific. Assessing renal function is recommended due to potential for kidney injury due to poor renal perfusion and the frequent importance of renal function in the pharmacology of drugs used to treat CHF.

Rhythm Assessment: Sinus tachycardia is the most common **rhythm**, while AF is also a common finding. Loss of atrial booster pump function at the time of AF development can be a tipping point for horses with long standing moderate-to-severe valvular disease and trigger signs of CHF due to the sudden loss of atrial function. Other bradyarrhythmias and tachyarrhythmias that can cause low output cardiac failure or syncope are described in the dysrhythmia section. Telemetric ECG monitoring is useful in acute CHF to monitor rhythm and rate response to therapy.

Imaging is an important piece of the investigation of CHF. Ultrasound is often key and Point of Care Ultrasound **(POCUS)** is more frequently being utilized to quickly evaluate the cardiovascular system (Bevevino et al. 2023; Eberhardt and Schwarzwald 2022). Common echocardiography findings are cardiac chamber enlargement, pulmonary artery larger than the aorta, pericardial effusion, and changes in echogenicity of the valves. The detailed echocardiographic changes associated with different cardiac diseases (Schwarzwald 2019) are beyond the scope of this chapter, but POCUS findings that are high yielding and can aid in triage are relevant to all equine practitioners evaluating emergencies. More details can be found in Appendix 12.2 (online). Radiographs are less commonly performed in horses to evaluate cardiac disease when compared to other species due to the less frequent availability and limited information obtained when compared to echocardiography and ultrasound. A diffuse interstitial to broncho-interstitial pattern, consistent with pulmonary edema and cardiomegaly, are the common findings when radiographs are performed in horses in CHF.

Important Decisions During Management

Decision for Hospital Referral

Hospital referral may be indicated for horses requiring intensive treatment or advanced diagnostics (Box 12.3).

Decision for Medical vs Surgical Treatment

Surgical treatment is not relevant to the management of CHF in the horse. There are exceptional descriptions of pericardiectomy in horses, but this is reserved for rare cases.

How to Manage Important Complications

If a patient initially stabilizes with appropriate emergency treatment, attention should be placed primarily on maintaining cardiac output while watching for organ dysfunction. Acute kidney injury is a common side effect of both CHF and aggressive treatment with diuretics, ACE inhibitors and inotropes. Laminitis, gastrointestinal injury, or central nervous system dysfunction can also occur secondary to CHF and its treatment.

Estimating Prognosis and Decision for Euthanasia

The prognosis for horses with CHF is poor. In a case series with 14 horses presented in CHF 7 were euthanized after initial evaluation, 5 died within a year, and 2 were lost to follow up (Davis et al. 2002), and all horses in a case series of severe mitral regurgitation died or were subjected to euthanasia within a year due to the severity of their MR and/or lack of response to therapy (Reef et al. 1998). Pharmacological and monitoring advances in the last 20 years mean that stabilization and short-term therapy are currently a feasible option in many horses. The prognosis for return to athletic function after CHF due to valvular or congenital heart disease is very poor and guarded to fair when acute CHF develops due to pericardial, myocardial disease or dysrhythmias that is amenable to treatment. Euthanasia is a fair option in horses in CHF for which appropriate treatment and monitoring are not option. Natural death in horses with untreated and unmonitored CHF is agonal and should be avoided at all costs.

What Can Be Done at the Farm

Triage and emergency treatment of horses in CHF can be performed on farm. Therapy and monitoring can be performed on farm in many scenarios when the evaluation and monitoring can be provided by ambulatory veterinarians.

Box 12.3 A horse may need referral to a hospital if it:

- Needs intensive therapy that cannot be provided on farm such as CRIs or oxygen.
- Needs intense or frequent monitoring that cannot be provided on farm.
- Needs advanced diagnostics (echocardiography) and this is not available on farm.
- Needs expertise that cannot be provided on farm.

What Can Be Done at a Referral Hospital

The possibility of round-the-clock monitoring and delivery of CRI medications are the main advantages of treating CHF in a hospital setting. The frequent presence of cardiologists or large animal internists with cardiac expertise is also an advantage although these experts can be available for ambulatory appointments in some regions.

How to Decide the Most Appropriate Course of Action?

Ultimately, the decision for referral or not will involve discussion with the owners about their financial situation, estimated prognosis and likelihood of a successful outcome, coupled with the severity of clinical signs that the patient is exhibiting. For cases of CHF where the underlying disease process can resolve (e.g., CHF secondary to overdose of sedation), advanced diagnostic testing and intensive monitoring and labor-intensive treatments (e.g., CRI's of medication) will be required, which are more easily handled in the referral setting than on farm. If severe underlying disease is suspected or known (e.g., pre-existing progressive mitral valve regurgitation), then the prognosis for horses with CHF is very poor and euthanasia is often indicated. The stress of transport to a referral institution can often worsen the clinical signs and cause the patient unnecessary distress.

Bacterial Endocarditis

Emergency Triage Steps

Patient Stabilization and Emergency Treatment Guidelines

Bacterial endocarditis is a valvular disease that deserves special consideration. The patient stabilization and treatment of the consequences of the valvular insufficiency may be similar than the stated above and the evaluation and treatment of the septic process is described below.

Diagnostic Plan

The common clinical and clinicopathological signs of active bacterial endocarditis are fever, shifting lameness, synovial effusion, increase in acute-phase proteins (fibrinogen and SAA), hyperglobulinemia, leukocytosis, neutrophilia, and anemia. The presence of nodular, vegetative valvular lesions or diffusely thickened valves and less commonly with other endocardial areas (e.g., chordae tendinea) supports a diagnosis of bacterial endocarditis, but there is overlap between the appearance of bacterial endocarditis and valvular degeneration.

Blood cultures are important in the workup of horses with suspected endocarditis. Bacteremia can be intermittent, and several blood cultures may be needed. There is not a stablished protocol in the horse and three cultures at least one hour apart have been described. Antimicrobial therapy based on culture and sensitivity and using bactericidal IV antibiotics is the first choice. Broad-spectrum coverage should be initiated promptly, and this often implies an empirical choice before culture and sensitivity results are received or after a negative culture is obtained in horses that the rest of the clinical presentation suggests bacterial endocarditis. Penicillin and gentamicin are common empirical choices when culture and sensitivity are not available or when cultures are negative and infective endocarditis is suspected. *Actinobacillus* spp., *Pasteurella,* and *Streptococcus* spp. are commonly described infectious agents associated with bacterial endocarditis (Henderson et al. 2020; Maxson and Reef 1997; Porter et al. 2008).

Other Treatments

The use of platelet aggregation inhibitors (clopidogrel 4 mg/kg, once, then 2 mg/kg, q24h) (Watts et al. 2014) is a reasonable adjunct therapy, but the benefits are unproven or controversial. The main benefit of the use of platelet aggregation inhibitor may be to halt the growth of the endocarditis lesion while the main counterargument is the potential benefit of platelets antibacterial effects.

Important Decisions During Management

Endocarditis associated with the mitral or aortic valve is more common in the horses and carries is grave prognosis. Endocarditis associated with the tricuspid or pulmonic valve also occurs, has a better prognosis, but this is still guarded.

Myocardial Injury and Myocarditis

Myocardial disease is relatively uncommonly reported in the horse although perhaps under-recognized.

Emergency Triage Steps

Patient Stabilization

Therapy for myocardial injury is aimed at treating the underlying cause and managing the sequelae and is a combination of the principles described for treating dysrhythmias, CHF, and intoxications. Corticosteroids, Vitamin E, selenium, or antibiotics are used in selected cases in which inflammation, oxidative damage, nutritional deficiency, or infections are associated with the case. Positive inotropic agents could aggravate ventricular dysrhythmias and are contraindicated in horses with an ionophore or cardiac glycoside intoxication and therefore should be used with caution (Decloedt 2019).

Diagnostic Plan Toxic insults and myocarditis may be the most common clinical problems in the emergency setting. Myocardial fibrosis, fatty and fibrofatty infiltrates are also reported and can trigger dysrhythmias or sudden death. The clinical interpretation of myocardial fibrosis is however complicated by the fact that this can be found also in necropsies of horses dead or euthanized due to known non-cardiac causes.

The multiple causes of myocardial disease have been recently reviewed and viral diseases (herpesviruses, influenza or equine viral arteritis) or bacterial diseases (*Streptococcus* spp., *Staphylococcus* spp., *Clostridium chauvoei*, *Borrelia burgdorferi*, Piroplasmosis, Leptospirosis, or *Neorickettsia risticiii*) can be suspected. Fungal disease or parasite migration have been rarely described. Intoxications from Ionophores (monensin, lasalocid, salinomycin) or plants containing cardiac glycosides such as *Digitalis* spp. (foxglove), *Taxus* spp. (yew), *Pimelea* spp., *Nerium oleander*, *Adonis aestivalis* (summer pheasant's eye), *Rhododendron,* etc. have been reported. Other intoxications with cardiac signs are: *Malva parviflora* (marshmallow), atypical myopathy/seasonal pasture myopathy caused by ingestion of hypoglycin-A from seeds of maple trees (*Acer* spp.), White snakeroot (*Ageratina altissima*), cantharidin (blister beetle), sodium fluoroacetate rodenticides, or heavy metals.

Other diseases that can be associated with myocardial insults are nutritional diseases (vitamin E/selenium deficiency), neoplasia (lipoma, pulmonary carcinoma, hemangioma/hemangiosarcoma, lymphoma/lymphosarcoma, melanoma, mesothelioma), systemic disease (systemic hypertension, pheochromocytoma/catecholamines), genetic disease (glycogen branching enzyme deficiency), tachycardia-induced cardiomyopathy, trauma, immune-mediated processes, amyloidosis, hypoxia, or hemorrhage.

Myocardial disease is often subclinical but can present as poor performance, exercise intolerance, sudden death, CHF, weakness, syncope, persistent tachycardia, or cardiac dysrhythmias. Murmurs are uncommonly but could occur due to abnormal filling patterns or to stretching of the atrioventricular valve annulus.

Echocardiography can show abnormal myocardial function, dimensions, or morphology or could be normal. Hyperechoic areas in the myocardium often represent myocardial fibrosis, while dilated cardiomyopathy is rare in the horse and, if recognized, is probably a sequela to a previous episode of myocarditis or myocardial injury/necrosis. Tissue Doppler imaging may be a more sensitive way to detect systolic or diastolic dysfunction than standard 2D echocardiography. Many of the imaging aspects of myocardial disease may be harder to detect with POCUS with the exception of moderate or severe hypertrophy or pseudohypertrophy. A hypertrophic appearance (increased relative wall thickness) can occasionally be seen during myocardial injury (due to edema or cellular infiltration), but a similar appearance can be seen due to dehydration (pseudohypertrophy) (Navas de Solis et al. 2013; Underwood et al. 2011).

Cardiac Troponins

Cardiac troponins are part of the cardiac muscle contractile apparatus and are specific markers of damage. Cardiac troponins (cTn) I and T are currently the most used and sensitive markers of myocardial damage. The half-life of cTns after experimental injection of recombinant cTns and after exercise (Kraus et al. 2013; Rossi et al. 2019) was 0.47 and 6.4 h. Peak concentration of cTns has been described at 3–6 h after exercise (return to normal after 24 h) and 24–72 h after monensin administration (Kraus et al. 2010). On many occasions, ongoing damage and elimination are likely to overlap, and the effective half-life may be longer than that described experimentally. It is important to recognize that cTns can increase in systemic diseases such as endotoxemia (Nostell et al. 2012), hemorrhage (Navas de Solis et al. 2015), or colic (Díaz et al. 2014), without the presence of primary myocardial injury.

Cardiac troponin I is most commonly measured in horses in most countries, but recently cardiac troponin T assays have been reported and validated (Ven Der Vekens et al. 2015). The reference range is specific for each cTnI assay and uniform for cTnT. Ultrasensitive cTnT, Roche Diagnostics assays, architect STAT High Sensitivity Troponin I (Abbott), Dimensions Vista Troponin I (Siemens) or Stratus CS Acute Care cTnI Cal Pak, (Siemens), have been reported. A point-of-care analyzer (iStat) has been used in several studies with apparently adequate performance to distinguish horses with and without myocardial insults, but formal validation of this assay is currently not available (Rossi et al. 2018; Shields et al. 2016).

Important Decisions During Management

Stall rest for at least one month after myocardial injury is recommended and monitoring of the cardiac rhythm at rest and during exercise after the recovery period is often indicated.

The prognosis depends on the severity of the myocardial injury, the degree of myocardial dysfunction, development of any dysrhythmias, and presence and degree of subsequent myocardial fibrosis. Horses with mild myocardial injury can fully recover, but the damage can be life-threatening in some cases.

Pericardial Disease

Pericardial disease is rarely identified in the horse and can be associated with infectious (bacterial, viral), neoplastic (lymphoma, mesothelioma) or immune-mediated diseases or develop secondary to CHF or pleuropneumonia (Worth and Reef 1998). Bacterial pericarditis can be caused by a variety of etiologic agents, with *Actinobacillus* spp. or opportunistic bacteria associated with exposure to eastern tent caterpillars being most frequently reported. Effusive or fibrino-effusive pericarditis are most common forms, but dry pericarditis can also occur and is more difficult to recognize. Pericarditis can cause cardiac compression (tamponade) due to increased pericardial pressure reducing ventricular filling, or less severe diastolic dysfunction. Because of the lower right-sided pressures, the main consequence is impaired venous return to the right heart, resulting in clinical signs of right-sided CHF including jugular vein distension, pulsation, and formation of peripheral edema.

Emergency Triage Steps

Patient Stabilization

Treatment is dependent on the cause and often includes drainage of pericardial fluid if tamponade is present, anti-inflammatory and antibiotics (broad spectrum, e.g., penicillin/gentamicin), and rest. After drainage, often an indwelling catheter is left to perform pericardial lavage, provide local antibiotic therapy, and avoid adhesions.

It is important to recognize that *diuretics are contraindicated* with pericardial effusion, as a rapid decrease in preload may compound the existing impaired filling and worsen clinical signs.

Diagnostic Plan

Clinical signs of pericardial disease are initially non-specific and often depend on the amount and rate of pericardial fluid accumulation and the underlying disease. Common signs include history of respiratory disease, fever, lethargy, exercise intolerance, tachycardia, venous distension jugular pulses, ventral edema, pleural or peritoneal effusions. Classically described cardiac auscultation includes muffled heart sounds or friction rubs.

Echocardiography is the most useful tool for diagnosis and assisting therapy and monitoring. Pericardial fluid analysis, with aerobic and anaerobic culture, can help narrow the list of differential diagnoses.

Important Decisions During Management

The prognosis is favorable for idiopathic pericarditis but guarded or poor for bacterial pericarditis or neoplasia. Early diagnosis and appropriate, aggressive therapy can allow full recovery and return to athletic function in some cases.

Cardio-Pulmonary Resuscitation

Cardio-pulmonary resuscitation (CPR) is required when patients suffer from cardio-pulmonary arrest (CPA). In general, CPR in horses should be approached in the same systematic manner as in humans and small animals; however, there are some important considerations (Fletcher et al. 2012). The size of patient will dictate the type of chest compressions and the effectiveness of any attempts at defibrillation. In general, the clinician should remember the CPR basics of ABCD: **A – airway, B – breathing, C – circulation,** and **D – drugs** to be administered. Prompt recognition of the need for CPR, and rapid delivery of basic life support (BLS) and advanced life support (ALS) is critical for CPR to have a successful outcome. The clinical scenario in which the CPA occurred is also important for determining the likelihood of return to spontaneous circulation (ROSC) (e.g., a patient under general anesthesia vs a patient that has collapsed in the field setting or a neonate that has arrested during parturition) (Conde Ruiz and Junot 2018; Duggan et al. 2022; Hopster et al. 2016; Wiechert-Brown et al. 2023).

It is very helpful to have discussed the client's wishes for CPR prior to any emergency or elective procedure that has the potential to result in CPA. Having your team aware of the CPR code status (e.g., green for full CPR including ALS, orange for BLS only and red for do not resuscitate) prior to starting a procedure can allow for timely provision of CPR in an emergent situation, without the need to first contact the owners to get permission. Performing CPR comes with additional costs and risks that the owners should be aware of.

Regular veterinary care team trainings should include running a CPR code so that all members of the care team are familiar with the CPR guidelines and understand their role when an animal suffers from CPA. Preparation and familiarity with the treatment guidelines are critical to performing successful CPR.

Emergency Treatment Guidelines

Graphical algorithms describing the basic and advanced life support steps for performing CPR in adult horses and foals are found in Figures 12.4 and 12.5, respectively. These can be printed out and placed on a crash cart for quick reference during emergencies. The common emergency medications used during CPR are found in Table 12.6.

If a patient is found unresponsive, the presence of breathing and a heart beat should be evaluated quickly by auscultation of the heart. Palpation of a peripheral pulse can be difficult in the emergency setting and therefore should not be relied upon. If no heart beat is appreciated, external chest compressions should be started immediately. The patient should be in lateral recumbency (preferably right lateral) with the head and neck extended. Forcefully compress the chest just behind the elbow with the resuscitator's knees or hands (if the patient is small). The pulse strength can be monitored to estimate the effectiveness of the compressions, in addition to direct measurements of arterial pressures if available.

If the patient is not breathing, an airway should be obtained either through naso-tracheal or oro-tracheal placement of an endotracheal tube. If this is not possible, an emergency tracheostomy should be performed, and the endotracheal tube placed directly in the trachea. The endotracheal tube's cuff should be inflated, and the tube attached to a demand valve, ambu bag (if the patient is small) or anesthetic machine with rebreathing bag, to provide ventilation. A tidal volume of 10 mL/kg is appropriate (5 L for a 500 kg horse), and the demand valve or rebreathing bag should be compressed to provide between 20 and 40 cm H_2O. If no pressure sensor is available, the amount of chest excursion should be visually evaluated. If arterial oxygenation can be monitored, room air is preferred over 100% O_2, but in the absence of such monitoring, 100% O_2 is appropriate.

One cycle of BLS is two minutes duration. During this time, maintain regular chest compressions (approx. 30–60/min for an adult, 90–120/min for a foal) and provide interposed ventilation (4–6/min for an adult, 10/min for a foal). Every two minutes the compressor should change out, to avoid fatigue and reduced efficacy. Chest compressions in adult horses will not provide sufficient flow to support life but may aid to prolong life in order to facilitate distribution of resuscitative drugs to vital tissues.

If the equipment is available to provide ALS, an ECG should be placed to monitor the cardiac rhythm, end-tidal CO_2 measured to assess the effectiveness of compressions, and IV catheter placed, and reversal of any anesthetic, sedative, or analgesic agents should be performed. End-tidal CO_2 is used to monitor for ROSC due to increasing pulmonary blood flow associated with effective cardiac output; values above 15 mmHg have been associated with more positive CPR outcomes in small animals.

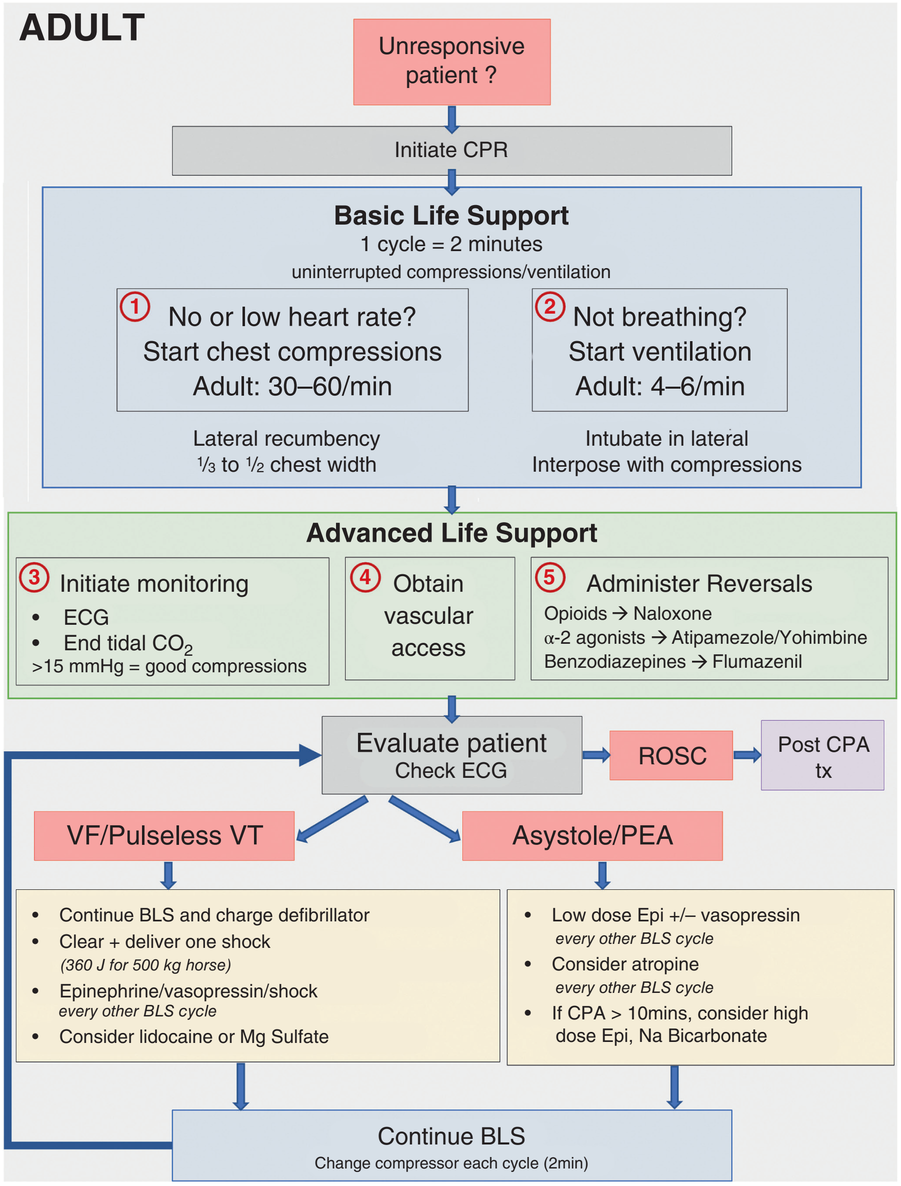

Figure 12.4 Cardio-pulmonary resuscitation (CPR) algorithm for adult horses. CPR, cardio-pulmonary resuscitation; BLS, basic life support; ALS, advanced life support; ECG, electrocardiogram; α-2, alpha 2; CPA, cardio-pulmonary arrest; ROSC, return of spontaneous circulation; VF, ventricular fibrillation; VT, ventricular tachycardia; PEA, pulseless electrical activity; Epi, epinephrine; Na, sodium; J, joule; Mg, magnesium.

Every two minutes, the patient should be evaluated for ROSC, including a pulse check and ECG rhythm check. Administration of low-dose epinephrine alternating with vasopressin (if available) is recommended every second cycle of BLS if ROSC has not occurred.

If a defibrillator is available and ventricular fibrillation (VF) or pulseless ventricular tachycardia (VT) is present, an attempt at external defibrillation can be made in smaller patients with a size similar to or smaller than an adult human. Successful defibrillation of horses up to 341 kg has been described using custom-made defibrillators, but the default settings and safety mechanisms of commercial defibrillators do not allow to replicate this experience in most settings. It is important to use the electrode gel on the defibrillator paddles and ensure everyone is clear of the horse before defibrillation is attempted. *Do not use* a defibrillator if *alcohol* is present on the skin of the animal, as this poses a fire hazard.

In the case of VT or VF, or after a 6 minute period (3 × BLS cycles) of unsuccessful CPR in the absence of an ECG/hemodynamic monitoring, a dose of lidocaine or magnesium sulfate could be given if several cycles of BLS have not shown any ROSC. If a horse has been in CPA for more than 10 minutes, the chances of successful CPR are very low.

If the patient does show signs of spontaneous circulation, it is important to provide appropriate IV fluid support, monitoring of systemic blood pressure and cardiac output with vasopressor assistance if required. Cerebral edema is a common sequalae of CPA; therefore, close monitoring and treatment (e.g., mannitol) may also be necessary.

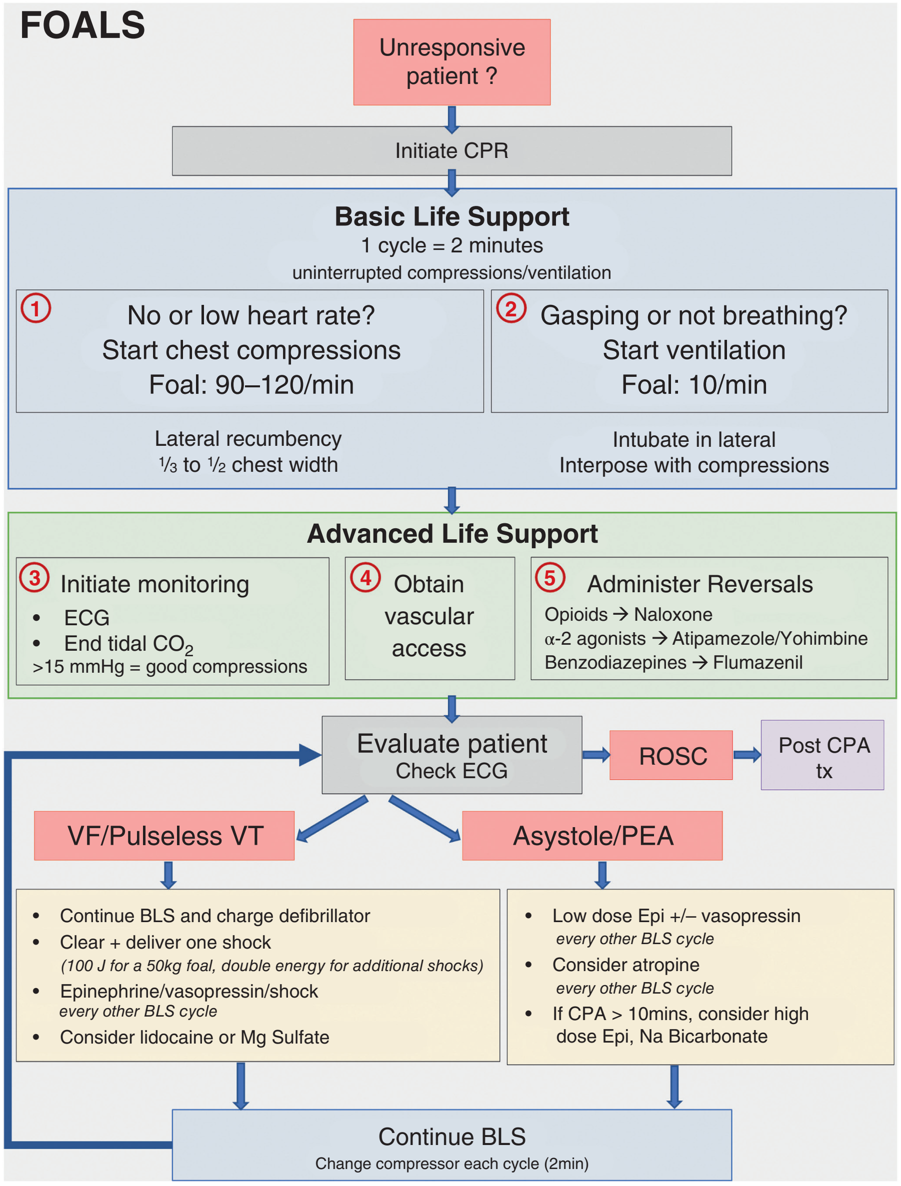

Figure 12.5 Cardio-pulmonary resuscitation (CPR) algorithm for foals. CPR, cardio-pulmonary resuscitation; BLS, basic life support; ALS, advanced life support; ECG, electrocardiogram; α-2, alpha 2; CPA, cardio-pulmonary arrest; ROSC, return of spontaneous circulation; VF, ventricular fibrillation; VT, ventricular tachycardia; PEA, pulseless electrical activity; Epi, epinephrine; Na, sodium; J, joule; Mg, magnesium.

Important Decisions During Management

Decision for Hospital Referral

In the situation of CPR, the decision regarding referral is based upon the availability of appropriate post-resuscitation care and monitoring. If the CPR is successful, the patient is still likely to require intensive care for a period of time and when this cannot be provided locally, then referral to an appropriate hospital facility is indicated.

How to Manage Important Complications

Depending on the reason for CPA and the speed at which ROSC occurs, there may be important sequalae particularly involving the central nervous, cardiovascular, gastrointestinal, and renal systems. Altered mental state, seizures, renal failure, cardiac injury, decreased cardiac function, dysrhythmias, thoracic trauma (e.g., fractured ribs, lung laceration), and gastrointestinal dysfunction are all possible consequences of CPR, and intensive supportive care is indicated when signs of organ dysfunction are present.

Table 12.6 Drugs required during cardio-pulmonary resuscitation (CPR).

Drug	Drug class	Dose recommendations	Dose for 500 kg bwt	Dose for 50 kg bwt	Comments
First-line medication during CPR					
Epinephrine (Adrenaline) 1:1,000; 1 mg/mL	Non-selective adrenergic agonist	Low dose: 0.01 mg/kg bwt IV High dose: 0.1 mg/kg bwt IV	Low dose: 5 mL High dose: 50 mL	Low dose: 0.5 mL High dose: 5 mL	β-adrenergic at lower doses, α_1-adrenergic at higher doses Avoid high doses of epinephrine during CPR where possible Indications: ventricular fibrillation/ pulseless ventricular tachycardia, ventricular asystole, cardiopulmonary resuscitation (use every other cycle, alternate with vasopressin if available) Adverse effects: muscle tremors, excitability, arrhythmias, VF. Overdoses can lead to hypertension, dysrhythmias, renal failure, pulmonary oedema, cerebral hemorrhage Contraindications: non-anaphylactic shock, dysrhythmias, hypertension
Arginine Vasopressin (Antidiuretic hormone) 20 IU/mL	V_1 receptor agonist	0.8 IU/kg bwt IV bolus	400 IU (20 mL) IV bolus	40 IU (2 mL) IV bolus	Indications: ventricular fibrillation/ pulseless ventricular tachycardia, ventricular asystole, cardiopulmonary resuscitation. Alternate with epinephrine during CPR (use every other cycle) Adverse effects: myocardial infarction, sweating, decreased gastrointestinal perfusion, hypertension Contraindications: hypertension, heart failure, renal failure, seizures
Atropine 15 mg/mL (note concentration)	Anticholinergic (vagolytic)	0.01–0.02 mg/kg bwt IV or IM	5–10 mg (0.3–0.7 mL) IV or IM	0.5–1 mg (0.03–0.07 mL) IV or IM	Indications: bradycardia, high grade 2nd degree AV block, ventricular asystole (use every other cycle) Adverse effects: Constipation, ileus, colic, bradycardia (at very low doses), tachycardia, dysrhythmias, CNS effects (stimulation, drowsiness, ataxia, seizures, respiratory depression) Glycopyrrolate is slightly less arrhythmogenic and rarely results in CNS effects Contraindications: Tachycardia, tachyarrhythmia, heart failure, GI disease, colic
Lidocaine (Lignocaine) 20 mg/mL	Class I_B Na^+ channel blocker	0.25–0.5 mg/kg bwt slow IV, repeat in 5–10 min to effect, up to 1.5 mg/kg bwt total dose; followed by 0.05 mg/kg bwt/ min CRI	125–250 mg (6.3–12.5 mL) per bolus up to total dose of 750 mg (37.5 mL); CRI 25 mg/ min	12.5–25 mg (1.3–2.6 mL) per bolus up to a total of 75 mg (3.8 mL); CRI 2.5 mg/min	Indications: VT/VF Adverse effects: Uncommon at therapeutic doses. Overdose can lead to ataxia, muscle tremors, CNS excitement, dysrhythmias, and collapse. Contraindications: SA, AV, or intraventricular block, bradycardia. Caution with hypovolemia, liver disease, shock, and heart failure.

Table 12.6 (Continued)

Drug	Drug class	Dose recommendations	Dose for 500 kg bwt	Dose for 50 kg bwt	Comments
Magnesium sulfate	Physiologic Ca^{2+} and K^+ channel blocker. Activator of membrane Na^+/K^+ ATPase.	2–6 mg/kg bwt/min IV to effect, up to 55–100 mg/kg bwt total dose	1–3 g/min; total dose 27–50 g	100–300 mg/min; total dose 2.75–5 g	Indications: VT/torsade de pointes Adverse effects: Rare, but overdose may lead to CNS depressant effects, weakness, trembling, bradycardia, hypotension. Very high doses cause neuromuscular blockade with respiratory depression and cardiac arrest. Contraindications: Bradycardia, SA and AV block, renal failure.
Amiodarone	Class III K^+ channel blocker	5 mg/kg bwt IV slow bolus	2.5 g IV slow bolus	250 mg IV slow bolus	Indications: VT/VF Also class I, II and IV effects. Adverse reactions: Hind limb weakness, weight shifting, *torsade de pointes*, SA and AV nodal inhibition, bradycardia, hypotension. Prolonged treatment may affect lungs, liver, heart, thyroid gland, GI tract, eyes, skin, and nerves. Contraindications: Sino-atrial node dysfunction, bradycardia, AV block, cardiogenic shock.
Naloxone	Opioid antagonist	0.01–0.03 mg/kg bwt IV	5–15 mg IV	0.5–1.5 mg IV	Indications: reverse opioids. Monitor for effect as duration of action is shorter than opioids, so redosing may be required Adverse reactions: Rare, wide safety margin, may cause seizures at very high doses Contraindications: Pre-existing cardiac abnormalities, patients with opioid dependency
Flumazenil	Benzodiazepine antagonist	0.01 mg/kg bwt slow IV	5 mg slow IV	0.5 mg slow IV	Indications: reverse benzodiazepines Adverse reactions: Seizures. Contraindications: If benzodiazepines were initially given to control seizures, use caution when considering reversal as further seizure activity may occur
Atipamezole	$Alpha_2$ receptor antagonist	0.05 mg/kg bwt slow IV	25 mg slow IV	2.5 mg slow IV	Indications: reverse $alpha_2$ receptor agonists Adverse reactions: reversal can occur rapidly, patients may become aggressive or excited Contraindications: Not recommended in pregnant or lactating animals (lack of safety data)

Bwt, bodyweight; Kg, kilogram; CNS, central nervous system; SA, sino-atrial; AV, atrio-ventricular; CRI, continuous rate infusion; GI, gastrointestinal; VT, ventricular tachycardia; VF, ventricular fibrillation; IV, intravenous; CPR, cardio-pulmonary resuscitation.

Estimating Prognosis and Decision for Euthanasia

Unfortunately, very few resuscitation attempts are successful in equine patients with the exception of the immediate post foaling period or in systemically healthy horses under general anesthesia. However, the logistics of performing effective CPR on a 500 kg animal and the size limitations with successful external defibrillation in larger patients during ALS means that many animals will not survive despite our best resuscitation efforts. If an animal does have rapid ROSC, then the prognosis for survival is improved; however, delayed ROSC will be associated with a larger number of complications and likely multiple organ dysfunction. Owners should be warned of this guarded prognosis, particularly when involving critically ill patients.

If you are performing field or hospital-based anesthesia, or in a practice that sees a number of neonates, it is recommended to organize an emergency kit with CPR equipment and coordinate regular training sessions with your staff to practice running a "code." Evidence from human and small animal practice shows that CPR is more effectively performed when the team is well trained and prepared.

What Can Be Done on Farm?

Given the urgency of CPA, if it occurs on farm, then CPR will be performed immediately in the field. Often only BLS can be provided in the ambulatory setting. Performing adequate CPR is exhausting, particularly if there is only a small number of people available to help.

What Can Be Done at a Referral Hospital?

A referral hospital may have more capacity to provide ALS, with ECG and end-tidal CO_2 monitoring, a larger team of people and possibly access to a defibrillator. If ROSC occurs, then the patient will likely still require intensive care with regular monitoring, that is easier to provide in a referral setting.

How to Decide the Most Appropriate Course of Action?

In the setting of CPA, the decision to refer will likely be made if ROSC occurs, although this happens very uncommonly after CPR in large animals. If an animal is considered at high risk of having CPA, then referral before this occurs is warranted.

Blood Coagulation Disorders

Emergencies of coagulation in the horse can be divided into those of hypocoagulation, resulting in the clinical sign of hemorrhage, or hypercoagulation, where excessive clotting or inappropriate thrombosis within the vasculature is evident (Dallap 2004; Epstein 2014; Hurcombe et al. 2022).

The normal process of hemostasis can be divided into three main components – primary hemostasis, with the formation of a platelet plug; secondary hemostasis (formation of a mesh containing cross linked fibrin); and fibrinolysis, which is the process of breaking down the cross-linked fibrin. The formation of blood clots, or thrombi, is common when there is injury or inflammation within the vasculature, primarily aimed at limiting blood loss or the spread of infection in the local tissues. To counterbalance the formation of clots, there are inhibitory and fibrinolytic pathways that aim to limit clot formation to avoid damage to healthy tissue. Balance is required between the pro- and anti-coagulation activity, and dysfunction of either system can promote excessive bleeding or clotting.

Emergency Triage Steps

Patients can present on emergency with obvious clinical signs of excessive hemorrhage (e.g., a wound that will not stop bleeding, epistaxis, progressively enlarging hematoma) but if the hemorrhage is contained within a body cavity (e.g., abdomen, thorax, pericardium, bladder) then the clinical signs consistent with hypovolemia will be evident, but the source of the hemorrhage may not be obvious without further investigations.

Patients with hypercoagulability may have more subtle or variable clinical signs, depending on the location and size of the thrombi. The signs may be related directly to the clot (e.g., palpable mass in a jugular vein consistent with a thrombus) or a result of obstruction to blood flow (e.g., distension of the venous vasculature and edema of the soft tissues distal to venous thrombus). Arterial thrombi will result in ischemia of the tissue being supplied by that artery (e.g., cranial mesenteric artery thrombosis result in ischemia to the gastro-intestinal tract, resulting in colic signs).

Patient Stabilization

Rapid recognition and stabilization of patients with excessive bleeding is critical. If the hemorrhage is external, then appropriate bandaging and splinting (where indicated) is the first step to preventing further bleeding. If epistaxis is present, it is essential to keep the patient's head in a neutral-to-slightly elevated position, horses can fatally bleed out quickly from upper respiratory tract origin hemorrhage when the head is held in a low position. Keeping the patient calm (and therefore lowering heart rate and blood pressure) can help minimize ongoing hemorrhage.

Internal hemorrhage is more difficult to stabilize, with the first step keeping the patient calm to prevent further bleeding. Patients experiencing severe blood loss can become quite anxious as hemorrhagic shock develops. Careful sedation may be necessary to keep the patient quiet and prevent worsening injury to themselves or the people working to stabilize them. *Be aware* that boluses of alpha$_2$ agonists can cause *transient increases in systemic blood pressure*, which can worse hemorrhage. Alpha$_2$ agonists should be administered slowly over 2–3 mins, to reduce this effect.

Diagnostic Plan

If the patient is hemodynamically unstable, then minimal diagnostic evaluation is indicated in the first instance, and you should prioritize lifesaving emergency treatment measures. These unstable patients will have clear evidence of hemorrhagic shock on physical examination, with heart rates >80 bpm, respiratory rates >30 bpm, cold patchy sweating, muscle fasciculations, pale or gray tacky mucus membranes with prolonged capillary refill time, poor jugular filling, and prolonged skin tent. They can have dull mentation with no interest in their environment but could also be anxious and agitated. Gut sounds are typically decreased to absent and extremities cold with weak, thready peripheral pulses. Some patients with hemorrhagic shock appear colicky, pawing, kicking at their abdomen, shifting weight, and trying to lie down. Patients with signs of hemorrhagic shock need urgent but controlled volume expansion to restore cardiac output and maintain critical organ perfusion.

When the patient is fairly stable, then you will have more time to perform a diagnostic evaluation and make decisions regarding the most appropriate treatment. If a clear cause for the hemorrhage is known or can be identified (e.g., trauma) then the diagnostic plan is to stabilize and treat the primary problem and the consequences of the excessive hemorrhage. When the cause of the excessive hemorrhage is not obvious, then further investigations will be necessary, and the decision should be quickly made if this is possible in the field, or if referral is indicated.

Patients with excessive coagulation will likely require more extensive diagnostic evaluation to determine the underlying cause and severity of the disease process.

Emergency Treatment Guidelines

Excessive Bleeding In the field setting, emergency treatment for excessive hemorrhage is first aimed at restoring the circulating blood volume to maintain cardiac output and critical organ perfusion. Crystalloids are the easiest fluid to start with in the emergency setting, while a quick determination of whether blood products are also indicated should also be made, as these fluids take time to acquire and administer.

The current recommendations for the treatment of hemorrhagic shock are goal directed, with a starting bolus of hypertonic saline at a rate of 4 mL/kg given rapidly IV, followed by a balanced isotonic crystalloid fluid (e.g., Hartmann's solution or Plasmalyte A) at 20 mL/kg given over 30 minutes using a large bore catheter placed in the jugular vein (Crabtree and Epstein 2021; Epstein 2014; Fielding 2014; Fielding and Magdesian 2011; Pantaleon 2010; Pantaleon et al. 2007). Reassessment of heart and respiratory rate, membrane color, and signs of peripheral perfusion should be performed regularly to monitor for any improvement and to guide decisions about the need for further fluid therapy.

When there is evidence of hypocoagulability (e.g., petechia or ecchymoses on mucus membranes, bleeding from catheter or venipuncture sites) then it is highly likely that blood products (fresh frozen plasma or whole blood) will be required to provide support for the coagulation system.

Antifibrinolytics (e.g., aminocaproic acid (20–40 mg/kg diluted in 1 L of 0.9% NaCl IV) or tranexamic acid (5–25 mg/kg diluted in 1 L 0.9% NaCl IV)) is frequently used in the face of excessive hemorrhage, despite a paucity of evidence that horses have increased fibrinolysis and inappropriate break down of clots as a cause for the bleeding.

Excessive Clotting If excessive clotting occurs in the arterial side of the vasculature, then the clinical signs will likely be more severe, but the origin of the thrombosis may not be immediately clear and referral will likely be indicated to perform more advanced diagnostics and provide emergency treatment (e.g., local or systemic anti-coagulant, anti-platelet, and thrombolytic therapy).

Venous thrombosis is typically less immediately life threatening, most commonly involving the jugular vein, where it needs to be determined if there is just thrombosis, or if it is complicated by concurrent infection (thrombophlebitis). Treatment usually involved anti-coagulation and anti-platelet therapies (e.g., aspirin 11 mg/kg PO SID or clopidogrel 4 mg/kg PO loading dose, then 2 mg/kg PO SID, enoxaparin 40–80 IU/kg SC SID), and if infection is present, appropriate anti-microbial therapy will also be indicated (Epstein 2014).

Important Decisions During Management

Decision for Hospital Referral

If the patient is hemodynamically unstable, then rapid referral is usually indicated, as these patients require intensive treatment and ongoing monitoring. Most referral facilities have access to a range of blood products (e.g., fresh frozen plasma and universal blood donors) and technical support for thawing, collecting, and administering these products where necessary.

Decision for Medical vs Surgical Treatment

If the source of hemorrhage is known or located and is not responding to conservative therapy, then surgical intervention may be required to stop further hemorrhage. Examples where surgical intervention can provide rapid resolution for the hemorrhage would include epistaxis from guttural pouch mycosis, where ligation of the common carotid arteries can be lifesaving or in a horse with excessive hemorrhage following castration – standing laparoscopic ligation of the spermatic cord will immediately stop further hemorrhage. Careful consideration of the pros and cons should be given to surgical procedures that require general anesthesia, particularly the risk of prolonged hypotension, and the patients should be appropriately stabilized before general anesthesia is considered.

In cases of excessive thrombosis, surgical intervention is rarely indicated, especially in the emergent situation.

How to Manage Important Complications

Excessive hemorrhage resulting in hemorrhagic shock is associated with complications associated with critical organ damage (e.g., acute renal failure, laminitis, gastrointestinal, myocardial dysfunction). It is important to recognize the potential for these complications and warn owners, as complications like laminitis can take several days to develop clinical signs. These complications can be mitigated by rapid recognition of excessive hemorrhage and instigation of early and appropriate therapy, including referral.

Thrombosis of large arterial vessels is likely to result in ischemia and subsequent dysfunction of the tissues directly supplied by those arteries which could lead to signs of organ failure. While these types of thromboses are rare, they are highly consequential; therefore, rapid referral is indicated.

Estimating Prognosis and Decision for Euthanasia

If the patient has severe clinical signs involving hemodynamic instability and the failure of multiple organs then the prognosis will be poor to hopeless and euthanasia is indicated, as these patients can rapidly deteriorate, suffer pain and distress, and die suddenly. If the clinical signs are less severe or respond quickly to appropriate emergency treatment and the patient can be stabilized, then the prognosis can be better, but the possibility of delayed organ failure (e.g., laminitis) should be made clear to the owners. If the excessive hemorrhage is the result of a genetic disorder, then the prognosis is guarded for long-term management, but if an underlying toxicity or systemic disease is identified and can be treated, then the prognosis may be more favorable.

What Can Be Done on Farm?

Basic life-saving measures can be performed on farm, including bandaging, packing, or splinting of wounds to prevent further hemorrhage. Additionally, the placement of an IV catheter and administration of hypertonic saline or a bolus of crystalloids may provide enough cardiovascular support to allow time for referral. However, the time taken and availability of equipment and appropriate support on farm to perform these treatments should be factored into the decision making. If the distance to the referral hospital is short, then it may make more sense to proceed with referral, while if the journey is likely to be several hours, then stabilization before transport will be a better option if available.

What Can Be Done at a Referral Hospital?
Most referral hospitals can provide intensive care, continuous monitoring and have access to a wider range of therapeutic interventions and treatments. This could include monitoring of coagulation function testing, access to a variety of blood products, regular assessment of the effect of therapy (e.g., NIBP monitoring), and early identification of any complications (e.g., anuria, laminitis).

How to Decide the Most Appropriate Course of Action?
Ultimately, the decision for referral or not will involve discussion with the owners about their financial situation, prognosis and likelihood of a successful outcome, coupled with the severity of clinical signs that the patient is exhibiting. For most emergent disorders of the coagulation system, advanced diagnostic testing and intensive monitoring and labor-intensive treatments (e.g., blood transfusions) will be required, which are more easily handled in the referral setting than on farm.

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