Blood Pressure Monitoring And Management

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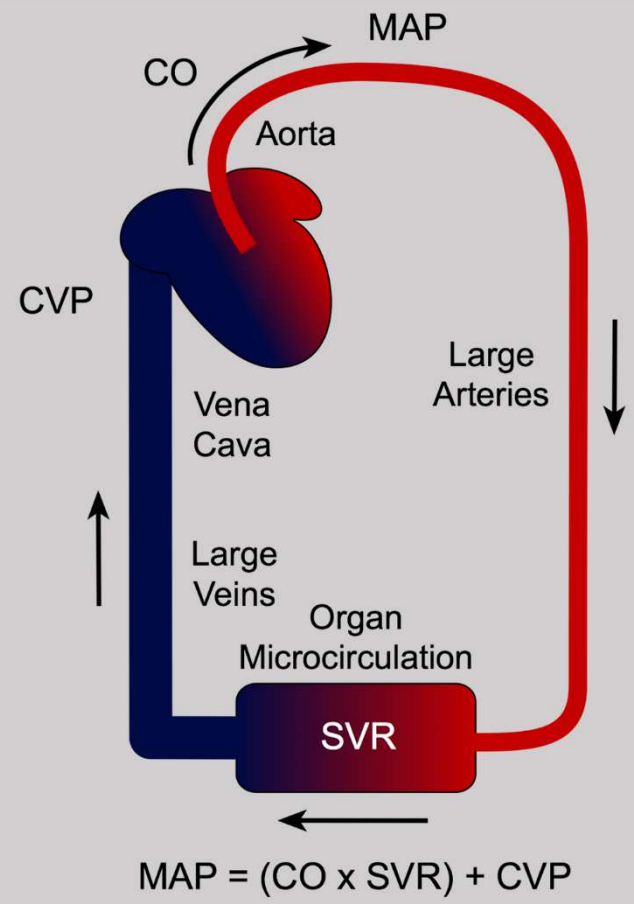
Quick Review

Our goal is adequate oxygen delivery to tissues/organs while under general anesthesia

This is dependent upon:

- Cardiac output (CO; L/min)
- Oxygen carrying capacity (CaO2)
  - Cassidy's lecture



Cardiac output

- **Directly related to organ perfusion**
- **Takes into consideration: heart rate, rhythm, contractility, preload (volume status), afterload, etc.**

- **But:**

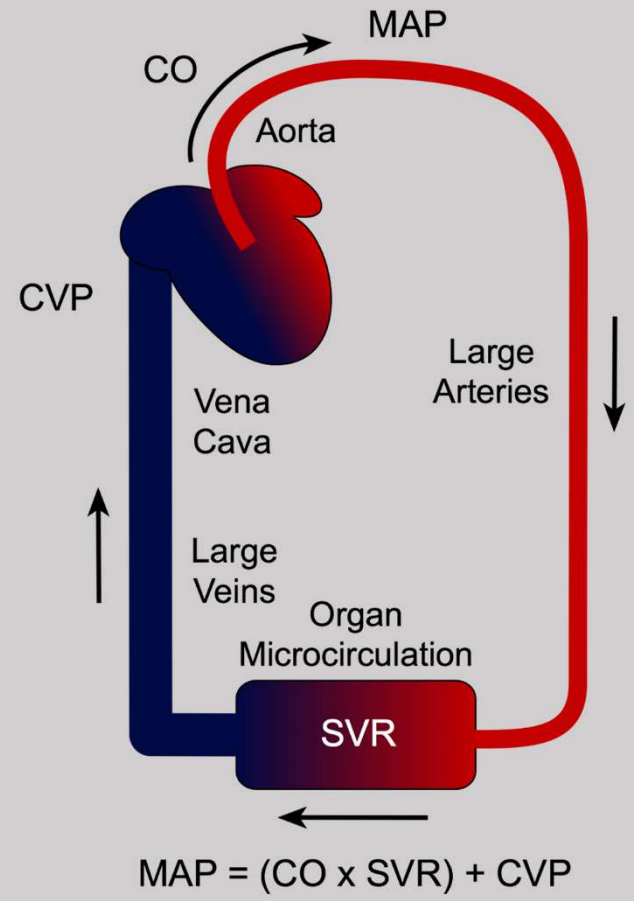
1. Involved
2. Expensive
3. Special training
4. Global perfusion parameter, does not specifically indicate individual organ perfusion

Quick Review

Our goal is adequate oxygen delivery to tissues/organs while under general anesthesia

This is dependent upon:

- Cardiac output (CO; L/min)
  - Vascular resistance (VR)
  - Blood pressure (BP)
- Oxygen carrying capacity (CaO2)



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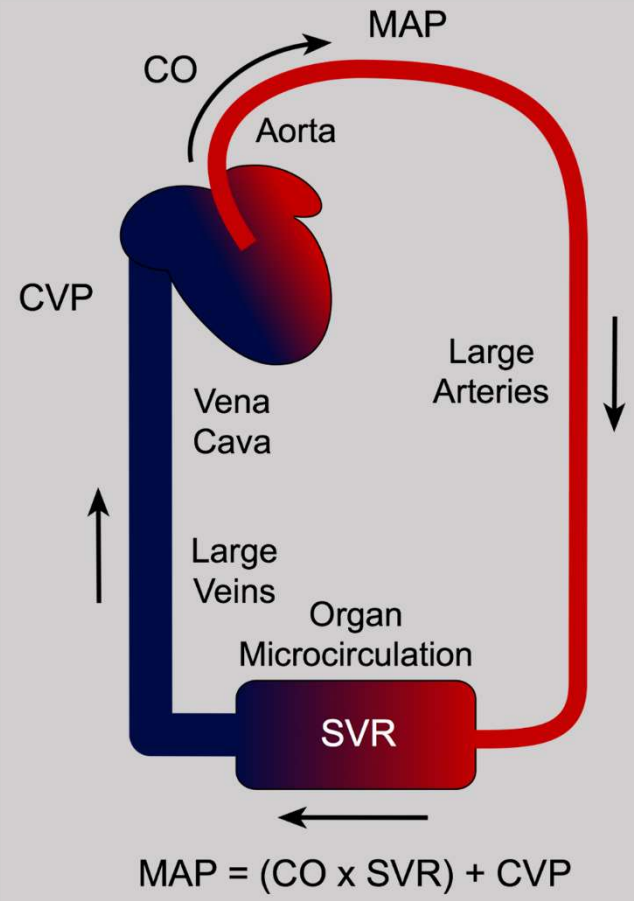
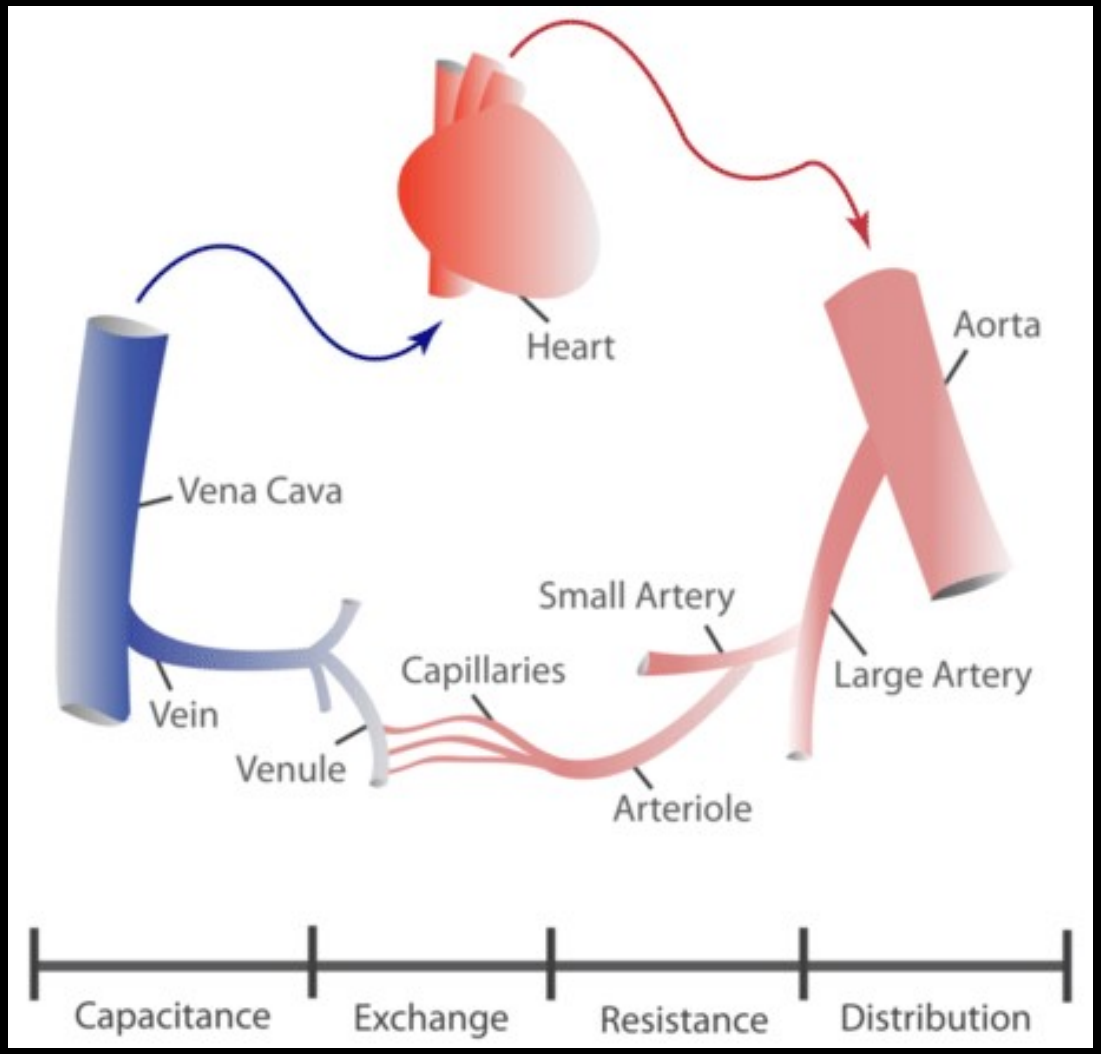
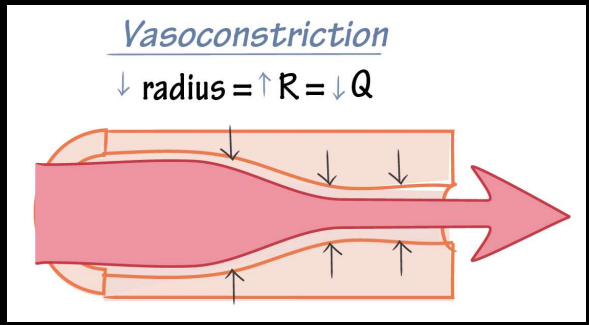


Blood pressure is a result of numerous variables,
…but:

- Proportional to CO (↑CO = ↑BP)
- Proportional to VR (↑VR = ↑BP)

Blood pressure is also easy to measure!





Regional vascular resistance determines organ perfusion

=

Vasoconstrict will improve BP; however, can impair perfusion in some circumstances

Target underlying derangements

Putting the physiology together

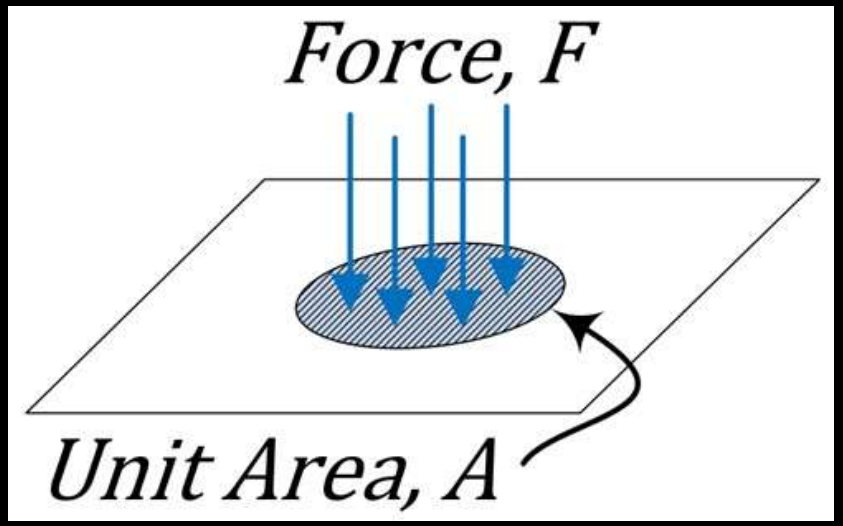
1. Inadequate oxygen delivery/perfusion to tissues will result in end-organ ischemia/ damage

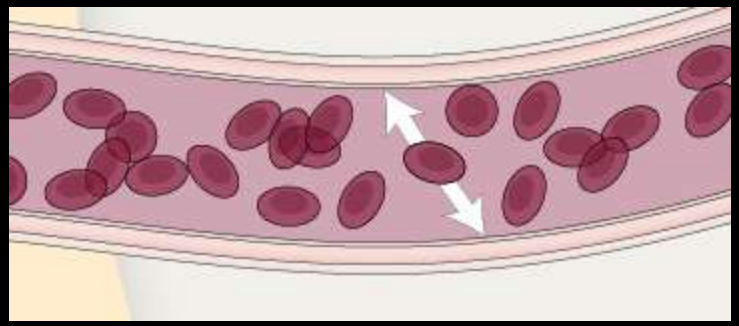
2. Organ perfusion is:
   - Proportional to CO
   - Indirectly proportional to *regional* VR

3. Normal BP may not produce adequate organ perfusion, depending on regional VR

4. Blood pressure is:
   - Proportional to both CO and VR
   - The only global parameter we can easily, cheaply, and accurately measure to assess organ perfusion

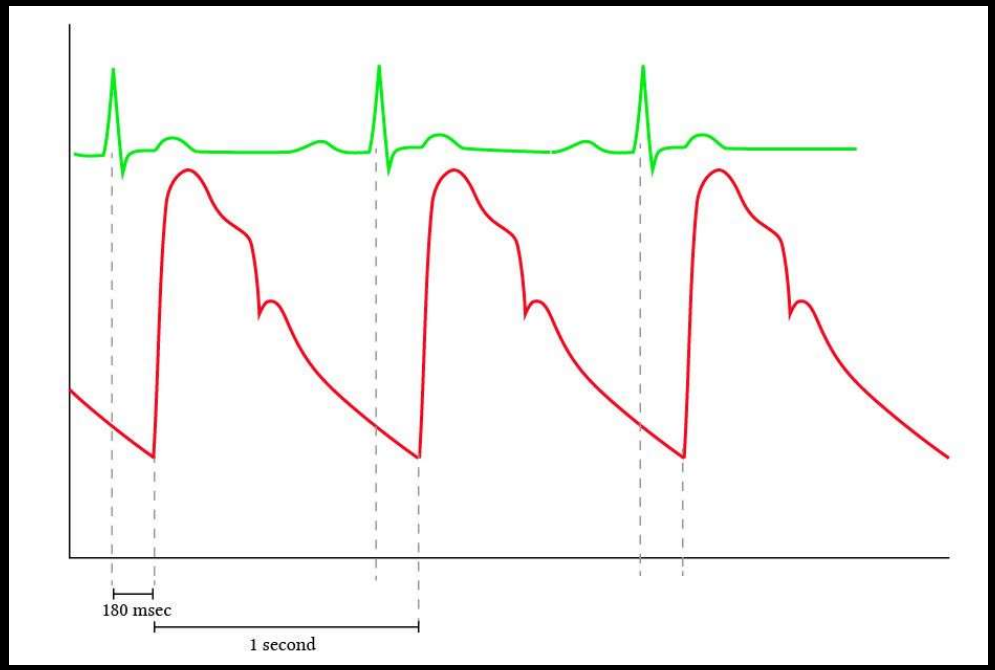
What Actually Is Blood Pressure?





Blood volume ejected into vasculature

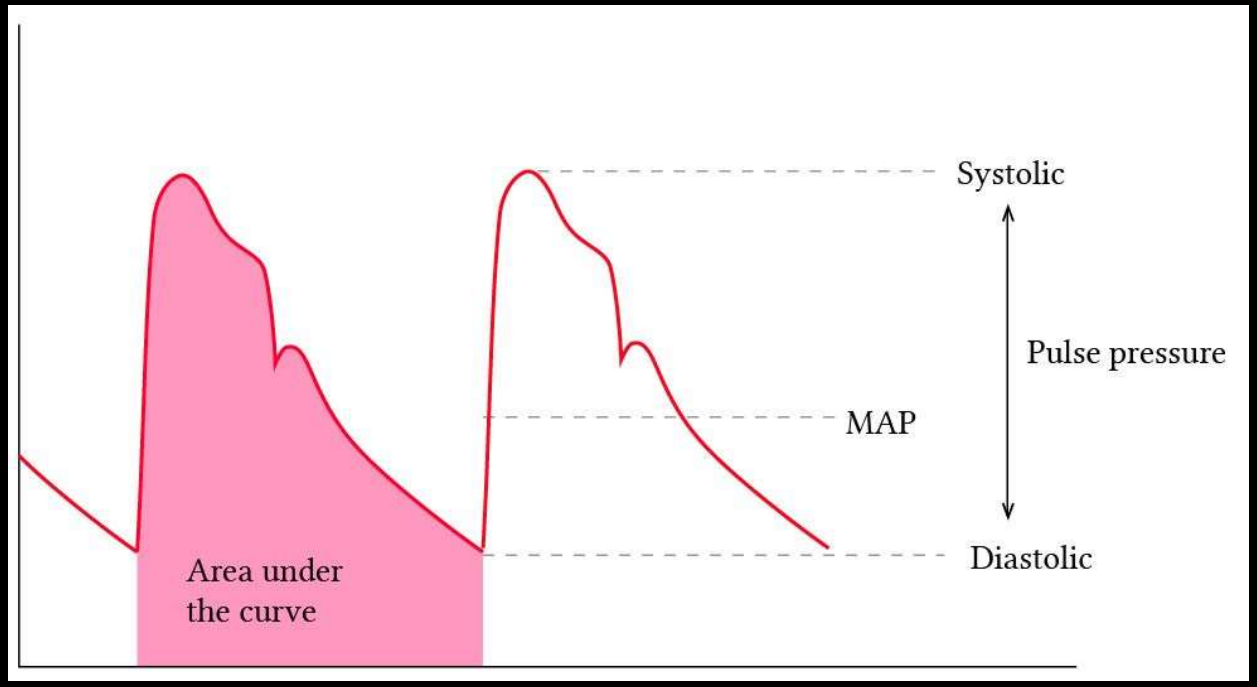
Pressure ↑, then ↓ (repeat!)



Mazzaferro and Wagner 2001; Gaynor et al 2001; Gordan and Wagner 2013; Ruffiato et al. 2015; Monk et al 2005; Son et al 2015

What Actually Is Blood Pressure?

Mean blood pressure is just the time-weighted average



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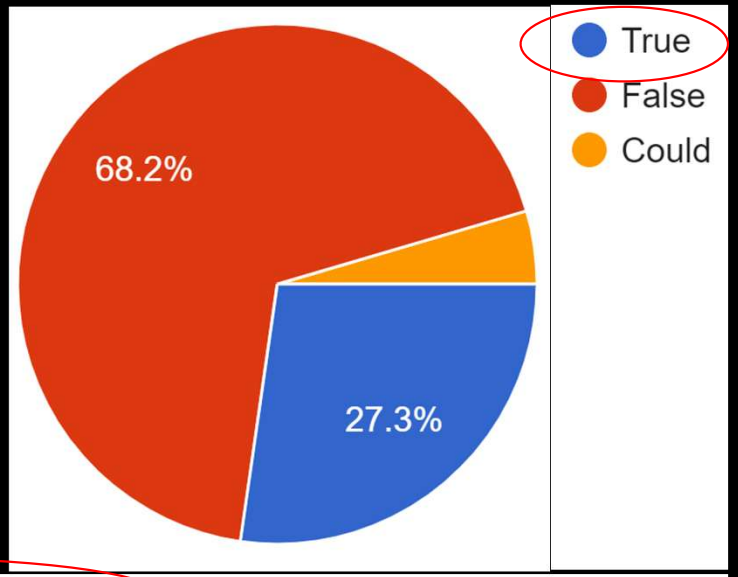
Low Blood Pressure Incidence

- The most common anesthetic-related complication reported, with incidence of:
  - Mixed health population of dogs: 58%
  - Healthy dogs only: 7.5-32%
  - Healthy cats only: 8-30%



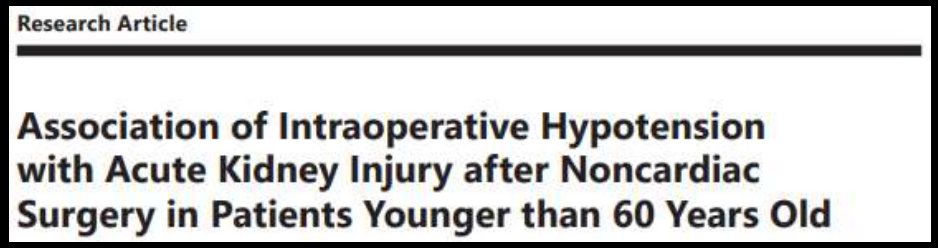
A healthy cat or dog generally will not become hypotensive during general anesthesia at an adequate plane of anesthesia.

Mazzaferro and Wagner 2001; Gaynor et al 2001; Gordan and Wagner 2013; Ruffiato et al. 2015; Monk et al 2005; Son et al 2015

Hypotension as an Adverse Effect

Hypotension associated complications in people:

- Acute kidney injury
- Acute coronary syndrome
- Increased all-cause mortality 1 year post anesthetic event



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Hypotension as an Adverse Effect

Very little outcome data in VetMed to-date

Mortality

Cats that experienced hypotension under GA had 2.6x increased odds of death

AKI in healthy adults

MAP <60mmhg for 11-20min = OR 1.8

MAP <55mmhg for 11-20min = OR 2.3

MAP <55mmhg for > 20min = OR 3.5

Mazzaferro and Wagner 2001; Gaynor et al 2001; Gordan and Wagner 2013; Ruffiato et al. 2015; Monk et al 2005; Son et al 2015

Hypotension is an *expectation* under general anesthesia

Does not mean anyone did anything wrong…it happens

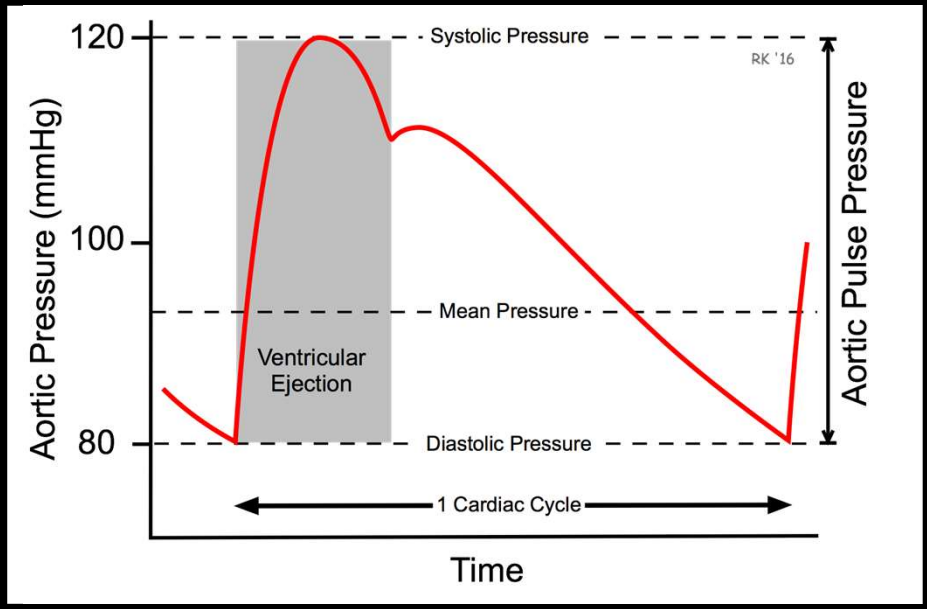
But it does have to be managed

Mazzaferro and Wagner 2001; Gaynor et al 2001; Gordan and Wagner 2013; Ruffiato et al. 2015; Monk et al 2005; Son et al 2015

Managing Hypotension

Need to monitor BP so that *when, not if,* hypotension is diagnosed, it can be treated

- Definition:
  - Systolic BP (SBP) < 90 mmHg
  - Mean art. BP (MAP) < 60 mmHg
- MAP is what we care about
  - Average pressure presented to organs
  - Organ-specific minimum perfusion pressure
    - E.g., Brain, kidneys = 50 mmHg
  - SBP - MAP = ~30-40mmhg
    - Where 90mmHg SBP target originates
    - Can vary depending on morbidities



Ruffiato et al. 2015

Monitoring Methods

- Indirect:
  - Non-invasive
    - Doppler flow probe
    - Oscillometric
  - Intermittent monitoring
  - Cheaper, easier to introduce to workflow
  - Modest accuracy/precision when applied correctly; some considerations

Ruffiato et al. 2015

Monitoring Methods - Indirect

Easily influenced from technical error

- Cuff sizing is important!
  - Cuff width needs to be 40% of circumference where placed
  - Why 40%?
    - Ensures cuff balloon makes it all the way around the limb
  - Measure!
- Placement
  - Tight placement, lightly taping around to keep Velcro attached – OK
  - Mid-antebrachium ideal (not over carpus)
  - Base of tail or hind limb OK

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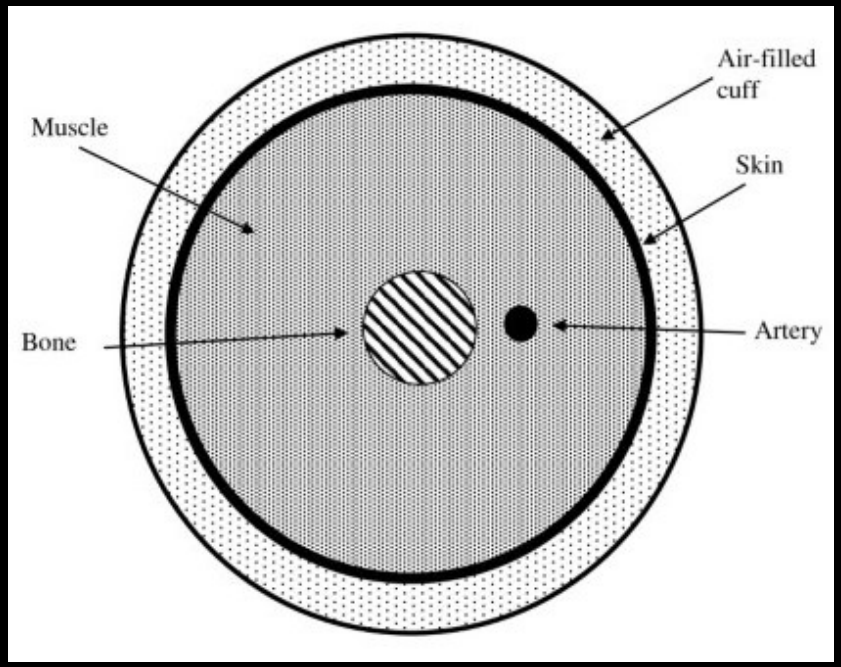
Table 7.2 Technical Factors in Indirect Blood Pressure Measurement: Causes of Disagreement Between Direct and Indirect Blood Pressure Measurement

Cuff problems
Size too small leads to overestimation
Fit in a conical-shaped arm
Extrinsic cuff compression
Limb position relative to heart
Rapid deflation leads to underestimation
Physiologic problems and method limitations
Rapid pressure changes
Dysrhythmias
Severe vasoconstriction and shock
Shivering and patient movement
Beat-to-beat variation (e.g., pulsus alternans)

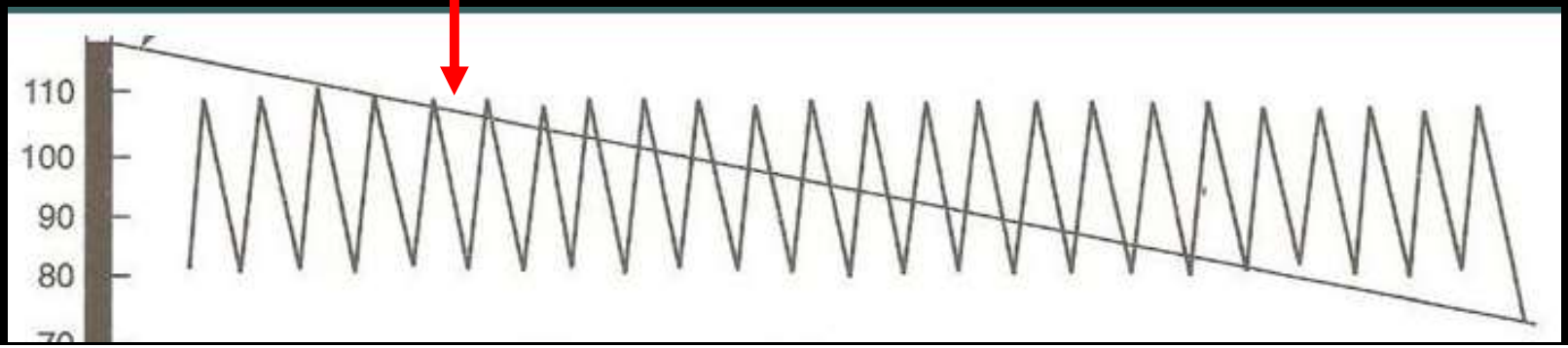
Ruffiato et al. 2015

Monitoring Methods - Indirect

- Occlusion based technology
  - Doppler –
    - Inflate cuff with sphygmomanometer
      - Release cuff pressure 3-5 mmHg/second
      - First sound = Systolic BP
    - Doppler estimates the SBP!

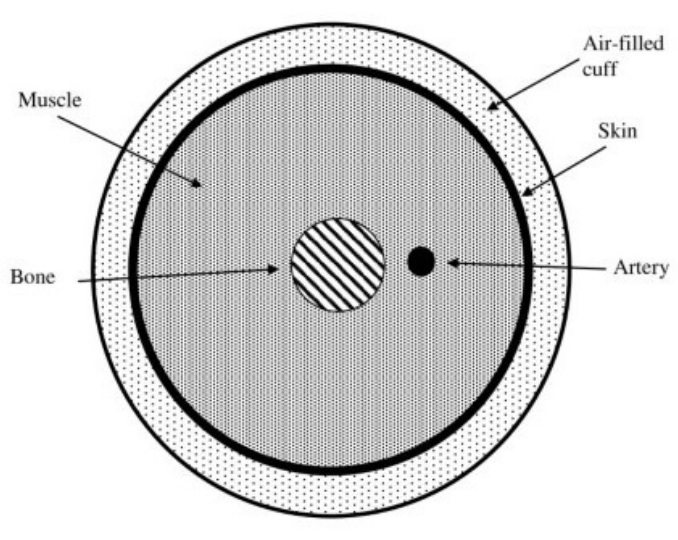


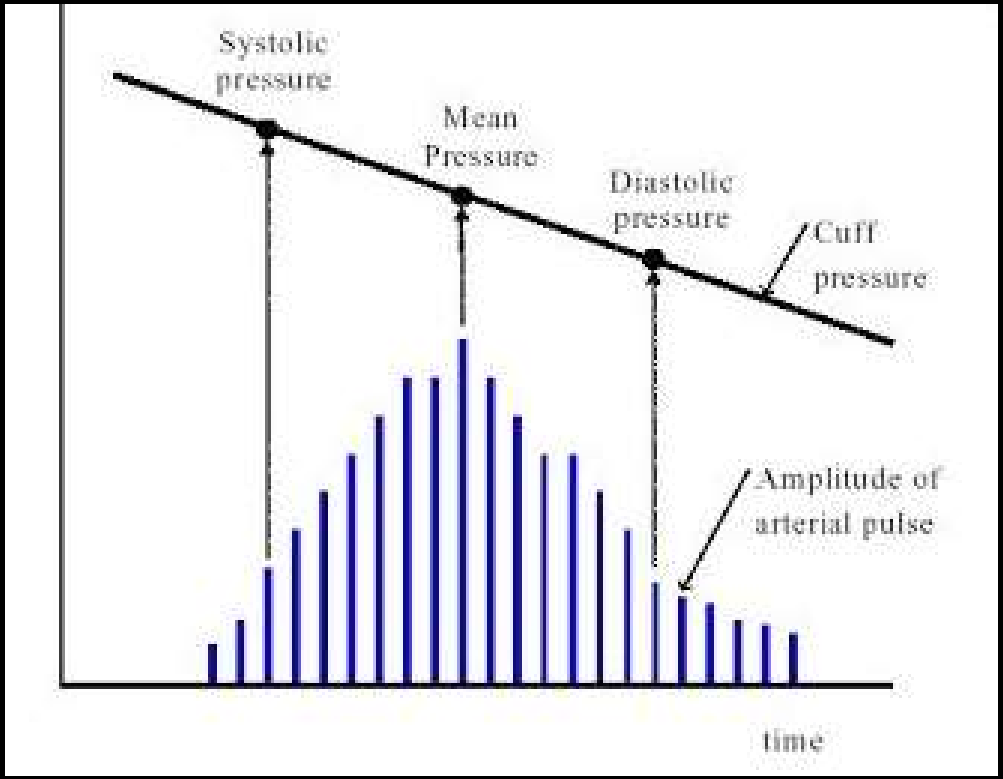
Systolic BP
(First sound heard)



Monitoring Methods - Indirect

- Occlusion based technology
  - Oscillometric –
    - Automated inflation/deflation several times
    - MAP is only measured value
    - Algorithm estimates SBP and DBP





Monitoring Methods

Both indirect technologies overestimate hypotensive pressure

If using these as thresholds to treat:

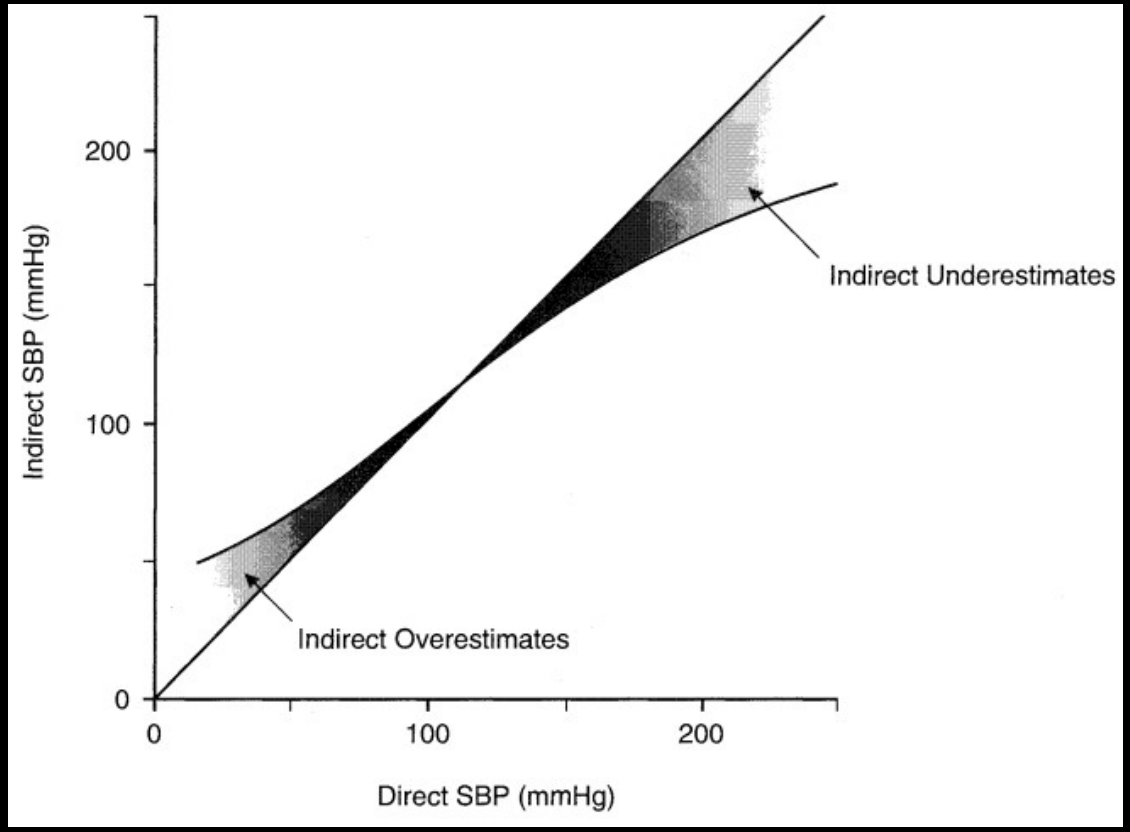
90 mmHg Doppler, SBP

60 mmHg Oscillometric, MAP

Can miss hypotensive patients

RECOMMENDATION:

- Doppler – Treat for hypotension if < 95 mmHg
- Oscillometric – Treat for hypotension if MAP < 65 mmHg

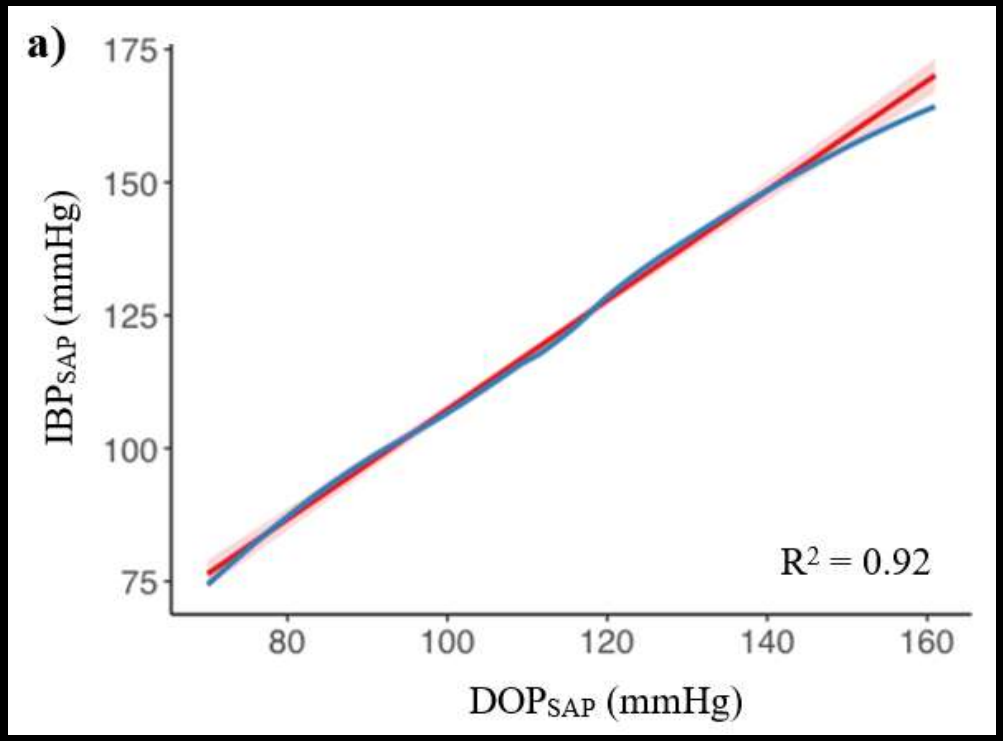


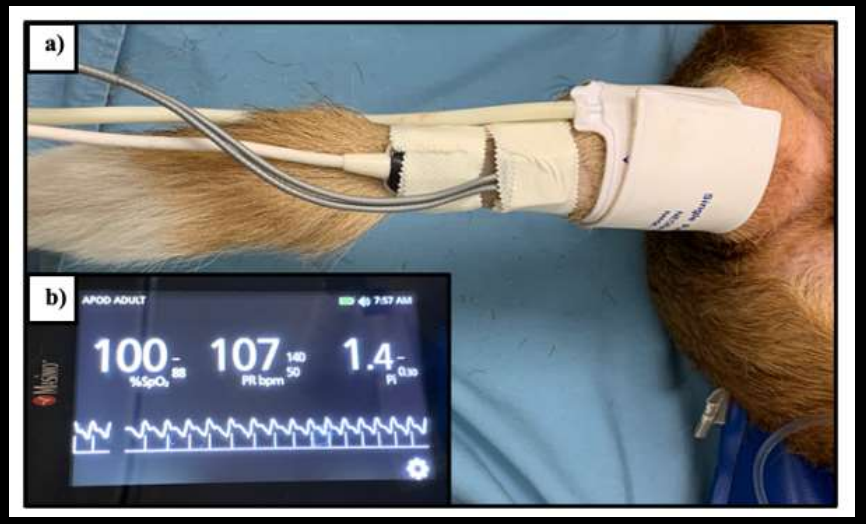
Comparison of doppler ultrasound and pulse oximeter plethysmography measured from the coccygeal artery with invasive blood pressure in anesthetized dogs

John H Whittaker[a], Nathaniel Kapaldo[a], Eduarda M Bortoluzzi[b], David C Rankin[a]

[a]Department of Clinical Sciences, College of Veterinary Medicine, Kansas State University, Manhattan, KS, USA

[b]Department of Anatomy and Physiology, College of Veterinary Medicine, Kansas State University, Manhattan, KS, USA





Ruffiato et al. 2015

Monitoring Methods

- Direct:
  - Artery catheterization
  - Accurate, real-time
  - Ideal for unstable, higher-risk patients
  - Time, money, not practical in healthy patients

Treating The Hypotensive Patient

Remember → BP is dependent on CO and VR
CO is dependent on intravascular volume, HR, contractility, afterload/VR

Goal is to target presumed most contributing factor

- Treatment of underlying cause will be more effective
- Not to just make the number better

Hypotension is going to be caused by:

- Reduced VR
- Reduced CO
- Reduced intravascular volume

Treating The Hypotensive Patient

Remember → BP is dependent on CO and VR
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Hypotension often/may be caused by:

- Reduced VR
  - Direct vasodilation – Isoflurane, sepsis, ACEIs/Amlodipine
  - Indirect vasodilation – reduced sympathetic tone (opioids, inhalants, alpha-2 agonists, trazadone)
- Reduced CO
  - Reduced contractility – Isoflurane, DCM, hypovolemia, sepsis
  - Reduced forward stroke volume – DMVD, HCM
  - Reduced HR – opioids, alpha-2 agonists, reduced sympathetic tone
- Reduced intravascular volume
  - Relative volume deficit – vasodilation*
  - Absolute – vomiting, reduced/no intake, hemorrhage etc.

Treating The Hypotensive Patient

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Choosing An Intervention

Simple Approach To Hypotensive Patients

1) Patient's baseline status (frame perspective)?
(e.g., volume deficit, pediatric, comorbidities?)

2) Assess/optimize patient's depth if too deep; assess for user error in BP measurement
(adjust anesthetic if indicated; re-take BP in 5 min to confirm BP improving)

3) Treat suspected direct cause
(bradycardic = anticholinergic; volume deficit suspected = volume; neither of the latter? Use vasopressor; unsure? = vasopressor rarely wrong; *assess for arrythmias** and treat if present)

4) Allow intervention time to work (~5 min)
(corrected bradycardia &/or still hypotensive = repeat/administer vasopressor; titrate to normotension)

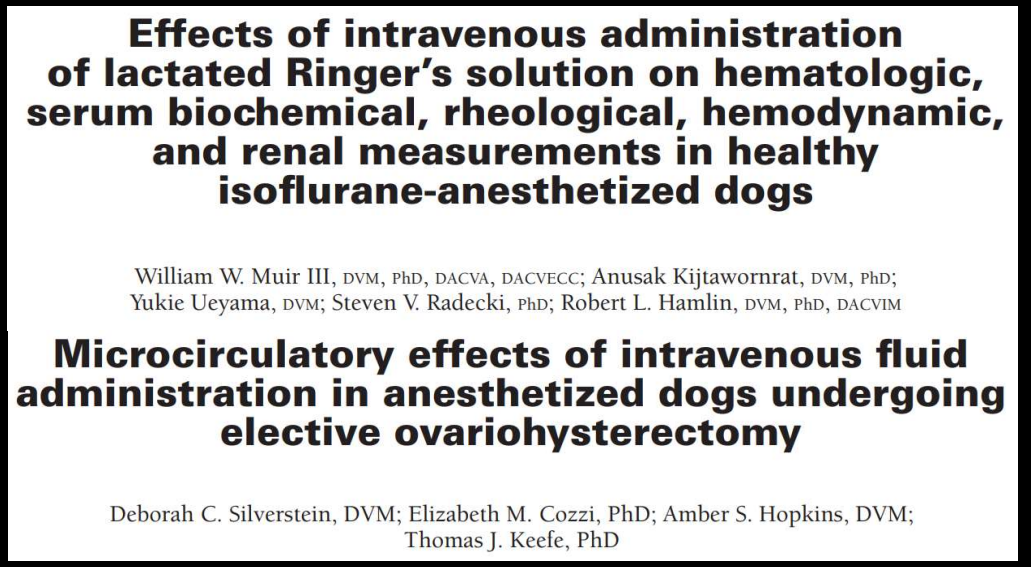
Treatment can target any of main factors because all are influenced by the drugs used.

- Vascular resistance
- Cardiac output
- Intravascular volume

But targeting most likely cause is best!

Fluid Rates & Boluses

- Low level of evidence supporting routine use of 'maintenance' IV fluids, in euvolemic animals
- Maintenance IVF up to 30 ml/kg/hr:
  - Do not prevent inhalant-induced hypotension
  - Do not alter blood pressure throughout anesthetic course
  - Do not increase microcirculatory perfusion (vessels < 20 um)
- Main benefits:
  - Maintain a patent IVC/port for medication administration
  - Address volume deficits, over time



Effects of intravenous administration of lactated Ringer's solution on hematologic, serum biochemical, rheological, hemodynamic, and renal measurements in healthy isoflurane-anesthetized dogs

William W. Muir III, DVM, PhD, DACVA, DACVECC; Anusak Kijtawornrat, DVM, PhD; Yukie Ueyama, DVM; Steven V. Radecki, PhD; Robert L. Hamlin, DVM, PhD, DACVIM

Microcirculatory effects of intravenous fluid administration in anesthetized dogs undergoing elective ovariohysterectomy

Deborah C. Silverstein, DVM; Elizabeth M. Cozzi, PhD; Amber S. Hopkins, DVM; Thomas J. Keefe, PhD

Fluid Rates & Boluses

Maintenance rates?

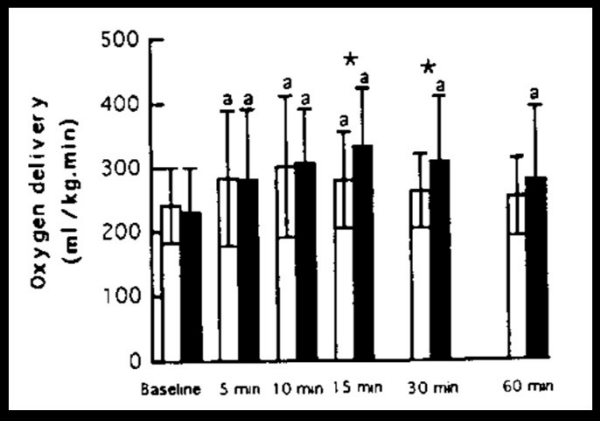
- We routinely use 5 ml/kg/hr for most dogs/cats, higher if concurrent deficit
- Due to convention more than anything else

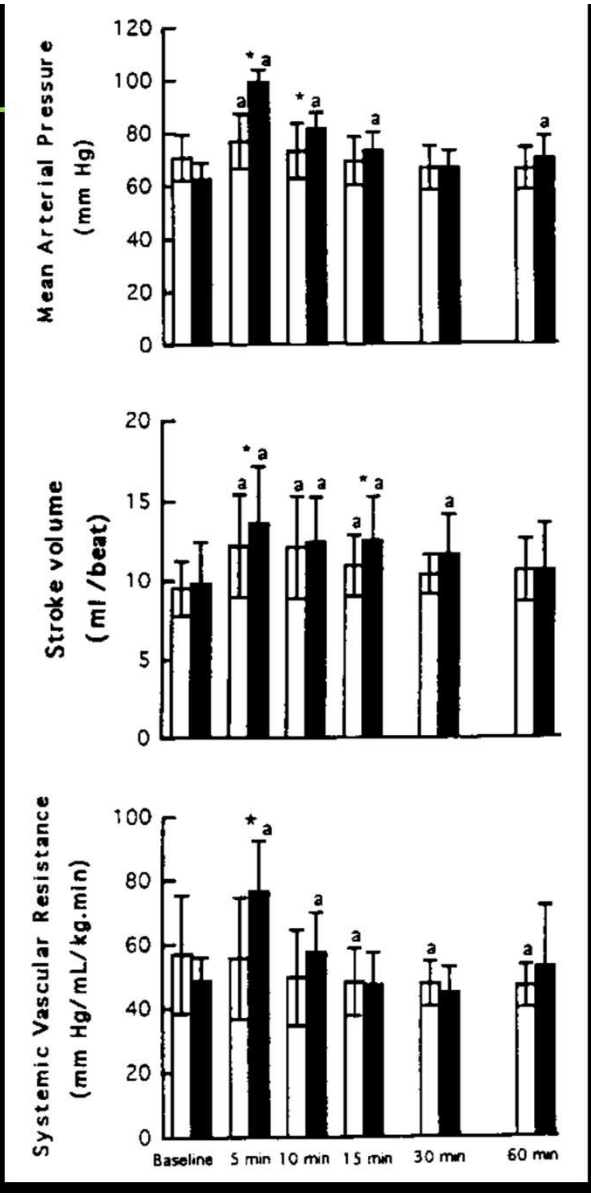
How you should implement boluses:

- To address calculated deficits
- 10-20 ml/kg over 10 min; repeat only as needed/to correct greater deficit
- To bridge time until another treatment is started/works (e.g., atropine, Glyco, Ephedrine, NE infusion)

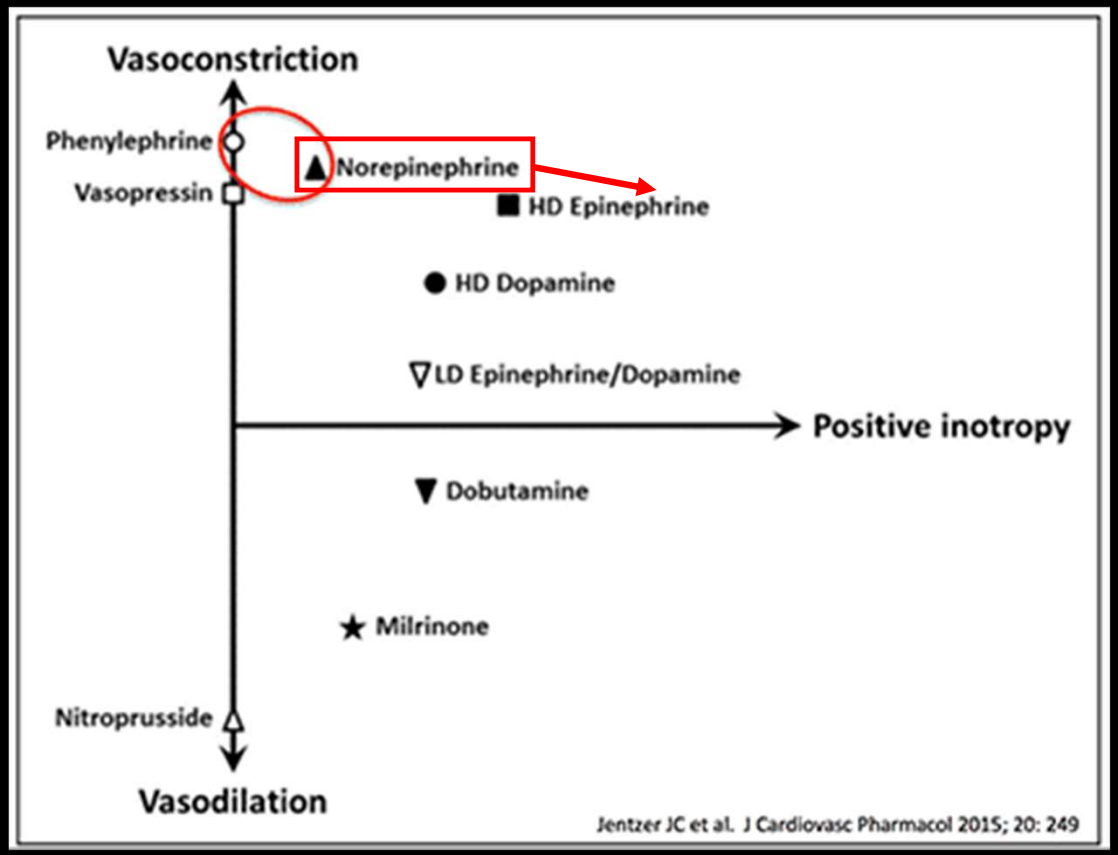
Vasopressors/inotropes

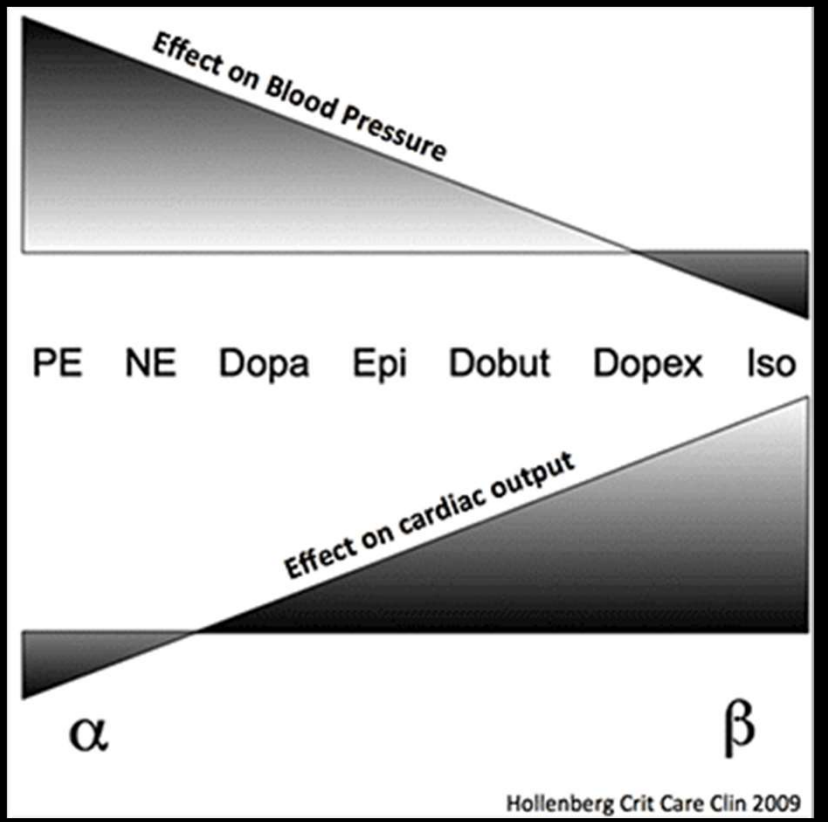
- Often not used due to fear of causing harm, they are safe when used correctly, dose calculations correct
- Can safely use all of them
  - Ephedrine, nore-/epinephrine, phenylephrine, dopamine, dobutamine
  - My vasopressor of choice - **NE**
  - Ensure dosing is correct, can cause harm
  - Our protocol → 1st ephedrine boluses/infusion, then NE infusion
- In KS, ephedrine is controlled; some states not
  - Boluses 'last' for 5-10 +/- min
  - Can be given as infusion





Vasopressors/inotropes





Vasopressors/inotropes

- If you picked two → develop comfort with NE and Dobutamine
  - Concern with vascular resistance? NE
  - Concern with contractility? Dobut.

  *Can be applied to 99% of patients

- My recommendation:
  - Invest in a syringe driver; create charge for its use
    - E.g., $1000-1,500 investment; 20$/use = 50-75 patients the pump has been paid for
  - Create protocol for norepinephrine
  - Call /email me or other anesthesiologist in your state for help protocolizing your anesthetic practices

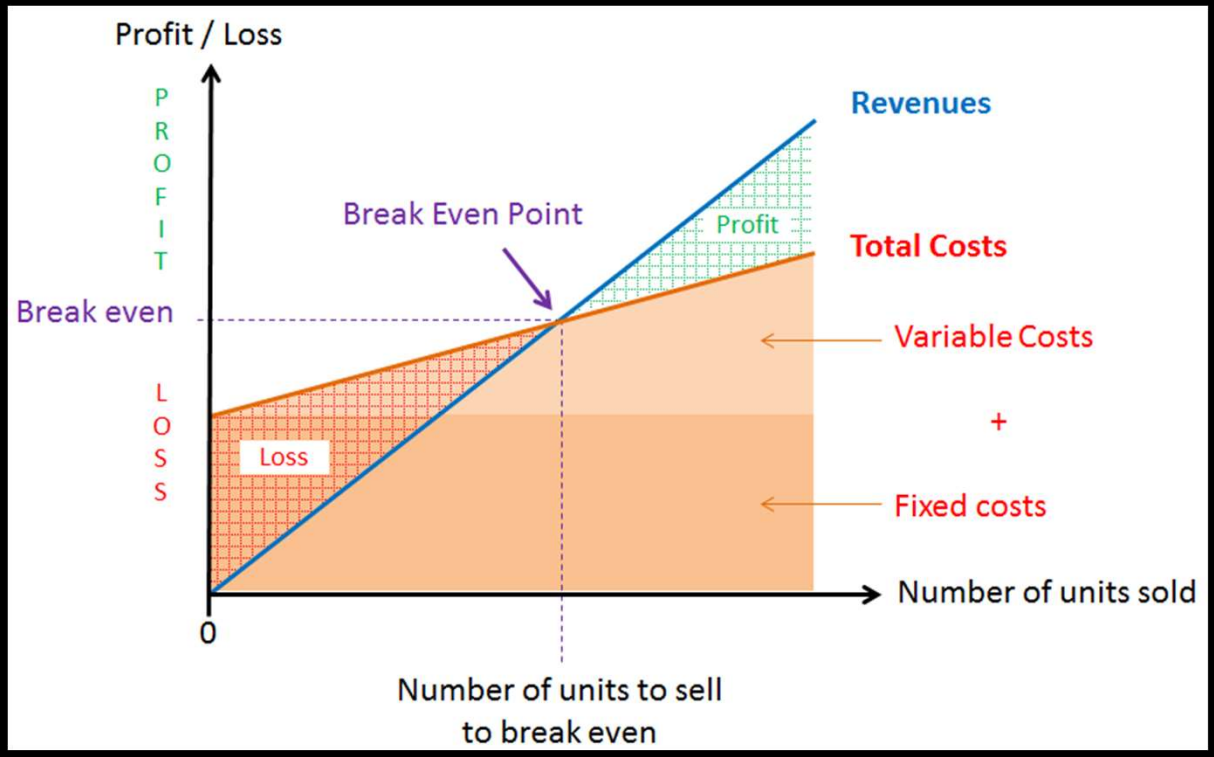
Drug	Dosing/comments
Ephedrine (alpha/beta agonist)	Sm. dogs or cats–0.2 mg/kg IV PRN Lg. dogs – 0.1-0.15 mg/kg IV PRN • Combine 1 ml vial (50 mg/kg) to 49 ml NaCl = 50 ml of 1 mg/ml → 0.1 ml/kg = 0.1mg/kg • Infusion of this formulation: 0.6ml/kg/hr = 10mcg/kg/min (titrate up to 20mcg/kg/min) • Sterilely draw from and keep mixture for 3-5 days* • *Cats: HR tends to increase from ephedrine • *Dogs: HR often stays the same or decreases
Norepinephrine (alpha>>beta agonist)	0.45ml NE (1 mg/ml) + 30 ml NaCl=30 ml (15 mcg/ml) • 1 ml/hr = 0.05 mcg/kg/min per 5 kg • 5 kg cat/dog = 1 ml/hr • 10 kg dog = 2 ml/hr • 15 kg dog = 3 ml/hr; etc. • Start at 0.05 mcg/kg/min, double q 3-5 min until normotensive
Epinephrine (beta/alpha agonist)	Mix 0.25 ml (250 mcg) into 250 ml 0.9 NaCl bag = 1 mcg/ml solution • **0.1** mcg/kg IV q 2-3 min • 10 kg dog = 1 ml • 5 kg cat = 0.5 ml

Return on Investment and Cost Containment

Practice Cost Containment

Cost containment in healthcare is all about finding ways to save money and reduce spending without sacrificing superior patient care.

Not having standard monitoring or anesthetic equipment is not practicing cost containment → it is hoping things work out with each patient.



Review / Questions

- Blood pressure is the only global assessment we can easily measure to estimate adequacy of organ perfusion in anesthetized patients.
- Minimum blood pressure for adequate end-organ perfusion is SBP ≥ 90mmHg or MAP ≥ 60mmHg; however, measurement techniques used underestimate hypotension, which alters treatment thresholds
- Hypotension is the most common reported complication from GA in dogs and cats.
- Maintenance IVF do not mitigate or prevent hypotension. Fluid boluses should be reserved for patients who demonstrate or are suspected of having a deficit.
- Approaching blood pressure is straight forward but should be individualized (e.g., patient's age, comorbidities, etc.). Interventions should progress logically/expeditiously and will be most effective when targeting primary cause of the hypotension.

Case #1

4 yr female spayed Shih Tzu (6.2 kg) is presented to you for tibial fracture fixation. The owner dropped the dog about 3 days ago.

On presentation, the patient is otherwise healthy, besides the MSK injury and associated pain/lameness. There is no overt vital sign abnormalities (HR normal, Temp normal, moist MM, no increased skin turgor etc.).

The patient is premedicated with 4 mcg/kg dexmedetomidine with 0.13 mg/kg hydromorphone IM. Induction to general anesthesia is with 0.3 mg/kg midazolam and 7 mg/kg ketamine titrated to effect.

35 minutes following induction, the patient is under general anesthesia at an 'adequate' plane of anesthesia (i.e., no palpebral, mild jaw tone, eyes are ventral); however, blood pressure just recently, over 5 min, reduced from 103 mmHg to 91 mmHg (via Doppler). HR is 112 bpm, RR is 14 (spontaneously breathing); EtCO2 is 57 mmHg; Temp is 97.9 F; there has been no significant bleeding throughout. Your anesthesia nurse is asking how they should proceed.

Assessment?

Case #1

4 yr female spayed Shih Tzu (6.2 kg) under GA for Tib/Fib Fracture.

Normocardic/ hypotensive/ hypoventilating; adequate plane of anesthesia.

Mechanism of hypotension suspected?

Ephedrine?

NE?

Dobutamine?

Lighten plane of anesthesia?

Fluid bolus?

Case #2

4 yr MN DSH (4.2 kg) is presented to you for elective OHE.

On presentation – Overtly healthy with normal physical examination; vitals normal (HR 188, RR 34, Temp 101.3, hydration status normal. Demeanor is relatively excitable, suspect unable to place catheter without sedation

Premedication: 6 mcg/kg dexmedetomidine with 0.03 mg/kg buprenorphine IM. Produced mild-to-moderate sedation. Induction of general anesthesia with propofol titrated to effect; required 3.9 mg/kg.

10 minutes post induction, 2nd Doppler BP reading is 68 mmHg (1st was 90 mmHg); HR 102; RR 15 and ISO vaporizer is at 1.75%. Depth is adequate to lighter side of adequate (i.e., no palpebral, mild-moderate jaw tone, eyes are ventral).

Assessment??

Hypotensive, Bradycardic

Cannot reduce depth at this time

What should you do?

Case #2

4 yr MN DSH (4.2 kg), healthy, under GA for elective OHE.

Premedication: 6 mcg/kg dexmedetomidine with 0.03 mg/kg buprenorphine IM. Produced mild-to-moderate sedation. Induction of general anesthesia with propofol titrated to effect; required 3.9 mg/kg.

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Mechanism of hypotension??

Treatment options?

Case #3

1 yr FI Golden (26 kg) is presented to you for elective OHE.

On presentation – Overtly healthy with normal physical examination; vitals normal (HR 108, RR panting/excited, Temp 101, hydration status normal. Demeanor is relatively excitable.

Premedication: 1 mg/kg maropitant, then 0.02 mg/kg acepromazine with 0.1 mg/kg hydromorphone IM. Produced mild sedation. Induction of general anesthesia with propofol titrated to effect; required 3.1 mg/kg.

4 minutes post induction →1st Oscillometric BP 109/60 (71) mmHg; HR 77; RR 7-9 bpm and ISO vaporizer is at 1.6%. Depth is on lighter side since just induced and only just on circuit with inhalant (O2 inhalant on 3 L/min for now to increase circuit partial pressure faster).

20 minutes later, BP has been trending down – 4th BP 96/47 (61) mmHg; Depth is adequate (not too deep) with vaporizer now at 1.5% (O2 flow had been turned down to 0.75 L/min)

Assessment?

Case #3

1 yr FI Golden (26 kg) is presented to you for elective OHE.

Mechanism of hypotension?

Options?

Case #3

11 yr FI Golden (26 kg) is presented to you for elective OHE.

Treated with ephedrine 0.1 mg/kg IV once, 3 min later → HR and BP relatively unchanged; 5 min later BP 94/44 (54) mmHg; repeated ephedrine dose, BP increased to 100/50 (64)

Next reading, BP decreased to 95/44 (59); HR 60-64 bpm

What is happening?

Options?

NE infusion

Constant rate infusions

- E.g.,: Norepinephrine CRI in syringe pump
  - 0.1 mcg/kg/min = 5 ml/hr
  - 0.1 mcg/kg/min x 26 kg = 2.6 mcg/min x 60 min = 156 mcg/hr → will make 4 hr worth; 156mcg/hr x 4 hr = 624 mcg NE (which is 1 mg/ml) → 0.62 ml NE
  - 4 hours worth at 5 ml/hr = 20 ml total infusion volume = simply add 0.62 ml NE to ~20 ml NaCl → administer at 5 ml/hr (which will = 0.1 mcg/kg/min)

- So if patient hypotensive, begin infusion; 5 min later patient still hypotensive? → double CRI rate (e.g., 10 ml/hr = 0.2 mcg/kg/min); Patient blood pressure responds? Perfect, leave it, or turn down as needed every few minutes if BP elevated significantly

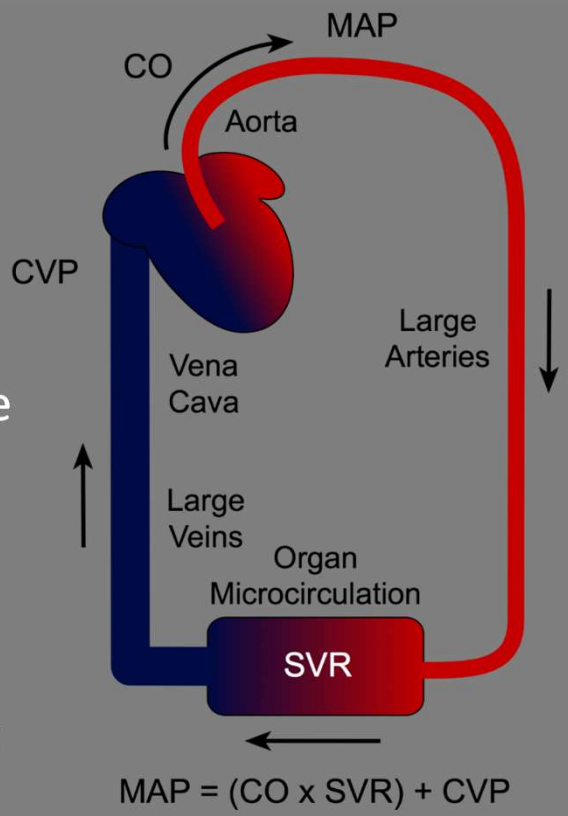
Case #3

Acepromazine and hypotension

Acepromazine molecule = analog to catecholamines (structurally similar)

Binds to alpha-1 adrenergic receptors, but does not agonize them (i.e., competitive antagonist)

Majority of alpha-1 receptors on venous side → exacerbates blood volume shift (reduces preload, thus CO, and BP)



Dexmedetomidine and hypotension

Dexmed = alpha-2 agonist → receptors present peripherally & centrally

Peripheral effects – vasoconstriction/ reflex bradycardia

Central effects— reduced sympathetic tone = reduced HR, reduced vascular tone, reduced contractility

Both occur simultaneously; but, peripheral effects only last 20-30 min → then subsequent vasodilation, continued bradycardia

Case #4

- 1.5 y MN (28 kg) poodle recovering following routine castration and gastropexy
  - Premedication: Maropitant 1 mg/kg; Dexmed 3 mcg/kg and Hydro 0.08 mg/kg IM
  - Induction was propofol titrated to effect, 2.7 mg/kg used
  - Maintenance was isoflurane titrated to effect; vaporizer dial settings ranged throughout (1.2-2%; though last 45 min before recovery was 1.6%); patient was hypotensive once but depth was modified and BP resolved right away. Otherwise normal anesthetic event
  - Procedure(s) lasted 70 minutes; another 0.04 mg/kg hydromorphone administered IV before recovery.

  - Patient is placed in recovery. Temp is 97.8 F, still anesthetized (eyes ventral; no palpebral) but breathing spontaneously. 8 min later the patient swallows, so is extubated. The patient is in sternal recumbency with a forced air warmer on it.
  - The attending anesthetist steps away for a few minutes to get ready for the next procedure while this patient continues to recover.

  - 5 minutes later the anesthetist returns, the dog is apneic/no pulses – compressions initiated, unsuccessful in the end. What happened?

Case #4 version #2

- 1.5 y MN (28 kg) poodle recovering following routing castration and gastropexy
  - Premedication: Maropitant 1 mg/kg; Dexmed 3 mcg/kg and Hydro 0.08 mg/kg IM
  - Induction was propofol titrated to effect, 2.7 mg/kg used
  - Maintenance was isoflurane titrated to effect; vaporizer dial settings ranged throughout (1.2-2%; though last 45 min before recovery was 1.6%); patient was hypotensive once but depth was modified and BP resolved right away. Otherwise normal anesthetic event
  - Procedure(s) lasted 70 minutes; another 0.04 mg/kg hydromorphone administered IV before recovery.

  - Patient is placed in recovery. Temp is 97.8 F, still anesthetized (eyes ventral; no palpebral) but breathing spontaneously. 5 min later SpO2 94%; another minute SpO2 92%; then 90%.
  - SpO2 is noticed, an oxygen mask is placed over the intubated patient's face – 1 min later SpO2 increases to 98%; patient is observed to swallow, patient is extubated with oxygen remaining.
  - Five minutes later oxygen is removed, SpO2 drops to 94% then settles and remains there for a minute, then increases to 95% - patient continues to recover uneventfully. What happened?

Case #4

- What is the cause of the desaturation?
- Hypoventilation associated hypoxia, hypoxemia → fatal arrythmia and arrest
  - THE most common reason patients die perioperatively

  - Hypoventilation = elevated PCO2 in the alveoli = reduced PO2 in the alveolus = hypoxic mixture
  - Hypoventilation is tolerated under GA, only when O2 is supplemented

- Every patient recovering should have pulse-oximetry
  - SpO2 ≤ 92% = oxygen supplementation (recheck SpO2 in 5 min, off O2)
  - SpO2 > 93% = no oxygen supplementation needed; continue monitoring SpO2 until up to temperature/patient doesn't allow it/head up etc.; suppl. O2 if SpO2 drifts, PRN
  - Post-operative hypoventilation improves as inhalant is ventilated out (can take 5-15+ min); slower in severely hypothermic patients; influenced by recent opioid/sedative administration

Case #5

Give me a scenario