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Effects of intramuscular sedation with alfaxalone and butorphanol on echocardiographic measurements in healthy cats

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Abstract

Objectives The aim of the study was to evaluate the effects of intramuscular (IM) injections of alfaxalone combined with butorphanol on echocardiographic (ECG) measurements in cats.

Methods Client-owned adult domestic shorthair cats younger than 5 years of age were recruited. All cats that were considered healthy on the basis of physical examination, blood work, urinalysis, blood pressure measurement and baseline ECG underwent a second ECG under sedation. Cats were sedated with two separate IM injections of butorphanol at 0.2 mg/kg and alfaxalone at 2 mg/kg. ECG variables were analysed using a linear mixed model, and sedation scores were analysed using an ordinal mixed logistic model. The significance level was set at $\alpha = 0.05$ and adjusted at $\alpha = 0.0017$ for multiple comparisons of the ECG measurements.

Results Ten healthy cats were included. Sedation was uneventful, and recovery was smooth and quick for all cats. The mean duration of lateral recumbency was 36.3 ± 4.37 mins. Reduction in heart rate following sedation approached statistical significance ($P = 0.002$). The thickness of the interventricular septum, the thickness of the left ventricular free wall, and the left ventricular internal dimensions in diastole and systole were not affected by the sedation. The changes in left atrium/aortic ratio and shortening fraction were statistically significant. Although the peak velocity of early diastolic transmitral flow (E) and late diastolic transmitral flow (A), the peak early diastolic (Ea) mitral valve annulus velocity, and the peak late diastolic (Aa) mitral valve annulus velocity changed after sedation, the ratios E/A, E/Ea and Ea/Aa were not significantly different after sedation.

Conclusions and relevance IM injections of alfaxalone and butorphanol induced rapid, deep and short-lasting sedation. The mean differences after sedation were not clinically significant for most echocardiographic measurements.

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Introduction

A complete echocardiographic (ECG) examination in cats usually lasts 10–20 mins. It requires one or two assistants to restrain the cat in lateral recumbency, and sedation is commonly used for the most fractious animals. Occult forms of feline cardiac diseases, such as hypertrophic cardiomyopathy, may not be detectable on physical examination alone.[1] It is therefore critical that sedatives administered to cats with a suspicion of cardiac disease have limited impacts on their cardiovascular system. Moreover, in order to be clinically useful, these agents should be easy to administer, have a short duration of action and, importantly, they should minimally alter ECG measurements compared with measurements obtained in non-sedated cats.

Alfaxalone is a synthetic neurosteroid anaesthetic currently approved for intravenous (IV) injection in France,

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Table 1 Sedation score system

Posture score
0 Able to walk/run normally
1 Moderate ataxia, able to stand up and to walk
2 Lateral/sternal recumbency, stands up under stimulation
3 Lateral recumbency, not able to stand up
Behaviour
0 Normal response to contact with assessor*
1 Slower response to assessor than normal*
2 Minimal response to assessor contact*
3 No response to assessor*
Muscle relaxation
0 Normal jaw and leg tone
1 Mild relaxation of jaw and leg tone (resistance to flexion of the limbs and opening of the mouth is observed)
2 Moderate relaxation and leg tone (resistance to flexion of the limbs or opening of the mouth is observed)
3 Profound relaxation of jaw and leg tone (no resistance to flexion of the limbs and opening of the mouth is observed)

*Contact with assessor included auditory stimulus (hand-clap close to ear) and mechanical stimuli (assessment of capillary refill time, palpation of femoral pulse and rectal thermometer placement)

Germany, New Zealand, South Africa, Canada and the UK. In Australia, it is also approved for intramuscular (IM) use. By this route of administration, it induces rapid and short-lasting sedation without evidence of any serious complications.[2,3] In addition, its effects on cardiovascular haemodynamic measurements are dose-dependent but minimal at clinically relevant dosages in healthy cats, suggesting that it might safely be used in cats with unknown cardiac status.[2,4–7] Although the pharmacological characteristics of alfaxalone appear interesting when sedation is required to complete an ECG examination, its effects on ECG variables have not been evaluated.

Therefore, the aim of this study was to evaluate the effects of an IM injection of alfaxalone combined with butorphanol on standard ECG measurements. The biological hypothesis was that sedation with butorphanol and alfaxalone would significantly alter some ECG measurements in healthy cats.

Materials and methods

Animals

The study was approved by the ethics committee of Vetagro Sup, Lyon, France. Client-owned adult domestic shorthair cats, younger than 5 years of age, were recruited with owners' consent. Each cat underwent a complete physical examination; complete blood count; blood chemistry profile, including serum cardiac troponin I to rule out myocarditis; urinalysis; and non-invasive blood pressure measurement by the Doppler method. Baseline ECG was performed without sedation. Cats with any functional or anatomical abnormalities were excluded from the study. Cats that were considered healthy on the basis of physical examination, laboratory tests and the first ECG underwent the second ECG examination under sedation 1 week later.

Sedation

Butorphanol at 0.2 mg/kg (Dolorex; Intervet) and alfaxalone at 2 mg/kg (Alfaxan; Vétoquinol) were slowly injected, using a 25 G needle, as two separate injections in the lumbar muscles. The cats were then placed in a quiet room and continuously monitored until the adequate level of sedation was obtained. A scoring system evaluating posture, behaviour and degree of muscle relaxation was used to describe the level of sedation (Table 1).[8–11]

All variables were evaluated at the time of injections, and after 2, 5 and 10 mins. The ECG was started when the cats had reached a score of 3 for posture, or 10 mins after injections if the cat had not yet reached this score. Time from injections to lateral recumbency, duration of lateral recumbency and time to recover from sedation, defined as the cat being ambulatory, were also recorded.

ECG

Cats were gently restrained in right and then left lateral recumbency on a customised ECG table with the front legs extended towards the head during the ECG examinations. Two areas on the left and right side of the thorax were clipped to improve image quality. ECGs were performed by the same operator (IB) using an Aloka Alpha 10 prosound unit (Hitachi) according to published recommendations.[12] An ECG synchronised with the ECG images was recorded. At the end of the study period, one investigator (IB) selected the ECGs in a random order and masked the animals' identifier and the date before submitting them to a single blinded observer (TR), who performed all the measurements. Heart rate (HR) was calculated 10 mins after the beginning of each ECG examination from the ECG displayed on the ECG monitor.

Left atrial (LA) and aortic (Ao) diameters were measured with digital calipers from a right parasternal trans-aortic short-axis view at the end of ventricular systole from a two-dimensional still image. Left ventricular end-diastolic (LVIDd) and end-systolic (LVIDs) dimensions, end-diastolic (LVFWd) and end-systolic (LVFWs) left ventricular free wall thicknesses and end-diastolic (IVSd) and end-systolic (IVSs) interventricular septum thicknesses were measured on an M-mode recording obtained from a right parasternal trans-ventricular short-axis view. The left ventricular fractional shortening (FS) was calculated as a percentage according to the following formula: FS = 100 × (LVIDd–LVIDs)/LVIDd. Right ventricular dimensions at end-diastole and end-systole were assessed on the same M-mode images used for left ventricular measurements. Ejection fraction (EF) was calculated as a percentage from the formula EF = 100 × (LVEDV–LVESV)/LVEDV with left ventricular end-diastolic (LVEDV) and end-systolic volumes (LVESV) obtained with the modified single plane Simpson's method from a right parasternal four-chamber long-axis view.[13]

The maximal velocity of blood flow in the right ventricular outflow tract (Vmax.RVOT) was measured by pulsed-wave Doppler from a right parasternal short-axis view. The maximal velocity of blood flow in the left ventricular outflow tract (Vmax.LVOT) was estimated by pulsed-wave Doppler from a left parasternal apical view. The LVOT spectral Doppler profile and the simultaneous ECG tracing were used to measure left ventricular ejection time (ET) and left ventricular pre-ejection period (PEP). Mitral inflow, recorded by pulsed-wave Doppler from a left apical four-chamber view, was used to estimate the peak velocity of early diastolic transmitral flow (E) and late diastolic transmitral flow (A). Finally, isovolumic relaxation time (IVRT) was calculated as the time between the end of LVOT spectral Doppler signal and the onset of the mitral E wave.

Tissue Doppler imaging (TDI) was used to measure mitral valve annulus velocities from a left apical four-chamber view. The sample was placed successively on the septal and the lateral mitral valve annulus in order to measure the following variables: peak early diastolic lateral (Ea Lat) and septal (Ea Sept) mitral valve annulus velocity, peak late diastolic lateral (Aa Lat) and septal (Aa Sept) mitral valve annulus velocity. Based on these measurements, the following ratios were calculated: E/A, E/Ea Lat, E/Ea Sept, Ea Lat/Aa Lat, Ea Sept/Aa Sept.

All measurements were repeated on three different images and then averaged, except for LVEDV and LVESV, which were measured on five images and averaged.

Statistical analysis

Repeated measures were analysed using a linear mixed model with ECG measurements as the outcome variables, sedation (dummy variable) as the fixed effect and cats as the random effect. Assumptions of linearity, normality and homoscedasticity of the residuals were checked on residual plots. The significance level was set at alpha = 0.05 and corrected with the Šidák equation at alpha = 0.0017 for ECG measurements because of multiple comparisons.

A coefficient of reliability (CR) was calculated for the variables evaluated as the proportion of the variance explained by the intraindividual variability: $\sigma_{cats}/(\sigma_{cats}+\sigma_{residual})$. It is equivalent to an intraclass correlation coefficient and measures the consistency of measurements within the same cat. Reliability was considered high when the coefficient value was $\geqslant$0.76, medium between 0.40 and 0.75, and low if $\leqslant$0.39.[14]

Posture, behaviour and muscle relaxation scores were analysed using an ordinal mixed logistic model. The significance level was set at alpha = 0.05 for those variables.

Normally distributed data are presented as mean ± SD. The mean difference is used to report the difference after sedation. Non-normally distributed variables are presented as median and range.

R statistical software (R Foundation) was used for statistical analysis with its related package for mixed modelling (R package 'nlme' [R package version 3.1-103]) and ordinal logistic mixed models ('ordinal' [R package version 2012.09-11]).

Results

Twelve cats were initially recruited. Two cats were excluded because of concurrent disease diagnosed at the time of inclusion: one cat had anaemia secondary to a myelodysplastic syndrome and one was equivocal for hypertrophic cardiomyopathy. The median age of the remaining 10 cats (seven females and three males) was 34.5 months (range 10–56 months) and the median body weight was 4.23 kg (range 3.2–5.8 kg).

There was a significant increase in posture, behaviour and muscle relaxation scores over time following drug injections (P <0.05 for all time points when compared with T = 0 mins). Signs of sedation were detected as early as 2 mins following injections in six cats and in three additional cats after 5 mins. Two cats reached a score of 3 for posture at 5 mins, whereas in six more cats it was recorded at 10 mins post-injection; in one cat, the posture score was 2 at 10 mins. Excluding the one cat that did not display signs of sedation within the first 10 mins following injections, but was moderately ataxic (posture score of 1) after 10 mins, the mean time from injection to recumbency was 7.5 ± 2.8 mins and the mean duration of recumbency was 36.3 ± 4.4 mins. Time from injection to ability of the cats to resume a standing position was 44.1 ± 5.1 mins. All cats had a smooth recovery, and no adverse effects were observed during the study.

Mean HR before sedation was 159 ± 24 beats per min. Overall, sedation did not have an effect on HR, although an apparent mild reduction in HR approached statistical significance ($P < 0.002$). The amplitude of HR variation after sedation differed between cats, which was reflected by a low CR (0.36). Indeed, HR decreased in nine cats and increased in only one, which was also the cat with a score of 2 on the posture scale at 10 mins.

All ECG views and measurements could be recorded in the 10 cats before and after sedation (Table 2).

On M-mode, mean IVSd, IVSs, LVFWd and LVFWs did not significantly change in sedated cats. However, individual values decreased in four cats and increased in six. Mean LVIDd and mean LVIDs were not altered by sedation. As for individual values, LVIDd increased and LVIDs decreased in three cats; in the seven other cats LVIDd decreased and LVIDs increased after sedation. Mean LA and mean LA/Ao decreased significantly ($P < 0.001$ for both variables). The mean RVIDd and RVIDs were not significantly different after sedation. The CR was medium for all the above variables (Table 2).

Measurements reflecting systolic function were altered by sedation, with the exception of mean LVIDs; the mean value for FS ($P < 0.001$) and EF ($P < 0.001$) decreased significantly. However, examination of individual responses to sedation showed that EF increased in three cats and decreased in seven cats; similarly, FS increased in two cats and decreased in eight cats. Finally, mean PEP and ET increased in sedated cats ($P < 0.001$ for both measurements). The CR was low for EF and medium for all the other variables.

Spectral Doppler interrogation revealed that Vmax. RVOT decreased after sedation ($P < 0.001$), contrary to mean Vmax.LVOT that remained unchanged. The Vmax. LVOT increased in five cats and decreased in the five other cats. The amplitude of variation was particularly important for two cats: one had an increase from 0.58 ms to 0.79 ms and one had a decrease from 0.98 ms to 0.73 ms. The Vmax.RVOT decreased in all cats after sedation but the amplitude of variation was variable. As a result, CR was medium for both variables.

All individual measurements of diastolic function decreased after injection of butorphanol and alfaxalone, with the exception of IVRT, which increased ($P < 0.001$). Thus, E, A, Ea Lat, Aa Lat, Ea Sept and Aa Sept decreased ($P < 0.001$ for all variables). Nevertheless, mean E/A, E/Ea Sept, E/Ea Lat, Ea Sept/Aa Sept and Ea Lat/Aa Lat ratios remained unchanged in sedated cats. CR was medium for these variables.

Discussion

IM injections of alfaxalone and butorphanol resulted in small but statistically significant variations of several ECG measurements. However, not all ECG measurements have the same clinical importance for the diagnosis of cardiac diseases in cats, and observed statistically significant changes may not translate into a clinically relevant effect.

In this study, left ventricular dimensions (IVSd, IVSs, LVFWd, LVFWs, LVIDd and LVIDs) did not significantly change after sedation. This is in contrast with most other sedation protocols using ketamine, acepromazine, butorphanol, midazolam or dexmedetomidine, which significantly alter at least one of those measurements.[9,15,16]

Mean LA/Ao significantly decreased compared with baseline. Although it stayed below the previously published intraday variation for this variable,[17] the difference between pre- and post-sedation values was slightly higher than those reported with other sedation protocols using drugs such as ketamine, acepromazine, butorphanol, midazolam and dexmedetomidine.[9,15,16]

Two markers of left ventricular systolic function (FS and EF) significantly decreased after sedation with butorphanol and alfaxalone, which suggests a mild depressant effect, or that the effect of stress from manual restraint on these measurements was abolished by sedation. It was also reflected by the increase in PEP and ET. The reduction in FS and EF was considered clinically relevant. It was also more pronounced than with other sedation protocols that used butorphanol, midazolam, acepromazine or ketamine, but a more dramatic reduction in systolic function has been reported with protocols combining dexmedetomidine with other drugs.[9,15,16] However, it is unclear if the reduction in systolic function could complicate the accurate diagnosis of left or right dynamic ventricular outflow tract obstruction in cats.[18,19] Alfaxalone alone injected intravenously did not modify cardiac output at a dose $\leqslant$5 mg/kg.[4] The absence of a significant effect of the sedation on LVIDd, LVIDs and HR suggests that sedation did not alter cardiac output despite the different route of injection used in this study or the combination with butorphanol.

Diastolic dysfunction is a hallmark of feline cardiomyopathies; therefore, ECG assessment of diastolic function is essential in this species. Effects of sedation on diastolic function in healthy cats have been evaluated in only one other study, which used sedation with acepromazine/butorphanol and acepromazine/butorphanol/ketamine.[15] Although the amplitudes of the differences for E, A, Ea, Aa and IVRT were higher with the protocol used in our study, the E/A, E/Ea and E/Ea ratios were not affected by sedation.

The reduction in HR, measured 10 mins after the beginning of the ECG examinations, approached statistical significance ($P = 0.002$). The effect of a combination of alfaxalone and butorphanol on HR is difficult to compare with the action of other sedation protocols because the time when this variable is recorded after sedation has not been standardised or detailed by investigators in previous publications. This possible reduction in HR could have

Table 2 Echocardiographic variables before and after sedation

Variable	Baseline		After sedation			Total σ†
	Baseline values	SD	Mean difference with sedation	*P* value	Coefficient of reliability	
IVSd (cm)	0.40	0.03	0.00	0.780	0.42	0.09
IVSs (cm)	0.64	0.09	−0.04	0.013	0.50	0.13
LVIDd (cm)	1.45	0.14	−0.06	0.018	0.56	0.23
LVIDs (cm)	0.89	0.12	0.05	0.046	0.54	0.19
LVFWd (cm)	0.39	0.05	0.00	0.860	0.46	0.10
LVFWs (cm)	0.61	0.07	0.00	0.680	0.49	0.13
RVIDd (cm)	0.35	0.12	−0.02	0.250	0.52	0.16
RVIDs (cm)	0.21	0.09	0.01	0.230	0.60	0.12
FS (%)*	38.95	6.04	−6.12	<0.001	0.45	10.10
EF (%)*	57.39	8.27	−9.32	<0.001	0.35	14.14
Ao (cm)	0.74	0.04	0.01	0.17	0.52	0.09
LA (cm)*	1.11	0.08	−0.05	<0.001	0.64	0.15
LA/Ao*	1.50	0.12	−0.09	<0.001	0.50	0.24
Vmax.RVOT (m/s)*	0.50	0.10	−0.12	<0.001	0.60	0.12
Vmax.LVOT (m/s)	0.64	0.14	−0.03	0.180	0.54	0.19
IVRT (ms)*	50.17	5.62	7.27	<0.001	0.50	11.30
Ea Lat (m/s)*	0.09	0.02	−0.01	<0.001	0.54	0.03
Aa Lat (m/s)*	0.05	0.01	−0.01	<0.001	0.60	0.02
Ea/Aa Lat	1.77	0.58	0.14	0.120	0.58	0.81
Ea Sept (m/s)*	0.07	0.01	−0.013	<0.001	0.47	0.02
Aa Sept (m/s)*	0.05	0.01	−0.010	<0.001	0.55	0.02
Ea/Aa Sept	1.31	0.22	0.018	0.660	0.56	0.38
E (ms)*	0.65	0.12	−0.12	<0.001	0.67	0.17
A (ms)*	0.45	0.11	−0.11	<0.001	0.43	0.14
E/A	1.48	0.28	0.08	0.150	0.45	0.38
E/Ea Lat	7.31	0.97	−0.29	0.250	0.44	1.78
E/Ea Sept	9.20	1.02	0.11	0.720	0.43	2.17
PEP (ms)*	47.20	9.88	12.47	<0.001	0.64	12.42
ET (ms)*	165.93	16.09	11.17	<0.001	0.61	25.31
HR (bpm)	159.9	24.66	−33	0.002	0.38	28.88

*Difference between baseline and after sedation was significant ($P < 0.0017$)
†Total σ: total SD for each variable
IVSd = interventricular septum thickness in diastole; IVSs = interventricular septum thickness in systole; LVIDd = left ventricular internal dimension in diastole; LVIDs = left ventricular internal dimension in systole; LVFWd = left ventricular free-wall thickness in diastole; LVFWs = left ventricular free-wall thickness in systole; RVIDd = right ventricular internal dimension in diastole; RVIDs = right ventricular internal dimension in systole; FS = fractional shortening; EF = ejection fraction; Ao = aortic diameter; LA = left atrium diameter; Vmax.RVOT = maximal velocity of blood flow in the right ventricular outflow tract; Vmax.LVOT = maximal velocity of blood flow in the left ventricular outflow tract; IVRT = isovolumic relaxation time; Ea = peak early diastolic mitral annulus velocity; Aa = peak late diastolic mitral annulus velocity; Sept = Doppler sample placed at the septal part of the mitral annulus; Lat = Doppler sample placed at the lateral part of the mitral annulus; E = peak velocity of early diastolic transmitral flow; A = peak velocity of late diastolic transmitral flow; PEP = pre-ejection period; ET = ejection time; HR = heart rate

been caused by butorphanol, a synthetic opioid analgesic that stimulates parasympathetic tone, because alfaxalone, when used alone at a dose of 5 mg/kg, does not modify HR.[4,20] The HR might also decrease because of reduced stress with sedation. A reduction in HR may facilitate acquisition of higher-quality images and cineloops, and may decrease the risk of myocardial ischaemia in animals with concentric left ventricular hypertrophy.[21]

The CR was medium for most ECG variables, and reflected both the degree of intraoperator and intraobserver variability and the interindividual response of the cats to sedation. This interindividual variability was especially pronounced for HR and EF. All but one cat showed a decrease in HR, but the amplitude of HR change varied between animals. The one cat that had an increase in HR was one animal that did not reach the maximum level of

sedation on the posture scale. In addition, because HR was only compared at two different time points, it is possible that fluctuations of HR over time were responsible for the interindividual variability for this variable. Comparison of HR averaged over longer periods of time could have provided a more accurate assessment of the effects of the sedation protocol on this measurement. Baseline and post-sedation EFs of individual cats were spread over a wider range of values than FS, although both measurements evaluate systolic function from left ventricular chamber dimensions. This result likely reflects inaccuracies in measuring left ventricular volumes using the Simpson's method and explains, in part, the low CR for this variable. The repeatability of EF has not been intensively studied in cats, but the coefficient of variation using the same method was reported to be 3.0% in one study.[15]

Besides having a minimal effect on cardiovascular function and most ECG measurements, IM sedation with a combination of alfaxalone and butorphanol did not share some of the side effects of other sedation protocols for ECG in cats. Butorphanol is often used in association with other agents to sedate cats because of its wide safety margin and sedative effects.[9,15] Dysphoric or aggressive behaviour, a common paradoxical effect of midazolam,[9,22] was not observed with the protocol used in this study. Although excitement and hyper-reactivity during recovery is common with ketamine and has been reported after IM administration of 10 mg/kg of alfaxalone in cats,[5] the low dose used in this study and the combination with butorphanol might have prevented the occurrence of this adverse effect.[2,23] Ketamine also increases HR and myocardial oxygen consumption,[16,24] which are detrimental to cats with hypertrophic cardiomyopathy. Conversely, the current protocol decreased HR in 9/10 cats, although the mean difference for the whole group did not reach statistical significance. Finally, although apnoea has been reported after IV injection of a high dose of alfaxalone,[4] none of the cats in the study experienced cardiorespiratory depression during sedation.

The duration of sedation following IM injection of alfaxalone and butorphanol was sufficient to perform a complete ECG examination, and the rapid recovery allowed the animals to be discharged shortly after the procedure. These characteristics are essential for clinical use and represent a significant advantage over long lasting drugs. For example, sedation with acepromazine, a relatively safe and strong sedative, can last up to 6 h in cats.[25] Finally, alfaxalone does not induce vomiting, which is one the most common side effects of alpha-2 agonists,[9,26] especially after IM injection.

For the purpose of this study alfaxalone and butorphanol were administered in two separate injections because no study has been carried out to determine if pharmaceutical interactions may occur when mixed in a single syringe. However, we typically mix alfaxalone and butorphanol in the same syringe in clinical practice and have not observed adverse effects or a less pronounced sedation.

The goal of sedation during ECG is to decrease stress, improve image quality and restrain fractious animals. Thus, a score of 3 on the posture scale (lateral recumbency) was required to start the ECGs. Although IM injections of alfaxalone and dexmedetomidine generate a loss of withdrawal reflex after a mean time of 7.2 ± 2.5 mins,[2] no data have been published regarding the time of maximum sedative effect of alfaxalone and butorphanol when injected intramuscularly. The route of administration influences the pharmacokinetics of alfaxalone; the maximum effect is expected in less than 1 min after IV injection and between 30 and 45 mins after subcutaneous injection.[3,4,6,27] This study showed that most of the cats included in this study reached a score of 3 within 10 mins of IM injection.

There are several limitations to this study. First, the operator (IB), who performed all ECGs, was not blinded to the treatment administered because only one protocol was investigated and the order of treatment was not randomised. However, all measurements were carried out by another blinded observer (TR) in order to reduce the motivation bias. Second, the post-sedation ECG was performed 1 week after baseline evaluation. Although all cats remained apparently healthy, we cannot exclude hydration or haemodynamic changes that might have influenced some of the differences observed after the sedation.[28,29] Third, the effects of butorphanol as a single agent on ECG measurements have not been evaluated; thus, the effects observed in this study cannot be solely attributed to the action of alfaxalone. Finally, our study was carried out on healthy cats. The effects of the combination with butorphanol and alfaxalone may be different in cats with haemodynamic perturbations or metabolic disorders, especially because alfaxalone has nonlinear pharmacokinetics.[27] These particular pharmacokinetics also mean that the effects of alfaxalone are not predictable if used at different dosages.

Conclusions

Sedation induced by IM injections of alfaxalone at 2 mg/kg and butorphanol at 0.2 mg/kg is deep and of short duration. Its effects on ECG measurements are not clinically relevant in healthy cats except for two markers of systolic function. These characteristics make this protocol useful when sedation is required to perform ECG in cats. However, further studies are needed to evaluate its safety and effects on ECG variables in cats with preclinical and clinical cardiac disease.

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