Pferdeheilkunde – Equine Medicine 34 (2018) 4 (July/August) 414-418 DOI 10.21836/PEM20180501

A third degree AV-block in a horse with a putative regular rhythm and physiological heart rate in general examination

Antonia Ertelt[1], Christof A. Bertram[2], Aleksandra Zuraw[2] and Heidrun Gehlen[1]

[1] Equine Clinic, Internal Medicine, Freie Universitaet Berlin, Berlin, Germany
[2] Department of Veterinary Pathology, Freie Universitaet Berlin, Berlin, Germany

Summary: An 8-year-old warmblood mare was referred to the Equine Clinic of the Freie Universitaet Berlin because of irregular heart rhythm and multiple syncope. The clinical examination revealed a regular heartbeat (38 bpm) with a holosystolic heart murmur, grade III/VI on the left side and I/VI on the right side, punctum maximum on the left side over the mitral valve and on the right side over the tricuspid valve. Furthermore, jugular vein pulsation and a prolonged capillary refill time was obvious. A 24-hour continuous ECG revealed a regular rhythm with complete dissociation of the atrial and ventricular activity with an atrial rate of 62 bpm and ventricular rate of 38 bpm. Later on, the horse developed tachycardia and intermittent asystole. An echocardiography including B-mode, M-mode, color flow and continuous wave Doppler revealed valvular regurgitation of the mitral valve, tricuspid valve and pulmonic valve. The heart's dimensions, especially on the right side, were enlarged. Furthermore, the horse suffered from lung edema and edema formation on the ventral chest. Congestive heart failure with concomitant third degree AV block and, later on, a ventricular tachycardia with intermitted asystole was diagnosed. The initiated treatment was unrewarding. Due to the grave prognosis, the horse was euthanized. Necropsy revealed chronic, multifocal to coalescing, lymphoplasmacytic and fibrosing epicarditis, myocarditis and endocarditis as well as a mitral valve dysplasia and severe, diffuse subendocardial fibrosis.

Keywords: cardiac arrhythmia, cardiac heart failure, ventricular tachycardia, myocarditis, third degree AV block, mitral valve dysplasia

Citation: Ertelt A., Bertram C. A., Zuraw A., Gehlen G. (2018) Case report of a third degree AV-block in a horse with a putative regular rhythm and physiological heart rate in general examination. Pferdeheilkunde 34, 414-418; DOI 10.21836/PEM20180501

Correspondence: Dr. Antonia Ertelt, Freie Universität Berlin, Klinik für Pferde, Oertzenweg 19b, 14193 Berlin, Germany; antonia.ertelt@fu-berlin.de

Introduction

In general, cardiac arrhythmias with complete loss of atrioventricular conduction are uncommon in horses. Atrioventricular dissociation is an electrocardiographic syndrome; a descriptive term for a variety of conditions of abnormal cardiac conduction which all feature independent function of the atria and ventricles (*Harrigan* et al. 2001). The most common, but nevertheless very rare, conductive disturbance with complete atrioventricular dissociation in horses is the third degree atrioventricular block. A condition in which the impulse generated in the sinoatrial node does not propagate to the ventricles. The reason is a block either in the atrioventricular node, in bundle of his or bundle branches. Horses with third degree AV block typically experience severe bradycardia. A less common conductive disturbance with complete atrioventricular dissociation is isorhythmic atrioventricular dissociation (*Segers* et al. 1946, 1947). We present a case of a horse with no previously known cardiac failure or dysrhythmia, which developed a life-threatening arrhythmia.

Case history

An 8-year-old, 500 kg warmblood mare was referred to the Equine Clinic of the Freie Universitaet Berlin to evaluate suspected cardiac diseases and cardiac tachyarrhythmia (92 bpm) with multiple syncope since one month. Initial treatment by the referring veterinarian consisted of the use of prednisolone (1 mg/kg) over one week. The horse's clinical signs improved initially, but worsened again after three weeks with increasing episodes of syncope and progressive weight loss.

Clinical findings

Abnormalities identified on physical examination included mild edema on the ventral chest and peripheral hypoperfusion (reddish mucous membranes with a 4-second capillary refill time, cold extremities, ears and muzzle). Laboratory abnormalities included leukocytosis (17.9×10^3/L, reference range: $5–10 \times 10^3$/L), neutrophilia (16.5×10^3/L, reference range: $3.1–8.9 \times 10^3$/L), hyperlactatemia (3.5 mg/dl, reference range: <1 mg/dl) and hypoxia in arterial blood (PaO_2 89.4 mmHg, reference: 95–105 mmHg). An increase in gamma-glutamyl transferase (52.9 U/L, reference range: 7–20 U/L) was also present. Multiple measurements of cardiac troponin I on three subsequent days did not reveal an increase as had been observed by the referral vet (Troponin I measured by referral vet: 0,57 ng/ml, reference value <0.02 ng/ml). Cardiac auscultation showed a regular rhythm with a heart rate of 38 bpm. A holosystolic heart murmur, grade III/VI on the left side and I/VI on the right side, punctum maximum on the left side over the mitral valve and on the right side over the tricuspid valve was auscultated and jugular vein pulsation was noticed. A base-apex ECG revealed a complete atrioventricular dissociation with an atrial rate of 62 bpm and a ventricular rate of 38 bpm. None of the atrial impulses was conducted to the ventricles. The rhythm was maintained by a junctional escape rhythm. Furthermore, P-waves had a decreased amplitude and duration (Fig. 1). Sub-

sequently, a 24-hour continuous ECG monitor with radiotelemetry revealed a continuous dissociation of P-waves and QRS-complexes. Several hours after treatment initiation, the ECG showed a reduction in ventricular rate (19 bpm) with an almost unchanged atrial rate (64 bpm). No collapse was seen in 24 hours. Echocardiographic examination revealed an enlarged right ventricle (internal dimension at end-systole: 10.4 cm) and right atrium (internal dimension at end-systole: 9.4 cm) (Fig 2 and 3). Color flow echocardiography revealed a mitral valve regurgitation consisting of three systolic high velocity jets (Fig 4). Continuous wave Doppler recorded a peak velocity of 5.93m/s. Furthermore, the tricuspid valve (Continues wave Doppler: 2.24 m/s) and pulmonic valve (2 m/s) showed a low velocity regurgitation. M-mode tracing demonstrated a left ventricle with diameters in the reference range and a marginally paradoxical septal motion (Fig 5). An enlargement of the left atrium was noticed (Fig. 6). Thoracic radiographs revealed a hazy interstitial pattern with cuffing around the bronchi and prominent blood vessel and an increased opacity in the ventral thorax (Fig. 7 and 8). These findings were consistent with lung edema.

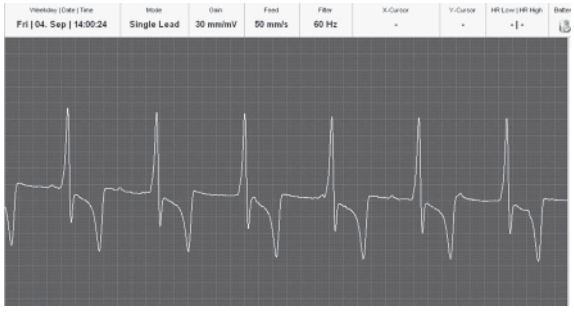

Fig. 1 A base-apex ECG revealed a complete atrioventricular dissociation with an atrial rate of 62bpm and a ventricular rate of 38bpm (Third degree AV-Block 2:1). P-waves had a decreased amplitude and duration.

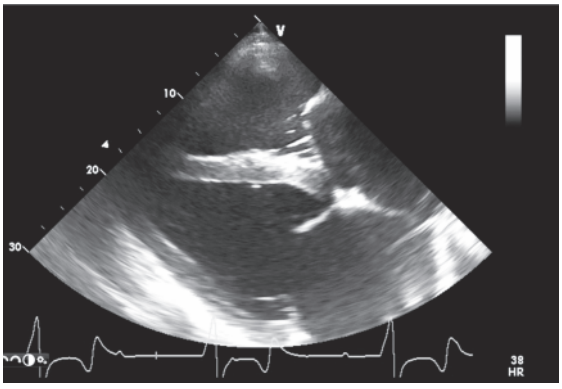

Fig. 2 Four chamber view (Left parasternal long axis view). Echocardiographic examination revealed an enlarged right ventricle and right atrium.

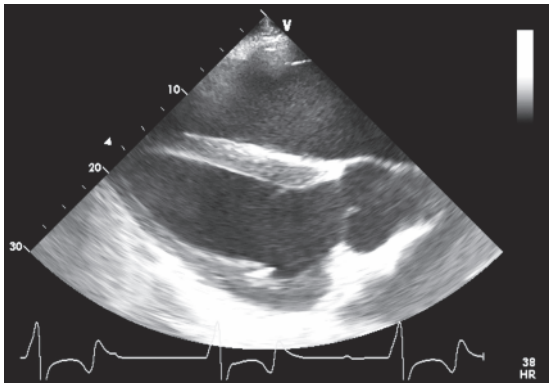

Fig. 3 Right parasternal long axis echocardiographic image of the left ventricular outflow tract and aorta. Echocardiographic examination revealed an enlarged right ventricle and right atrium.

Outcome

Clinical findings were consistent with third degree AV Block and congestive heart failure. Furthermore, the horse was suspected to suffered from myocarditis as well as mitral valve, tricuspid valve and pulmonic valve regurgitation. Treatment with furosemide (1 mg/kg, q12 h, IV), prednisolone

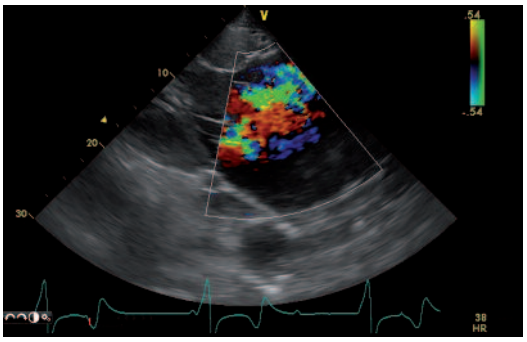

Fig. 4 Left parasternal left ventricular outflow tract. Color flow echocardiography revealed a mitral valve regurgitation consisting of three systolic high velocity jets.

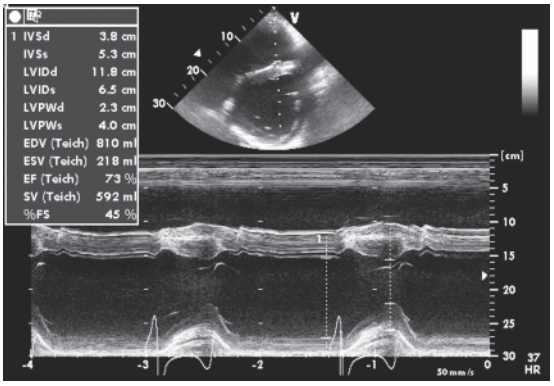

Fig. 5 Right parasternal short axis view. Anatomical M-mode tracing demonstrated a left ventricle with diameters in the reference range and a marginally paradoxical septal motion.

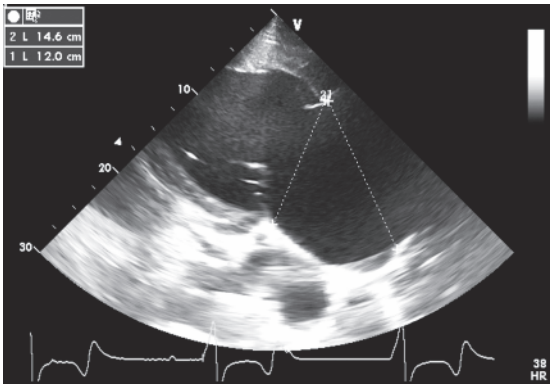

Fig. 6 Left parasternal left ventricular outflow tract. Enlargement of the left atrium.

(1 mg/kg, q24 h, PO) and trimethoprim/sulfadiazine (5mg/25mg/kg, q24h, PO) was initiated. Five days later, the horse developed ventricular tachycardia with 92 bpm (Fig. 9). Furthermore, the horse showed an increasing frequency of collapses due to ventricular asystole with a duration of 14 second duration with no visible P-waves (Fig. 10). Because of grave prognosis and rejection of a treatment with pacemaker, the horse was euthanized and a post mortem examination was performed.

Post mortem findings

At necropsy, the horse had a moderate body condition (5/9 Henneke body condition score). The subcutaneous tissue in the ventral chest region showed severe edema. In the abdominal cavity, severe ascites of approximately 20 L was found. The heart showed severe right ventricular dilatation and left ventricular hypertrophy (Fig. 11). Moreover, the horse showed dysplasia of the mitral and tricuspid valve, which were moderately thickened and both consisted of three distinct cusps (Fig. 12). The myocardium showed multifocal areas of pale discoloration. Furthermore, the lung showed severe, chronic, diffuse alveolar and moderate interstitial edema in addition to mild, acute, multifocal emphysema on the lung borders.

Histopathological examination of the pale discolored areas of the cardiac muscle revealed moderate, chronic, multifocal to coalescing, lymphoplasmacytic and fibrosing epicarditis, myocarditis and endocarditis (Fig. 13). The endocardium showed severe, diffuse subendocardial fibrosis. The thickened mitral valve cusps comprised of increased amounts of loosely arranged collagen fibers and fibrocytes and were mildly infiltrated with neutrophils and lymphocytes. The coronary vessels exhibited severe intimal and medial hyperplasia. Histopatho-

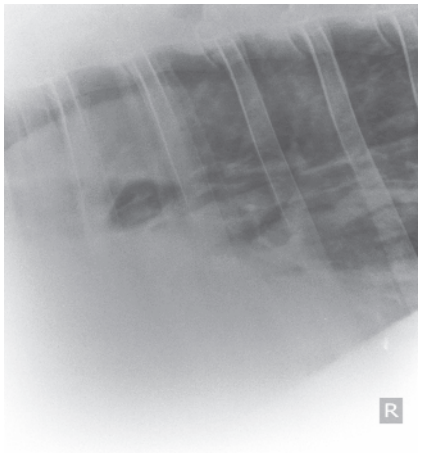

Fig. 7 A right lateral radiograph of the dorsal caudal thorax revealed a hazy interstitial pattern with cuffing around the bronchi and prominent blood vessel and an increased opacity in the ventral thorax

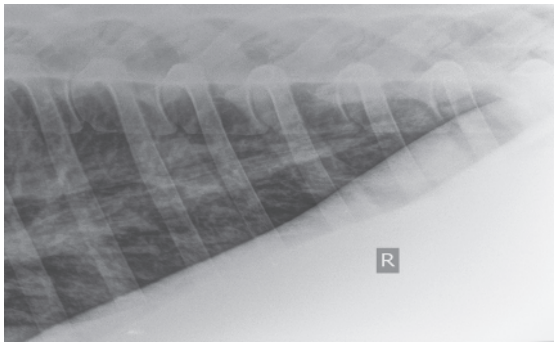

Fig. 8 A right lateral radiograph of the ventral caudal thorax revealed a hazy interstitial pattern with cuffing around the bronchi and prominent blood vessel.

Fig. 9 A base-apex ECG; ventricular tachycardia with 92bpm.

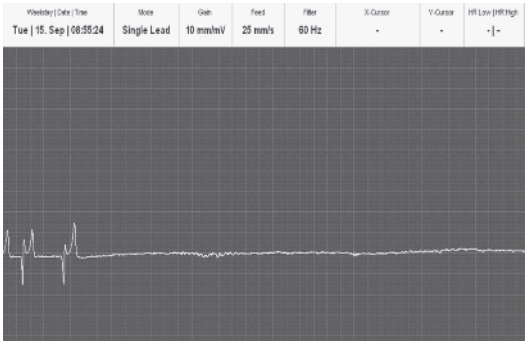

Fig. 10 A base-apex ECG; ventricular asystole up to a 14s second duration. No evident P-waves.

Fig. 11 Severe right ventricular dilatation and left ventricular hypertrophy.

Fig. 12 Dysplasia of the mitral valve with moderately thickening of the cusps and severe, subendocardial fibrosis.

logical findings of the lung included detection of several hemosiderin-laden macrophages (so-called heart failure cells) in the alveoli and moderate interstitial fibrosis consistent with left heart failure. The liver exhibited centrilobular degeneration and moderate bridging fibrosis consistent with right heart failure.

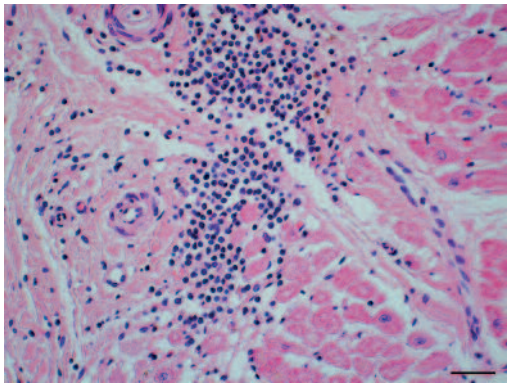

Fig. 13 Histopathological examination of the pale discolored areas of the cardiac muscle. Moderate, chronic, multifocal to coalescing, lymphoplasmacytic myocarditis.

Discussion

Third degree AV block is a rare but well studied arrhythmia in horses. Usually, third degree AV block is accompanied by bradycardia. The current case was unusual because a normal heart rate and regular rhythm was first auscultated, hiding an arrhythmia. Finally, the ECG revealed a rhythm disturbance with complete dissociation between the P-wave and QRS-complex. A differential diagnosis, which is even less common, is isorhythmic atrioventricular dissociation (IAVD).

IAVD is a condition in which the atria and ventricles do not activate in a synchronous fashion but beat independently of each other. In IAVD, atrial and ventricular depolarization occur at identical rates (*Segers* et al. 1946, 1947, *Bright* et al. 1983, *Ettinger* et al. 1968, *Perego* et al. 2012). Persistent IAVD falls into two distinct groups: in type I, the P-wave fluctuates cyclically back and forth across the QRS-complex (*Levy* and *Edflstein* 1970). In type II, the P-wave is in a fairly constant position relative to the QRS-complex. Here, the P-wave is usually coincident with the QRS-complex or appears on the ST segment or first half of the T-wave (*Levy* and *Edflstein* 1970).

On initial examination, the ECG revealed a higher atrial rate than ventricular rate. This indeed designates the arrhythmia as a third degree AV block rather than an IAVD.

Usually, patients with block at the atrioventricular nodal level, in the absence of ischemia, can benefit from sympathomimetic agents or vagolytic agents (*Whitton* and *Trim* 1985). This therapy was rejected because of the presence of a physiological heart rate and arterial hypoxia. Generally, cardiac glycosides are contraindicated in horses with third degree AV block (*Bonagura* et al. 2010). In case of permanent third degree atrioventricular block a pacemaker can be implanted. Van Loon and colleges implanted a dual-chamber, rate-adaptive pacemaker successfully in a horse with sick sinus syndrome (*van Loon* et al 2002). Pibarot and colleges implanted a programmable atrioventricular pacemaker in a donkey with complete atrioventricular block and syncope and Reef and colleges implanted a permanent transvenous pacing catheter in a horse with complete heart block and syncope. (*Pibarot* et al. 1993, *Reef* et al. 1986). In the particular case, the owner refused this treatment option because of financial constrictions.

The causes of atrioventricular dissociation in the current case can be best explained by the inflammation of the myocardium and conduction tissue, fibrosis of the myocardium, and marked enlargement of the right atrium. Other potential causes are severe drug toxicity, rattlesnake envenomation, loss of connection in portions of the healthy conduction system and abnormal high vagal activity but there was no evidence for those causes in the present case (*Whitton* and *Trim* 1985, *Reef* et al. 1986, *Pibarot* et al. 1993, *Lawler* et al. 2008).

A certain change in arrhythmia occurred a few days later. The horse developed ventricular tachycardia with episodes of asystole. To distinguish between supraventricular and ventricular tachyarrhythmia, the location of the P-wave relative to the QRS-complex is important. When there is clear AV dissociation (slower, independent atrial rhythm), a junctional or ventricular tachycardia is most likely. In the present case, there was, prior to ventricular tachycardia, a complete AV dissociation, making it doubtless that the developed tachyarrhythmia is of ventricular or junctional origin. The separation of junctional and ventricular rhythms can sometimes be made by inspection of the QRS-complex. Junctional impulses are more likely to result in a narrow, relatively normal-appearing QRS-complex with normal initial activation and electrical axis (*Bonagura* et al. 2010). This is because they originate above the ventricular myocardium. Complexes of ventricular origin are conducted abnormally and result in a widened QRS-complex, an abnormal QRS-orientation and an abnormal T-wave (*Bonagura* et al. 2010). The QRS-T complex in the present case matches most to the last condition. The change to ventricular tachyarrhythmia might be explained by progressive hypoxemia due to biventricular cardiac failure including progressive lung edema. Hypoxemia leads to increasing damage and necrosis of the myocardium, pacemaker and conducting tissue with further fibrosis, creating a vicious cycle. Therefore the ventricular tachycardia can be initiated by ongoing ischemia and damage in pacemaker tissue. The consequence is a reduction of the resting potential with the subsequent inactivated state of some sodium channels. The latter can lead to increased excitability of pacemaker tissue leading to rhythm disturbance.

Unfortunately, the cause of myocarditis could not be determined. Under certain circumstances, diagnosing rhythm disturbance can be challenging and determining the cause, especially in chronic cases, even more. Ongoing inflammation of the heart can lead to further and dramatically changes in heart rhythms. Therefore, a continuous monitoring of patients as well as a thoughtful selection of treatment is of great importance.

Literature

Bonagura J. D., Reed M. S., Bayly W. M., Sellon D. C. (2010) Equine internal medicine. Third Edition, Sounders Elsvier; p. 425 and 463

B*right J. M., Lombard C. W.* (1983) ECG of the month: Atrioventricular junctional tachycardia producing atrioventricular dissociation. J. Am. Med. Assoc. 182, 580–581

Ettinger S., Buergelt C. D. (1968) Atrioventricular dissociation with accrochage in a dog with ruptured chordate tendinae. Am. J. Vet. Res. 29, 1499-1503

Harrigan R. A., Perron A. D., Brady W. J. (2001) Atrioventricular dissociation. Am. J. Emerg. Med. 19, 218-222

Lawler J. B., Frye M. A., Bera M. M., Ehrhart E. J., Bright J. M. (2008) Third-degree atrioventricular block in a horse secondary to rattlesnake envenomation. J. Vet. Intern. Med. 22, 486-490

Levy M. N., Edflstein J. (1970) The mechanism of synchronization in isorhythmic AV dissociation. II. Clinical studies. Circulation 42, 689-699

Pibarot P., Vrins A., Salmon Y. (1993) Implantation of a programmable atrioventricular pacemaker in a donkey with complete atrioventricular block and syncope. Equine Vet. J. 25, 248-251

Perego M., Ramera L., Santilli R. A. (2012) Isorhythmic Atrioventricular Dissociation in Labrador Retrievers J. Vet. Intern. Med. 26, 320-325

Reef V. B., Clark E. S., Oliver J. A. (1986) Implantation of a permanent transvenous pacing catheter in a horse with complete heart block and syncope. J. Am. Vet. Med. Assoc. 189, 449-452

Segers M. (1946) Les phenomenes de synchronization au niveau du coeur. Arch. Int. Physiol. 54, 87-95

Segers M., Lequime J., Denolin H. (1947) Synchronization of auricular and ventricular beats during complete heart block. Am. Heart. J. 33, 685-692

van Loon G., Fonteyne W., Rottiers H., Tavernier R., Deprez P. (2002) Implantation of a dual-chamber, rate-adaptive pacemaker in a horse with suspected sick sinus syndrome. Vet. Rec. 151, 541-545

Whitton D. L., Trim C. M. (1985) Use of dopamine hydrochloride during general anesthesia in the treatment of advanced atrioventricular heart block in four foals. J. Am. Vet. Med. Assoc. 187, 1357-1361