Clinical and electrocardiographic characterization of cattle with atrial premature complexes

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Summary: Atrial premature complexes (APC) were identified in 16 cows over a 2-year period. Fourteen cows had concurrent gastrointestinal disease. Variation in the intensity of the first heart sound and an occasionally irregular heart rhythm were evident during thoracic auscultation. Neither cardiac murmurs nor pulse deficits were detected in any cows, and clinical signs of heart failure were lacking. Three cows had APC immediately prior to or after development of atrial fibrillation.

The heart rate when APC were diagnosed ranged from 48 to 124 beats/min (mean, 77 ± 20 beats/min), and the APC frequency ranged from < 1 to 23/ min (mean 9.4 ± 8.0). The P-wave morphologic characteristics in 4 cows with APC was abnormal. The coupling index of the APC varied between 0.44 and 0.95, with a mean of 0.73. Aberrant ventricular activation was usually associated with a short coupling interval (coupling index < 0.60) and was observed in 3 cows.

Ten cows were determined to be hypocalcemic and 4 cows hypokalemic when APC were identified. Atrial ectopic activity could not be detected in 12 cows after resolution of the concurrent gastrointestinal disorder or electrolyte abnormality. Atrial premature complexes may be a functional cardiac disorder in cattle, unrelated to structural heart disease. The potential for APC to progress to sustained atrial arrhythmias such as atrial fibrillation should be considered.

The most commonly reported cardiac arrhythmia in cattle is atrial fibrillation (AF). Supraventricular arrhythmias other than AF are rarely reported in cattle, although they may occur at a greater frequency than the literature would suggest. Sinus arrhythmia apparently is rare in adult cattle, and few reports of sinus, atrial, or junctional tachycardia exist. Atrial premature complexes (APC) have been previously identified in 4 cows, 3 of which had APC before or after resolution of AF. APC were diagnosed in 16 cows with AF in previous studies, the purpose of the study reported here was to determine the clinical, electrocardiographic, laboratory findings, and treatment outcome of 16 cows with APC.

Materials and Methods

All cattle admitted to our teaching hospital between Jan 1, 1987 and Dec 31, 1988 were evaluated for cardiac rhythm disturbances. Cattle were included in this study if and only if cardiac arrhythmias first identified during thoracic auscultation were confirmed to be APC by electrocardiography. Sixteen cows were determined to have APC. Records were kept of cattle diagnosed with other supraventricular arrhythmias. A standard bipolar (base-apex) lead was used to evaluate heart rate and rhythm. The left forelimb lead (+ electrode) was placed on the left thoracic wall at the cardiac apex, and the right forelimb lead (− electrode) was placed in the right jugular groove, one third of the distance from the manubrium to the mandible. The ECG was calibrated (10 mm = 1 mV) and recordings were taken for up to 4 minutes, with a minimal duration of 30 seconds. Heart rate, P and QRS wave duration and morphologic characteristics, PQ interval, and APC frequency were determined from ECG tracings taken at a paper speed of 25 mm/s. The atrial premature complex (P' wave) duration and P'Q interval also were measured. Wave and segment duration were measured 6 times in each cow and the mean value recorded. The APC frequency per minute was estimated by determining the number of atrial premature complexes on the entire ECG. The relative prematurity of the ectopic beat was examined by determining the coupling index. The prematurity of an individual APC was expressed as the ratio of the coupling interval to the preceding cycle length, or:

\[ \text{coupling index} = \frac{\text{coupling interval}}{\text{preceding cycle length}} = \frac{P2 - P'}{P1 - P2} \]

where P' represents the APC, P2 the immediately
preceding atrial discharge, and P1 the atrial discharge immediately preceding P2.\(^8\) When the APC was buried in the preceding T wave, this formula was modified to:

\[
\text{coupling index} = \frac{\text{coupling interval}}{\text{cycle length}} = \frac{R2 - R'}{R1 - R2}
\]

where R' represents the QRS complex following the APC, R2 immediately preceding QRS complex, and R1 the QRS complex immediately preceding R2.

Blood samples for blood gas, pH, and serum biochemical analysis were obtained within 2 hours of identification of APC. Blood samples were collected at the discretion of the primary care clinician and were not obtained from all cows. Blood gas analysis was performed on blood obtained anaerobically into a heparinized syringe from either the coccygeal or jugular vein. Analysis was completed within 5 minutes of sampling, using an automated blood gas analyzer.\(^c\) Serum was obtained from the coccygeal or jugular vein for biochemical analysis.\(^d\)

Treatment in 14 cows was directed at the primary gastrointestinal problem and included appropriate treatment with Ringer solution given IV or calcium borogluconate given SC. Antibiotics were administered parenterally to cows with metritis, pneumonia, or peritonitis.

Hearts from 2 cows that were euthanatized were examined grossly at necropsy. Histologic examination of the atrial myocardium of one cow was done.

Data are reported as mean ± SD. Statistical comparison of P and P' wave duration, as well as PQ and P/Q interval, was by Student t test, with a significance level of \(P < 0.05\).

**Results**

Atrial premature complexes were diagnosed in 16 cows, representing 0.7% of the bovine population or 2.0% of the dairy cows admitted to this hospital. The age of affected cattle ranged from 2 to 12 years, with a mean of 5.4 ± 2.5 years. Thirteen cows were Holsteins and 3 were Jerseys. Gastrointestinal disease was usually diagnosed when APC were diagnosed. The abdominal disorders included left displaced abomasum (LDA, 7 cows), abomasal volvulus (2 cows), abomasal impaction (1 cow), cecal volvulus (2 cows), peritonitis (1 cow), and ileus (1 cow). Some of these cattle also had metritis (2 cows), pneumonia, or peritonitis. Atrial premature complexes were also diagnosed in 2 aged Jersey cows without apparent gastrointestinal disease. The heart rate when APC were diagnosed ranged from 48 to 124 beats/min (mean, 77 ± 20 beats/min), and the APC frequency ranged from <1 to 23/min (mean, 9.4 ± 8.0). Atrial fibrillation was diagnosed in 18 cows, sinus arrhythmia identified in 7 cows, and 2nd degree atrioventricular (AV) block diagnosed in 2 calves over the same 2-year period.

Auscultatory findings were similar in all cows. An occasionally irregular heart rhythm was identified with varying intensity of the first heart sound (S1). The S1 of the APC was usually louder than the S1 of the preceding or following sinus beat. The only auscultatory abnormality in 2 cows with sinus bradycardia was prolongation of the interval between the S2 of the APC and the subsequent S1. In these 2 cows, the coupling interval of the APC was relatively long (0.87 to 0.95), making it difficult to detect that the S1 associated with the APC was earlier than expected. The prolongation was not attributed to nonconducted atrial extrasystoles. Neither cardiac murmurs nor clinical evidence of heart failure was detected in any cow. A decrease in aortic pulse amplitude associated with the APC could be detected in some cows during palpation per rectum. Pulse deficits were not detected.

The P wave duration (88 ± 10 ms), QRS wave duration (93 ± 12 ms), and PQ interval (211 ± 30 ms) appeared to be within normal limits for lactating cows.\(^9\) The P' wave duration (112 ± 17 ms) and P/Q interval (219 ± 29 ms) were not substantially different from the respective P wave and PQ interval for any of the 4 cows in which it could be determined. The morphologic characteristics of the APC were abnormal in 4 cows (Fig 1), suggesting aberrant atrial depolarization. In the other 12 cows, the premature atrial complex was consistently buried in the T wave, making it impossible to accurately assess its morphologic features (Fig 1). The pause following the conducted P' wave was less than compensatory in all cases, indicating penetration and resetting of the sinus node.\(^10\)

Aberrant ventricular activation initiated by the atrial premature depolarization was seen in 3 cows (Fig 2) and was observed more frequently when the coupling index was less than 0.60. Premature ventricular complexes were not observed.

The coupling index varied markedly between cows (ranging between 0.44 and 0.95), with an overall mean of 0.73. The coupling index also varied for sequential APC in all cows; however, in 7 cows this variation was small (<0.1).

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\(^a\)Institutional Laboratory System 1302, Lexington, Mass.
\(^b\)Dacos, Coulter Electronics, Hialeah, Fl.
Atrial fibrillation preceded or immediately followed APC in 3 cows (No. 1 to 3). Cow 1 was determined to have a cardiac arrhythmia on initial examination and AF was confirmed electrocardiographically. Two days later, the beat had converted to normal sinus rhythm (NSR), with an APC frequency of 1/min. Atrial premature complexes were not detected on subsequent days. Two days after APC were identified, cow 2 developed AF. Prior to the development of AF, electrocardiography revealed episodes of paroxysmal supraventricular tachycardia and numerous APC (coupling index 0.71 to 0.93; Fig 3). Atrial fibrillation persisted for 2 days before the cow’s heartbeat converted to NSR. Cow 3 was determined to be severely hypocalcemic (serum Ca, 4.3 mg/dl) following surgical cor-

Figure 1—Base-apex lead ECG of 3 cows with atrial premature complexes. Paper speed, 25 mm/s; 10 mm = 1 mV.
A—Ten-year-old Jersey cow with chronic mastitis. Notice the variation in P’ wave morphologic characteristics.
B—Four-year-old Holstein cow with left displaced abomasum and metritis. Notice the prolonged P’ wave.
C—Six-year-old Holstein cow with cecal volvulus and hypocalcemia. Notice burying of the P’ wave in the preceding T wave and prolonged QT interval.

Figure 2—Base-apex lead ECG of a 4-year-old Holstein cow with left displaced abomasum. The premature atrial complex (P1’) has a coupling index of 0.44, leading to aberrant ventricular activation and abnormal QRS and T morphologic characteristics. A later premature atrial complex (P2’) has a longer coupling index of 0.74 and does not cause aberrant ventricular activation. Paper speed, 25 mm/s; 10 mm = 1 mV.
Figure 3—Base-apex lead ECG of a 5-year-old Holstein cow with a markedly arrhythmic heart detected on auscultation. Notice multiple atrial premature complexes, with one episode of paroxysmal supraventricular tachycardia (SVT) with an accelerating rhythm. Paper speed, 25 mm/s; 10 mm = 1 mV.

Figure 4—Base-apex lead ECG of a 6-year-old Holstein cow with cecal volvulus and hypocalcemia. Paper speed, 25 mm/s; 10 mm = 1 mV.
A—Atrial premature complexes with prolonged QT interval.
B—Atrial fibrillation in the same cow 1 hour after intravenous infusion of calcium borogluconate. Notice the irregular R-R intervals, and the lack of P waves, and normal QT interval.
rected by adrenocortical stimulation. The APC coupling index varied from 0.68 to 0.90 and the QT interval was prolonged (480 ms; Fig 4). Calcium borogluconate (21.4 g of calcium) was administered by slow intravenous injection, but administration was stopped when the heart rate became markedly arrhythmic. Electrocardiography performed 1 hour later revealed AF, normal QT interval, and the serum calcium concentration at that time was 7.0 mg/dl. Additional calcium borogluconate was administered SC. The next day, the cow appeared bright and alert. The heart had a regular rhythm, and serum electrolyte concentrations were normal. Electrocardiography revealed NSR, and APC were not observed. Normal sinus rhythm was maintained until the cow was discharged 3 days later.

Ten of the 11 cows that were lactating were determined to be hypocalcemic, and 4 of these cows were also hypokalemic. Four cows were alkalotic (blood pH > 7.45).

Surgical correction of an abomasal volvulus (2 cows), LDA (3 cows), and cecal volvulus (1 cow) was performed immediately following diagnosis, via a right flank celiotomy done under a regional nerve block. Ectopic atrial activity could not be detected in 2 of these cows after they became excited during preparation for surgery. In addition, ectopic atrial activity could no longer be detected in another 2 cows within 48 hours of surgery. The remaining 2 cows were treated on an outpatient basis, and one was reported by the referring veterinarian to have a regular cardiac rhythm 5 days later. Treatment outcome in the other cow was lost to follow up.

Three cows developed APC within 2 days of surgical correction of their gastrointestinal condition. The cardiac rhythm was regular in all 3 cows prior to discharge.

Three cows (No. 4 to 6) with LDA were treated medically before surgical correction was performed. Cow 4 was hypocalcemic (6.3 mg/dl) and hypokalemic (2.5 mEq/L) on admission, but atrial ectopic activity resolved within 24 hours of admission, at which time the serum electrolyte concentrations were normal. The LDA was subsequently corrected surgically. Atrial premature complexes were not detected in cow 5 within 24 hours of hospitalization. This cow was euthanatized 3 days later because of pneumonia and toxic metritis that were unresponsive to treatment. Surgical correction of the LDA was not attempted. The heart of this cow appeared normal at necropsy. In cow 6, APC persisted for 6 days after surgical correction of the LDA. Peritonitis was identified at surgery, and the cow’s clinical condition deteriorated until she was euthanatized at the owner’s request. The heartbeat of this cow appeared normal grossly, although sarcoceysts were observed on histologic examination.

Discussion

Atrial premature complexes appear to be more common in dairy cows than previously reported and are frequently associated with gastrointestinal disease. Atrial premature complexes were identified in 16 cows during the 2 years of this study, a frequency similar to that of AF (18 cows). The relatively high number (16) of cattle with APC in this study contrasts with low numbers previously reported in cattle with cardiac arrhythmias. We could not determine any obvious reason for this difference.

The typical auscultatory finding in cattle with APC is an occasionally irregular heart rhythm accompanied by variations in the intensity of S1. Increased intensity of the S1 associated with the APC is thought to result from the lack of partial presystolic closure of the AV valve. Prolonged auscultation of the thorax (> 2 minutes) may be required in some cattle so that the arrhythmia can be detected because of variation in APC frequency. Electrocardiography is required to confirm the diagnosis of atrial premature contractions because APC cannot be reliably differentiated from ventricular premature contractions on auscultation.

Although atrial premature complexes represent an electrical event and atrial premature contraction signifies a mechanical event, the 2 terms are often used interchangeably. We have used APC in this report to signify atrial depolarization.

The characteristic electrocardiographic sign of an APC is a premature, abnormal P’ wave (P’). The abnormal morphologic features of the P’ wave result from initiation and propagation of atrial depolarization from outside the sinus node. Atrial premature complexes are usually conducted normally through the ventricular myocardium, resulting in a normal QRS-T complex. However, premature impulses can enter the ventricle when it is still partially refractory, resulting in abnormal QRS and T configurations. Moreover, APC may not be conducted into the ventricle if the AV node is refractory to premature stimulation. A nonconducted APC causes a pause between ventricular beats (S1-S1) that is longer than the prevailing sinus R-R interval. Nonconducted APC were not observed in cows in this report; however, aberrant ventricular activation occurred occasionally and usually followed a P’ wave with a short coupling index.

The sinus node can respond in 4 ways (interference, reset, interpolation, or reentry) to APC, depending on the relative prematurity of the APC and whether the APC penetrates the sinus node. APC causes a pause between ventricular beats (S1-S1) that is longer than the prevailing sinus R-R interval. Nonconducted APC were not observed in this report; however, aberrant ventricular activation occurred occasionally and usually followed a P’ wave with a short coupling index.

Atrial premature complexes can result from a number of mechanisms. A reentrant mechanism is mentioned as the most common cause of APC in
human beings, although abnormal impulse formation, primarily by enhanced automaticity of subsidiary atrial pacemakers, may also result in APC. The association of APC with gastrointestinal disease in cattle suggests a possible role for increased vagal tone in the cause of APC. In human beings, gastrointestinal disorders such as diarrhea, nausea, and vomiting have been associated with vagotonia and supraventricular arrhythmias. In this study, abomasal distention (associated with left displacement, impaction, or volvulus of the abomasum) was observed in a number of cows when APC were identified. Vagotonia probably resulted from this distention, because gastric distention has been shown to cause vagally mediated bradycardia in dogs and rats. Depression of the sinus node by increased vagal tone could allow subsidiary atrial pacemakers to depolarize at a faster rate, resulting in ectopic atrial activation. In human beings, APC can disappear with increases in heart rate (following exercise or atropine administration) suggesting maneuvers that increase heart rate can override suppress the APC locus. This response may explain the disappearance of atrial ectopic activity in 2 cows of this report after they were excited. Vagal stimulation also decreases the effective refractory period of the atria in a nonuniform manner, leading to an increase in atrial heterogeneity. Reentry is facilitated by temporal or spatial dispersion of atrial refractoriness; however, APC arising from reentry should have a fixed coupling interval, which was not observed in all cows in this report.

Increased vagal tone has been suggested as favoring the induction and perpetuation of AF. We have recently reported on the development of AF in 3 cows during treatment with neostigmine, an anticholinesterase agent that enhances vagal activity. The induction of AF by intravenous infusion of calcium borogluconate in a cow in this report (No. 13) further supports the role of increased vagal tone in the genesis of AF in cattle. Hypercalcemia-induced arrhythmias in cattle are thought to result from vagal stimulation, because these arrhythmias (usually bradyarrhythmia and AV block) are abolished by the administration of atropine. Atropine was not administered to cow 3 in this report because of its prolonged inhibitory effects on gastrointestinal motility.

Hypokalemia and sympathetic stimulation can enhance diastolic depolarization in subsidiary atrial pacemaker fibers, leading to ectopic atrial activity. Hypokalemia increases the slope of phase-4 diastolic depolarization, slows conduction, and increases the sensitivity of myocardial tissue to vagal stimulation. Four cows in this report were determined to be hypokalemic when APC were diagnosed, suggesting that hypokalemia, particularly when accompanied by increased vagal tone, can lead to APC in cattle. Sympathetic stimulation increases the rate of phase-4 depolarization in the sinus node and subsidiary pacemakers, although the effect has been suggested to be more pronounced in the latter. The finding in this study that the frequency of APC was generally much higher in cows with tachycardia suggests that catecholamines may have a role in the development of APC in cattle. In addition, alkalalemia in 4 cows may have augmented the cardiac response to hypokalemia and sympathetic stimulation. Arhythmias associated with alkalalemia are usually supraventricular in origin and are thought to result from the concomitant development of hypokalemia. Alkalalemia also alters the myocardial sensitivity to circulating catecholamines, potentiating their effect in generating arrhythmias. Finally, although hypocalcemia was commonly identified in cows with APC, the serum calcium concentration was not considered low enough to be a factor in the origination of APC.

Atrial premature complexes can probably be considered a functional arrhythmia in cattle with gastrointestinal disease. The disappearance of the arrhythmia after resolution of the gastrointestinal disorder and the lack of clinical signs of heart disease or demonstrable lesions in the 2 cows examined at necropsy support a functional cause. However, APC were also identified in 2 aged Jersey cows that did not have gastrointestinal disease. These 2 cows may have had cardiac lesions, although clinical signs of heart disease were not detected. In dogs and horses, APC are often associated with cardiac lesions. However, atrial arrhythmias can develop in people without detectable heart disease.

Specific antiarrhythmic treatment of APC in cattle does not appear warranted unless hemodynamic compromise is evident. Treatment should be directed at correction of electrolyte and metabolic abnormalities (ie, hypokalemia and alkalalemia) and resolution of any gastrointestinal disease. The association between APC and AF observed in this and other studies has clinical importance. In human beings, calcium borogluconate may follow paroxysmal supraventricular tachycardia or one properly timed APC. Although the coupling index before the development of AF in 2 cows in this report was greater than that reported to induce AF in human beings, monitoring of the cardiac rhythm over a longer period may have identified shorter coupling intervals. Additionally, paroxysms of supraventricular tachycardia were observed in one cow that subsequently developed AF.

We found that APC occur as commonly as AF in adult dairy cattle and are frequently associated with gastrointestinal disease. In addition, both APC and paroxysmal supraventricular tachycardia need to be identified in cattle because of the possibility of the development of AF. We hypothesize that in-
creased vagal tone may be an important factor in the development of supraventricular arrhythmias in cattle.

References