
Anesthesia in cats with HCM
Manuel Martin-Flores MV, DACVAA
Section of Anesthesiology
Cornell University Hospital for Animals

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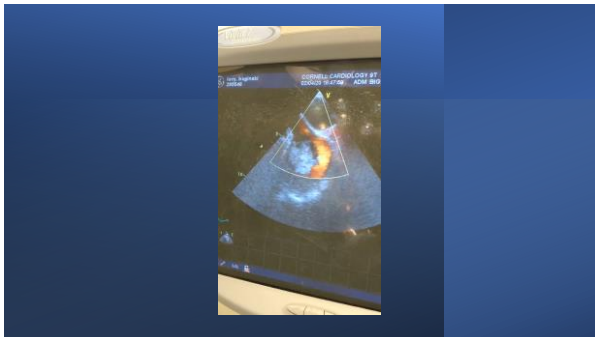

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In other words
What HCM does to a heart
What anesthetics do to a heart (and to a heart with HCM)
How do I prepare for this case

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What (little) I know about HCM

Feline Hypertrophic Cardiomyopathy: An Update

Jonathan A. Abbott, DVM Vet Clin NA 2010; 40

Genetic basis
Frequent finding in cats – **Most common** myocardial dz diagnosed in cats
Mean age at diagnosis is **6 years**
Many asymptomatic at presentation (30%-50%)

Heterogeneous disease: have some long survival, some develop CHF or thromboembolism, some die suddenly
Some but not all cats with HCM have a **murmur** (1/3 in one study)
Many will develop **LVOTO** (**SAM**)
Few cases of **LVOTO** without hypertrophy

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Pathophysiology of HCM

Feline Hypertrophic Cardiomyopathy: An Update

Jonathan A. Abbott, DVM Vet Clin NA 2010; 40

Generally accepted as **diastolic dysfunction**

Delayed ventricular **relaxation** and **diminished compliance**
Higher filling pressures at low to normal volumes
Pulmonary **congestion** or edema
Atrial enlargement

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Pathophysiology of HCM

Feline Hypertrophic Cardiomyopathy: An Update

Jonathan A. Abbott, DVM Vet Clin NA 2010; 40

Global **systolic** function is commonly **normal** or **hyperdynamic**

LVOTO (or SAM) is a **common, complex, associated** phenomenon
Results from systolic motion of valve leaflets towards septum

It is **dynamic**, as opposed to anatomical (**fixed**) obstructions (stenosis)
This is important, as the **magnitude** of LVOTO may be **manipulated**

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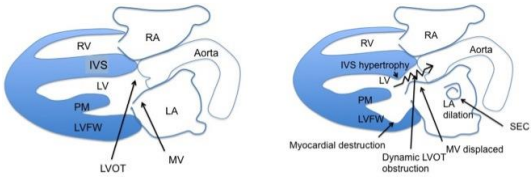

Normal HCM

ACVIM.org

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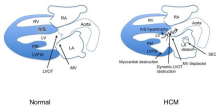
LVOTO



Normal HCM

Displaced valve **leaflets** cause **mechanical obstruction**
The cause is probably multifactorial

Venturi effect initially proposed
Drag forces proposed more recently
Abnormal geometry + chordal laxity

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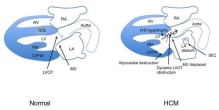
LVOTO



Normal HCM

SAM impedes LV outflow and **increases wall tension**

Myocardial perfusion abnormalities have been described with SAM

Narrow coronary arteries

Mitral valve regurgitation also observed

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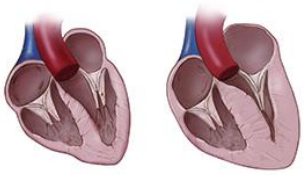
HCM summary

Poor relaxation (lower filling volume and higher filling pressures)
Reduced ventricular preload

Systolic function **normal** or **hyperdynamic**

SAM

Others
Arrhythmias
Thrombi
Possibly **reduced perfusion**



Cornell Feline Health Center

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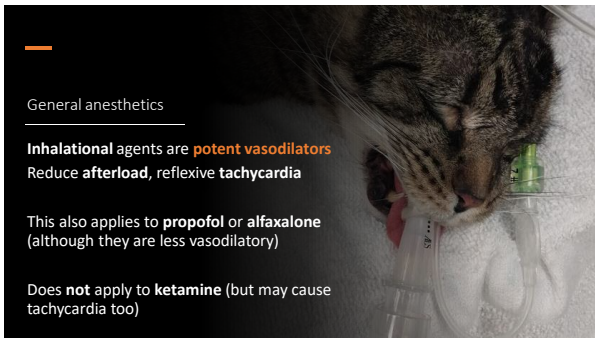

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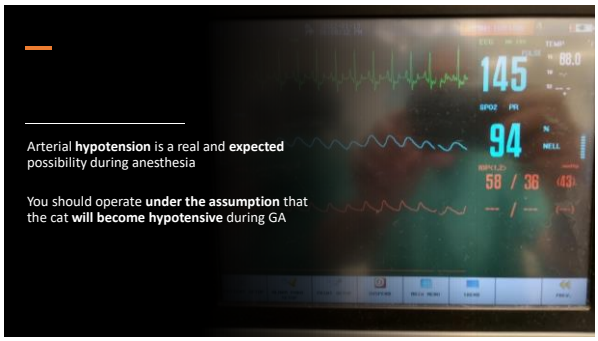

Arterial **hypotension** is a real and **expected** possibility during anesthesia

You should operate **under the assumption** that the cat **will become hypotensive** during GA

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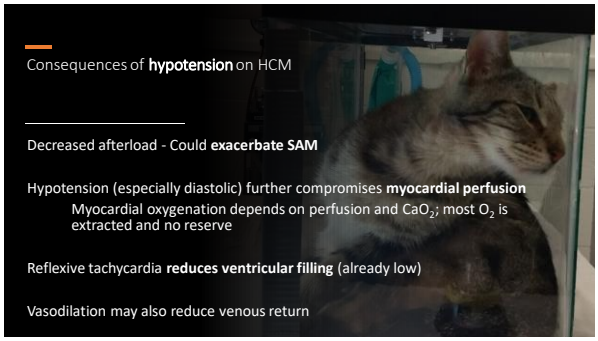

Consequences of **hypotension** on HCM

Decreased afterload - Could **exacerbate SAM**

Hypotension (especially diastolic) further compromises **myocardial perfusion**

- Myocardial oxygenation depends on perfusion and CaO_2; most O_2 is extracted and no reserve

Reflexive tachycardia **reduces ventricular filling** (already low)

Vasodilation may also reduce venous return

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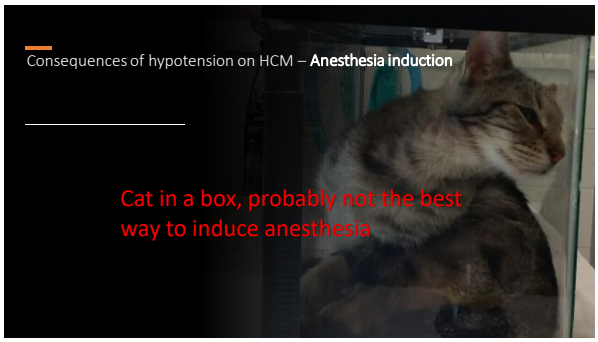

Consequences of hypotension on HCM – **Anesthesia induction**

Cat in a box, probably not the best way to induce anesthesia

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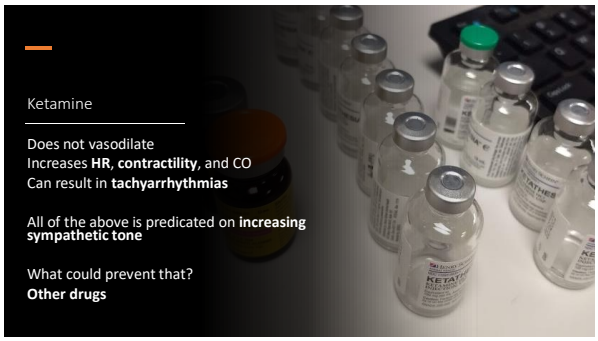

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Treatment of hypotension during GA

I keep fluid therapy to minimum (2-5 mL/k/hour).
Fluid therapy is typically **ineffective** for treating hypotension

Hypotension may come from different sources:
Low afterload - very common with GA

Tachycardia – low filling volume

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Dexmedetomidine

Increases afterload (may reduce SAM)
Increases blood pressure (increase in diastolic may help with myocardial perfusion)

Decreases HR (better filling, less tachyarrhythmias)

It's reversible. And analgesic. And not controlled

Sounds pretty good

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SMALL ANIMALS

Doppler echocardiographic effects of medetomidine on dynamic left ventricular outflow tract obstruction in cats

Leigh A. Lamont, DVM, MS; Barret J. Bulmer, DVM; David D. Sisson, DVM, DACVIM; Kurt A. Grimm, DVM, MS, DACVA, DACVCP; William J. Tranquilli, DVM, MS, DACVA

Objective—To evaluate the effects of medetomidine on dynamic left ventricular outflow tract (LVOT) obstruction in cats with left ventricular hypertrophy.

Design—Clinical trial.

Animals—6 domestic shorthair cats with echocardiographic evidence of dynamic LVOT obstruction.

Procedure—Cats were restrained in lateral recumbency, and baseline M-mode and Doppler echocardiographic examinations were performed. An ECG was recorded continuously, and blood pressure was measured indirectly with Doppler instrumentation. Medetomidine (20 µg/kg [9.1 µg/lb]) was then administered IM, and examinations were repeated 15 minutes later.

Results—Significant decreases in heart rate, LVOT velocity, and the LVOT pressure gradient were documented following medetomidine administration. After adjusting for the effects of heart rate by ANCOVA, there were no significant differences in any other systolic or diastolic indices of left ventricular function.

Conclusions and Clinical Relevance—Results suggest that administration of medetomidine to cats with dynamic LVOT obstruction may result in elimination of outflow tract obstruction; medetomidine may be a suitable sedative and analgesic agent in this subpopulation of cats. (J Am Vet Med Assoc 2002;221:1276–1281)

diographically that systolic anterior motion is initiated by flow drag forces acting on the protruding mitral valve leaflet and facilitated by the large angle created with hypertrophy of the ventricle between the leaflet and the jet of left ventricular inflow.[4] Systolic anterior motion of the mitral valve has also been observed in cats with hypertrophic cardiomyopathy and LVOT obstruction[5] and probably operates through the same mechanism.

Left ventricular outflow tract obstruction increases systolic left ventricular pressure, systolic wall tension, and myocardial work.[1] In addition, coronary perfusion pressure is decreased as aortic diastolic pressure decreases and left ventricular diastolic pressure increases.[4,6] In human patients, these hemodynamic abnormalities may lead to sinus tachycardia associated with hypotension, bradyarrhythmias, ventricular tachyarrhythmias, and myocardial ischemia, which can ultimately cause sudden death.[6] Similarly, sudden cardiac death in cats with idiopathic hypertrophic cardiomyopathy has been reported[7] and is presumably a result of similar types of arrhythmias or ischemia secondary to impaired coronary perfusion.

Many cats with LVOT obstruction do not have any clinically apparent abnormalities and are examined by a veterinarian for a variety of other reasons. In these instances, diagnosis of the underlying cardiac disease may be incidental. Consequently, it is not uncommon for cats with LVOT obstruction to be sedated or anesthetized without prior diagnosis of their condition. However, even if the condition is identified, this par-

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Dexmedetomidine is not forbidden (in fact, we use it often)

What could go wrong?

Bradyarrhythmias (sinus b, AV block, AIVR)

Reversal (likely safe if only sedation, different if under GA)

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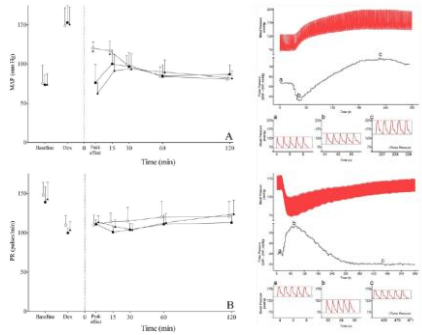
Reversal isn't magic



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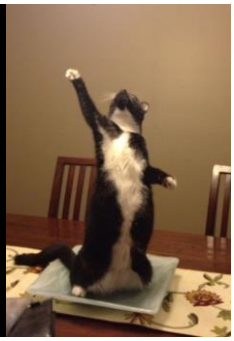
To treat or not treat (*bradyarrhytmyas*)?

I do not if BP is acceptable – risk of tachycardia and hypertension

I have **developed** a **tolerance** to low HR and benign blocks – this **took time**

Low HR, blocks, and AIVR that affect BP are treated by **increasing the HR**:

- reduce anesthetics
- reduce opioids
- atropine



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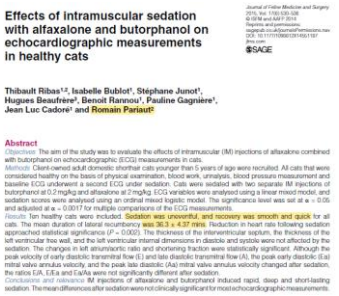
Sedation

Dexmedetomidine + opioid

Dexmedetomidine + alfaxalone (meaner cats)

Alfaxalone + opioid

I avoid ketamine but know a guy (who knows a guy) who uses it for HCM cats



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

Journal of Feline Medicine and Surgery
SAGE

Thibault Ribas[1,2], Isabelle Bublot[1], Stéphane Junot[1], Hugues Beaufrère[3], Benoit Rannou[1], Pauline Gagnière[1], Jean Luc Cadoré[1] and Romain Pariaut[2]

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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Planning GA
Exam, history, and echo (I walk to cardio to discuss)
If cat is **receiving medication** (typically a beta-blocker), I **do not discontinue** it (except ACEi)
Discuss increased **risks** of negative events with **owner**
I **always preoxygenate**
Materials, drugs, and emergency drugs **ready**
Machine **checked**
(all of that before I give a sedative)

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Summary of anesthetic management
I try to
Avoid excessive anesthetic doses (inhalational and propofol)
Avoid tachycardia
Avoid substantial drops in afterload (vasodilation)
I favor vasopressors and low heart rates

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I follow GA from the **sedation** we already discussed
And no matter what the cardiac problem is, the **basics of anesthesia** are always applied
Airway. Monitoring. Equipment. Contingency plans
Don't get **tunnel vision** and forget the rest

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Typical anesthetic plan (for me)

Sedation: opioid + dexmedetomidine, o alfaxolone

Induction: propofol, or alfaxolone

Maintenance: inhalant + opioid or dexmedetomidine infusion (if long sx expected)

Local anesthesia: yes, except epidural anesthesia

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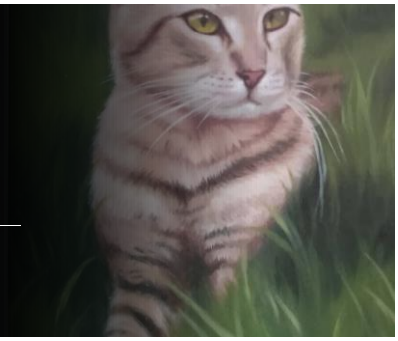
Thank you



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