Fostering Hemodynamic stability during outpatient sedation

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Hemodynamic stability:

a physiologic state where the circulatory system effectively maintains adequate and consistent blood flow and pressure to perfuse the vital organs.

Important Considerations for assessing Hemodynamic Stability

Expert opinion is to maintain intraoperative BP targets above MAP ≥60 to 65 or SBP >90 mm Hg.

There is currently insufficient trial evidence to support higher BP targets.

Thompson A, et. al. Guideline for Perioperative Cardiovascular Management for Noncardiac Surgery: A Report of the ACC/AHA on Clinical Practice Guidelines. Circulation. Sep 2024

Pulse oximetry is reliable with a systolic blood pressure > 80 mmHg. The lower the BP, the lower the pulse oximetry readings leading to a bias of up to -45%.

Not 100% Accurate!

C. Cro… …sociation between blood pressure and pulse oximetry accuracy: an observational study in patients with suspected or confirmed COVID-19 infection BMJ Em Medicine Journal Volume 40, Iss 3

(ATLS)}, palpable pulses help estimate systolic blood pressure (SBP) in trauma: carotid only suggests SBP 60-70 mmHg, carotid & femoral indicates SBP 70-80 mmHg, and a radial pulse present means SBP >80 mmHg.

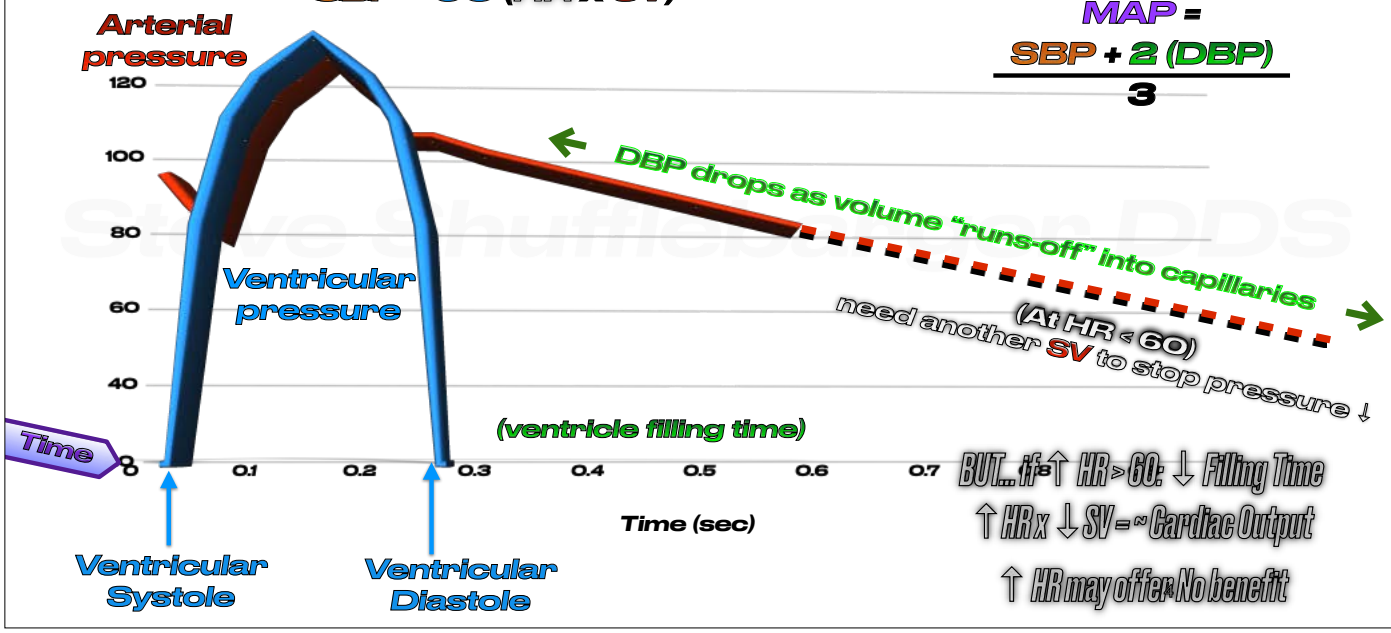
Advanced Trauma Life Support (ATLS) guidelines are written by the American College of Surgeons (ACS) Committee on Trauma

the Physiology behind BP

DBP = arterial resistance. (diameter + volume)

SBP = CO (HR x SV)

$$MAP = \frac{SBP + 2(DBP)}{3}$$



Cardiac Output (CO) = heart rate (HR) x stroke volume (SV)

At end of diastole: ventricles are full (EDV)

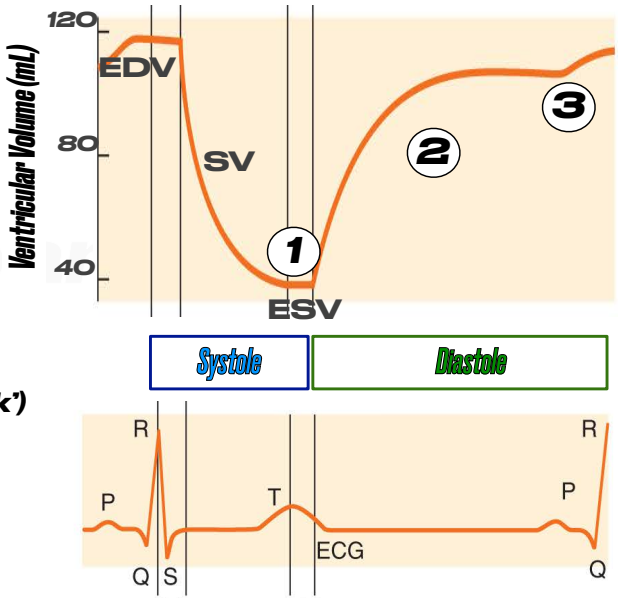
They eject SV
(EDV – ESV = 80)

then, Reload—>

1. *Filling Commences at ESV*
2. *Initially due to Passive Filling*
3. *Finishes with atrial contraction ('atrial kick')*

ECG: (End of T thru Mid P)

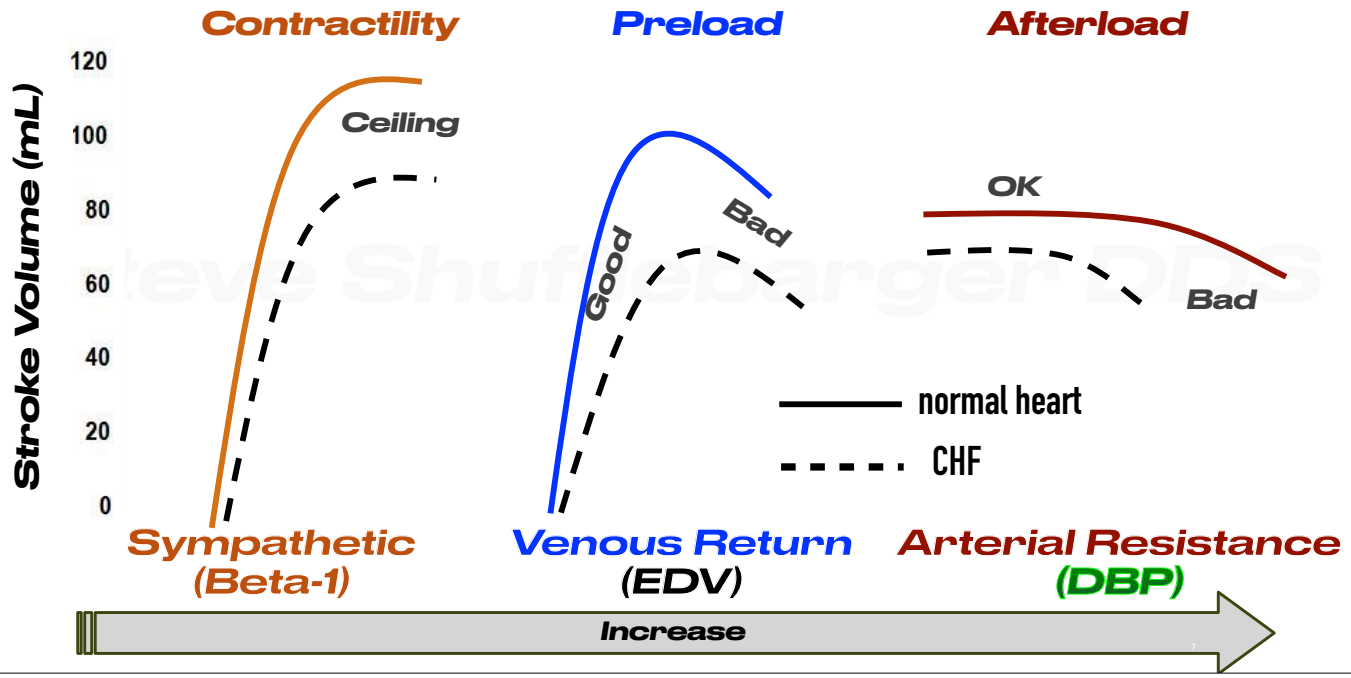
Notice Passive Filling is KEY!



	HR	x	SV	=	CO	BP
Making Blood Pressure	44 (This patient either Has or Needs a Pacer!)	x	100 mL	=	4.4 L/m	low
	60	x	100 mL	=	6 L/m	better
	100	x	60 mL	=	6 L/m	same
	160 (This is a Crisis!)		25 mL	=	4 L/m	low

same BP, but less favorable physiology for 79 yoa!

3 factors influence Stroke Volume



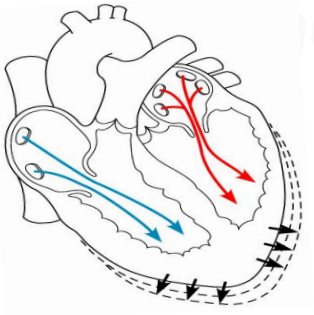
"Pre" vs. "After" load

"Loads" are terms to emphasize strains or tensions on the heart muscle.

Preload

= Diastolic Wall Tension

Determined by venous return



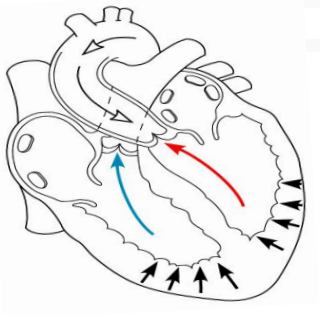
Venodilation ↓ venous pressure & return

Venoconstriction ↑ venous pressure & return

Afterload

= Systolic Wall Tension

Determined by arterial Resistance



Arterial dilation ↓ pressure & resistance

Arterial constriction ↑ pressure & resistance

General Guidance. Not absolute

"Calculation of maintenance fluid requirements is of limited value in determining intraoperative fluid requirements"

calculation of .. fluid requirements is useful for estimating water and electrolyte deficits... from preoperative restriction of .. food and fluids

Hourly and Daily Maintenance Water Requirements

Weight (kg)	Water (mL/kg/hr)	Water (mL/kg/day)
1 - 10	4	100
11 - 20	2	50
21 - n+1	1	20

In healthy adults, sufficient water is required to balance:

gastrointestinal losses (100 to 200 mL/day)

insensible losses (500 to 1,000 mL/day)

and urinary losses (1,000 mL/day)

Baresh; Clinical Anesthesia. pg 391

Mixed Messages on Intake

Guidelines vary widely and may only be helpful at the extremes?

On average, a sedentary adult should drink 1.5 L of water per day. Water is the only liquid nutrient that is really essential for body hydration.

Jéquier E, Constant F. Water as an essential nutrient: the physiological basis of hydration. Eur J Clin Nutr. 2010 Feb;64(2):115-23. Epub 2009 Sep 2.

Steve Shufflebarger needs 3.22 L of water per day when he is sedentary.

Baresh et al; Clinical Anesthesia : 2018; pg 391

To be well hydrated, the average sedentary adult man must consume at least 2.9 L fluid per day, and the average sedentary adult woman at least 2.2 L

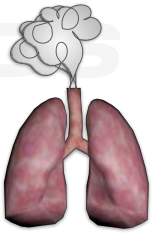
Kleiner SM. Water: an essential but overlooked nutrient. J Am Diet Assoc. 1999 Feb;99(2): 200-6. J Am Diet Assoc 1999 Apr;99 (4):411

Water requirements aren't based on minimal intake because it might lead to water deficit via factors that modify need (climate, physical activity, diet, NPO duration, etc.).

Iversen PO, Nicolaysen G. Vann--for livet [Water--for life]. Tidsskr Nor Laegeforen. 2003 Dec 4;123(23):3402-5. Norwegian. PMID: 14713981.

patients lose H^2O when fasting

- Urine – most significant under normal conditions
  - Renal filtration and subsequent secretion of H^2O
- Sweat - can be most significant if extreme exercise or heat
  - depends highly on environment, temperature, activity level, clothing, stress, etc.
- Respiration – a massive alveolar surface area facilitates gas exchange-> and fluid exchange !!
  - manufacturing ATP yields CO^2 and H^2O —> H^2O loss increases with activity
  - ↑ Temperature and ↓ Humidity = maximizes H^2O loss via exhaled CO^2 and H^2O vapor.
    - i.e. flying in an airplane (Dry), sitting outside (Hot), or hiking in the Arizona desert (Hot, Dry, and active) can contribute to dehydration.



- Substances : —> Natural diuretics like caffeine and alcohol
  - Alcohol inhibits ADH (vasopressin)
  - Not just urinating from excess liquid consumed, but excess urination vs. same volume of non diuretic liquid consumed

Patients' ability to replace H^2O during fasting?

- PO Plain Water – "the standard"
  - The tonicity of pure water is essentially zer

In most ASA I and II patients without acute illness, NPO duration is likely the critical factor in determining fluid status.

Saraghi M.
Intraoperative Fluids and Fluid Management for Ambulatory Dental Sedation and General Anesthesia.
Anesth Prog. 2015 Winter;62(4):168-76;

- Food – vary widely in water content
  - We consume a surprisingly large amount of our water in foods
  - Foods contain salt, sugar and other Osmoles
  - Solid foods contribute approximately 1L water?.. With an additional 250 mL coming from the water of oxidation.
  - High fat diets reduce water consumption from food

- PO Other Beverages – may have added Electrolyte, Vitamins, Minerals, Carbohydrate, Fat and/or Protein.
  - vary widely in tonicity
  - vary widely in caloric content
  - EtOH and Caffeine can promote water loss

Kleiner SM. Water: an essential but overlooked nutrient. J Am Diet Assoc. 1999 Feb;99(2): 200-6. J Am Diet Assoc 1999 Apr;99 (4):411

Detecting fluid loss

Inpatient vs. Outpatient settings

- In the Hospital ICU: Both Hydration and Volume status are monitored via catheters in major veins (vena cava) with pressure transducers quantifying central venous pressure.
  - Further, many ICU Patients have Bladder catheters measuring urine output.
    - Kidneys are very sensitive to volume changes.
      - like the brain, the kidneys weigh ≈2% of total body weight, but receive ≈ 20% of total blood flow.
        - urine output drops—> typically an early sign of low circulating volume

- Outside the Hospital —no catheters!... (unquantified) urine output may be the best indicator of hydration status
  - Fluid balance generally regulated by poorly understood mechanisms of thirst*, hunger, and urine elimination. These mechanisms cross multiple organ systems.
  - Contrary to logic, studies mostly fail to reliably demonstrate urine color as an indicator of dehydration or volume depletion.

Iversen PO, Nicolaysen G. Vann--for livet [Water--for life]. Tidsskr Nor Laegeforen. 2003 Dec 4;123(23):3402-5. Norwegian. PMID: 14713981.

Consequences of fluid loss

- Dehydration or Volume depletion
  - ↓plasma volume = ↓ preload = ↓ contractility ——> Results ↑ HRs
  - In performance, ↑ chamber volume is desirable to ↑ contractility and EF.
    - early sign of dehydration: elevated HR—> because HR x SV = CO.
      - in dentistry, stress hormones: Catecholamines, (Epi, NE) contribute to even higher HRs.

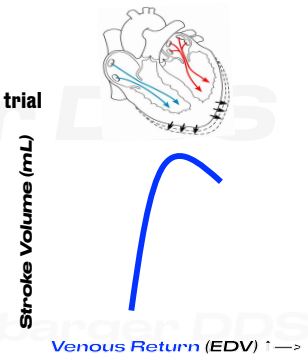
Physical findings of dehydration or hypovolemia may include changes of ↓BP and narrowing of pulse pressure, but more noticeably ↑HR.

Stoelting RK, Miller RD. Basics of Anesthesia. 5th ed.; 2007

Morgan GE, Mikhail MS, Murray MJ. Clinical Anesthesiology. 4th ed; 2006.

- Loss of 1% to 2% body mass in dehydrated vs. hydrated athletes in a 90 min time trial...
  - HR ↑ 15 BPM — core body Temp ↑ ≥ 1 °C — relative perceived exertion ↑ ≥ 15%. — 15% longer to complete time trial
  - 5 - 10% ↓in power output

- Practical Consideration:
  - If a provider sees 5-10 BPM higher under same conditions —> consider hydration status relative to normal



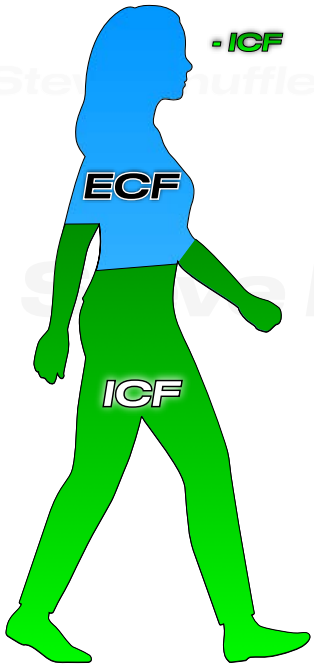
- CO is 1 of 2 things that matter for cardiorespiratory performance. The second is at the tissue level — VO^2
  - Hydration doesn't meaningfully impact VO^2 (how much O^2 is utilized), unless actual blood loss.
    - Consider recent donations, injuries, renal disease, or surgeries.

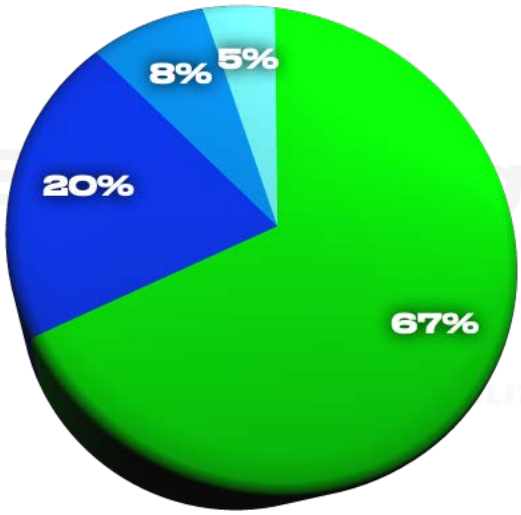
LJ James, et al; Hypohydration impairs endurance performance: a blinded study. Physiologic Reports Vol 6 Iss 12 July 2017

H M. Logan-Sprenger, et al; The effect of dehydration on muscle metabolism and time trial performance during prolonged cycling in males. Physiologic Reports Vol 3 Iss 8 Aug 2015

The Big Picture

- ICF
- Interstitium
- Vasculature
- Everything Else





Summary of total body water and location:

67% inside cells.

20% in the Interstitium

8% in the Vascular System.

5% everything else

Replacing fluid and Osmosis:

equilibrium via diffusion of H^2O across semipermeable membranes

Tonicity compares concentration Solution A vs. Solution B

Human Tonicity is relative to the concentration of a Solution A = ICF , because cells are the dominant source of water

Sodium is the predominant determinant of human tonicity / osmolality

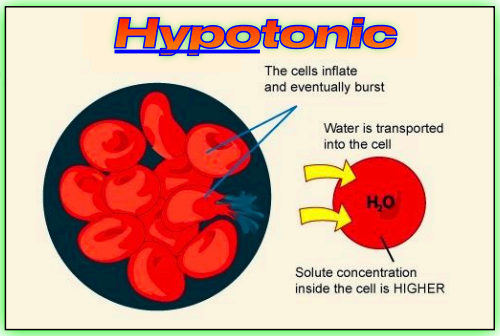
RBCs illustrate this— i.e., put RBCs into a solution B, see how H^2O moves between the two.

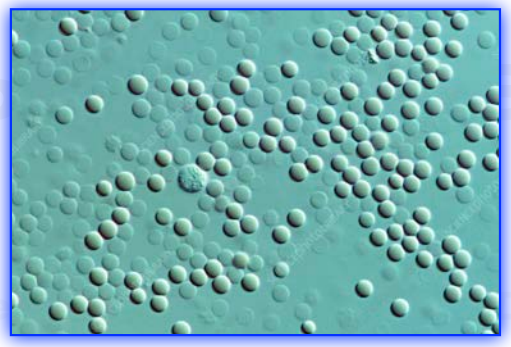
consider Tonicity to optimize replacement

Tonicity

if administer Hypotonic solutions

Hypotonic– RBCs (A) into a less concentrated (dilute) solution B



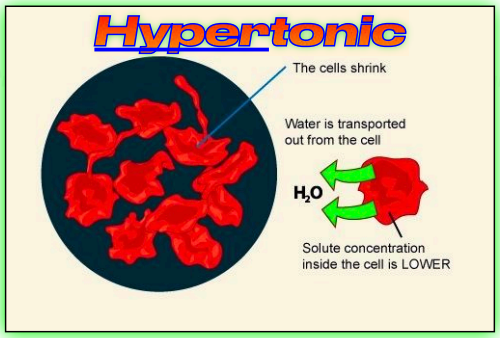


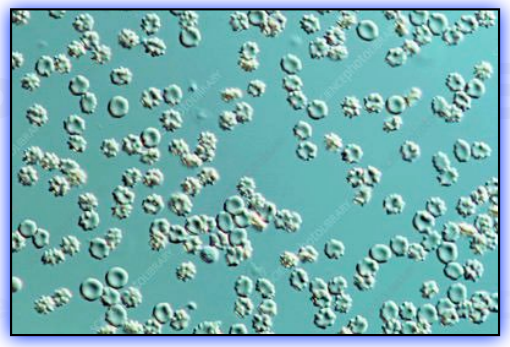
FLUID flow IN of the cells > FLUID flow OUT to the cells . The RBCs expand and can rupture

Tonicity

if administer Hypertonic solutions

Hypertonic – RBCs (A) in a very concentrated (high tonicity) solution B



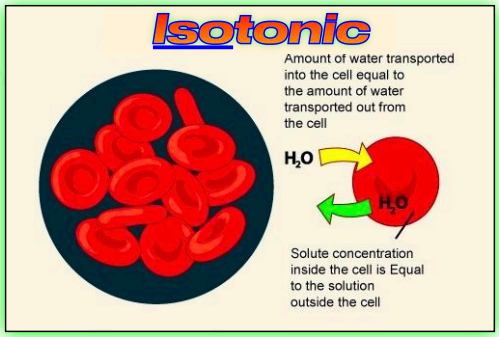


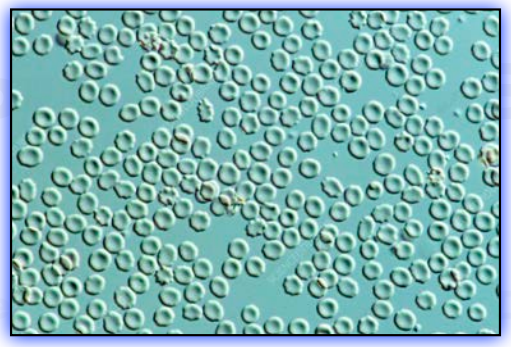
FLUID flow OUT of the cells > FLUID flow IN to the cells . The RBCs "shrivel up"

Tonicity

if administer Isotonic solutions

Isotonic – put RBCs (A) into solution B with the same tonicity





FLUID flow OUT of the cells = FLUID flow IN to the cells . The RBCs don't change in shape at all

Tonicity and IV solution selection

- **generally, IV fluids must be nearly , but not exactly isotonic. baseline isotonic = 0.9% NaCl**
  - **can adjust up or down NaCl, other minerals/ions, and glucose (all contribute to tonicity) prn.**
    - **Na^+ and it's counter-ion Cl^- are the primary drivers of human tonicity**
      - **NaCl (salt) accounts for about 85% of the tonicity of the extracellular space.**
- **Glucose and Blood Urea Nitrogen contribute to tonicity**
  - **BUN -> the amount of N coming from Urea (waste product of the urea cycle eliminating excess protein).**
  - **The general formula which gets you very close to estimate Plasma osmolality**

$$\text{Plasma Osmolality} = 2[Na+] + (\text{Glucose} / 18) + (\text{Blood Urea Nitrogen} / 2.8)$$

the formula is complicated, just remember that an isotonic solution to human plasma is ≈ 290 milliequivalents/kg

as we start to think about rehydration and where we need to be in relation to that

selecting IV fluids to replace losses

commercially available crystalloid solutions

The NEW ENGLAND JOURNAL of MEDICINE

	Glucose	Sodium	Chloride	Potassium	Buffer†	Calcium	Magnesium	pH	Osmolarity
	g/dl	*millimoles per liter*							*mOsm/liter*
Human plasma	0.07–0.11	135–144	95–105	3.5–5.3	23–30	2.2–2.6	0.8–1.2	7.35–7.45	308
5% Dextrose in water	5	0	0	0	0	0	0	3.5–6.5	252
4% Dextrose in 0.18% saline	4	30	30	0	0	0	0	3.5–6.5	282
5% Dextrose in 0.2% saline	5	34	34	0	0	0	0	3.5–6.5	321
5% Dextrose in 0.45% saline	5	77	77	0	0	0	0	3.5–6.5	406
5% Dextrose Ringer's lactate	5	130	109	4	28	1.5	0	4.0–6.5	525
5% Dextrose in 0.9% saline	5	154	154	0	0	0	0	3.5–6.5	560
5% Dextrose multiple electrolytes injection, type 1, USP	5	140	98	5	50	0	1.5	4.0–6.5	547
Ringer's lactate	0	130	109	4	28	1.35	0	6–7.5	273
Ringer's acetate	0	130	112	5	27	1	1	6–8	276
Hartmann's solution	0	131	111	5	29	2	0	5.0–7.0	278
0.9% Saline	0	154	154	0	0	0	0	4.5–7	308
Multiple electrolytes injection, type 1, USP§	0	140	98	5	50	0	1.5	4.0–6.5	294
Isotonic electrolyte solution¶	0	140	127	4	29	2.5	1	4.6–5.4	304

Moritz ML, Ayus JC. N Engl J Med 2015;373:1350-1360.

IV Fluids to Hydrate

Hypotonic, Isotonic, Hypertonic?

Solution B (1 L)	Change ICF	Change ECF
Normal Saline	0	1000 mL
Lactated Ringer's	100 mL	900 mL
D5W	667 mL	333 mL

Manipulating BP : CO = HR x SV

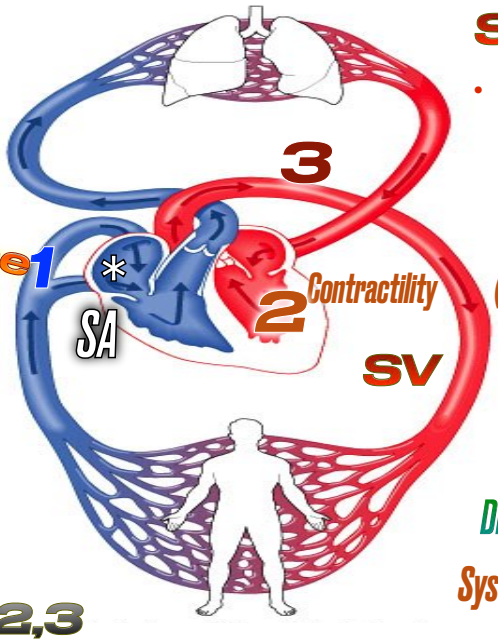
HR - SA Node

- Sympathetic ↑ (β_1)
- Parasympathetic ↓ (M_2)

Atropine

SV

- Preload (+) : Venous Return
  - elevate legs / IV fluid
  - Venoconstriction = ↑ (α)
  - Venodilation = ↓ (β_2) ??



SV

- Afterload (-) : DBP
  - Arterial Constriction = ↑ (α)
  - Arterial dilation = ↓ (β_2)

(+ ↑) = (β_1)

Stroke Volume 1,2,3

Diastolic BP [Arterial Resistance]

Systolic BP is KEY for EM management

MAP matters for ambulation

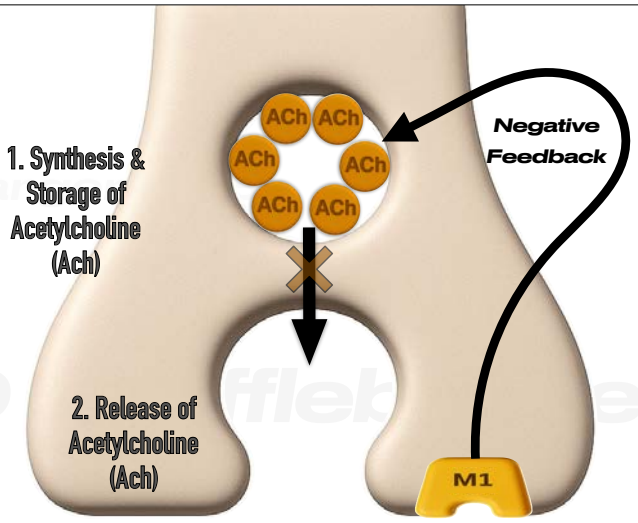
Synaptic Vesicle of an Cholinergic Neuron

ACh

Peripheral —> Parasympathetic effects

Central —> Excitatory

AchE



1. Synthesis & Storage of Acetylcholine (Ach)

2. Release of Acetylcholine (Ach)

ACh = AcetylCholine

Atr = Atropine

3. Activation of PERIPHERAL Cholinergic Receptors: Muscarinic (M_{1-3})

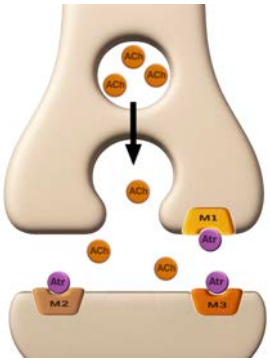
4. Termination by of Ach by Acetylcholinesterase (AchE)

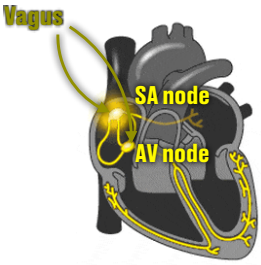
Anticholinergic Atropine comes in multiple concentrations.

Bradycardias

Doses < 0.5 mg associated with paradoxical vagotonic effects further slowing HR !

Bradycardia Dose= 1 mg





Likely blocks M_1 presynaptic receptors, preventing negative feedback and allowing more Ach release.

if NO Bradycardia.. ↓Secretions Dose 0.3-0.4 mg

Goodman and Gilman's 13th edition 2018

AHA ACLS manual 2020

Katzung's 14th edition 2018.

Adrenergic Agents - "Pressers?"

Agonist	Beta-1	Beta-2	Alpha-1
Epinephrine	+++	++	++
Norepinephrine	++	O	++
Ephedrine (Indirect)	++	+	++
Dopamine	++	+	++
Albuterol	O,+	+++	O
Phenylephrine	O	O	+++

non-specific Hypotension?

Vasopressors	Epinephrine	Ephedrine	Phenylephrine
Action	α, β_1, β_2	α, β_1, β_2 / + Indirect	α Danger!
SBP	↑ (Contractility)	↑ (Contractility)	↑ (venous return)
DBP	↓ (↑ high dose)	↓ (↑ high dose)	↑
MAP	↔ (↑ high dose)	(↑ high dose)	↑
HR	↑ (SA Node)	↑ (SA Node)	↓ (Reflex to MAP)
Duration	3-5 min	1-2 hr	15 min
Formulation	1 mg/mL	50 mg/mL	10 mg/mL
Dosage (IV increment)	10 mcg	10 mg	0.1 mg

Safe! YES!

Tuberculin 1mL

Dated! NO!

1. Load 0.1mL (100mcg)
2. Dilute to 1 mL
3. 0.1 mL (10 mcg)

1. Load 1 mL (50mg)
2. No Dilution
3. 0.1-0.3 mL (5-15mg)

1. Load 0.1 mL (1mg)
2. Dilute to 1 mL
3. 0.1 mL (0.1mg)

Danger! NO!

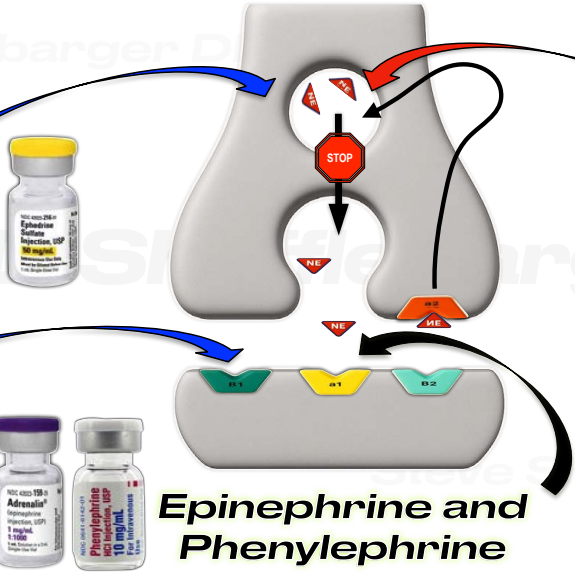
Low BP 2ndary to an A-2 agonist?

Ephedrine

Stimulates NE release from presynaptic neurons

+

Activates postsynaptic receptors (α, β)

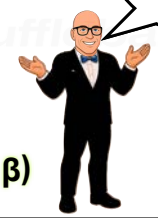


Epinephrine and Phenylephrine

——-> only activate postsynaptic receptors (α, β)

Alpha-2 Agonists

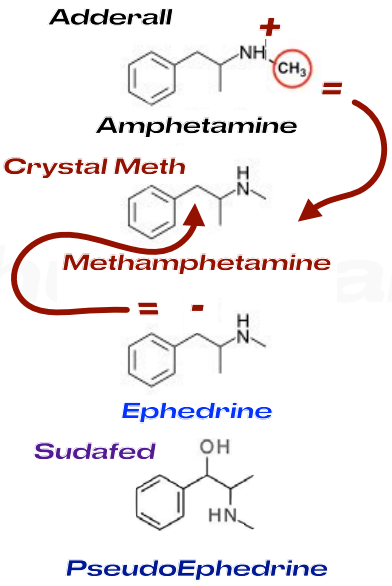
Inhibit NE release from presynaptic neurons.



"On" the Adrenergic "Throttle"

Note the Minor differences between these chemical structures

Ephedrine is a dehydrogenase RXN from Crystal Meth



Adderall is one methyl group from Crystal Meth

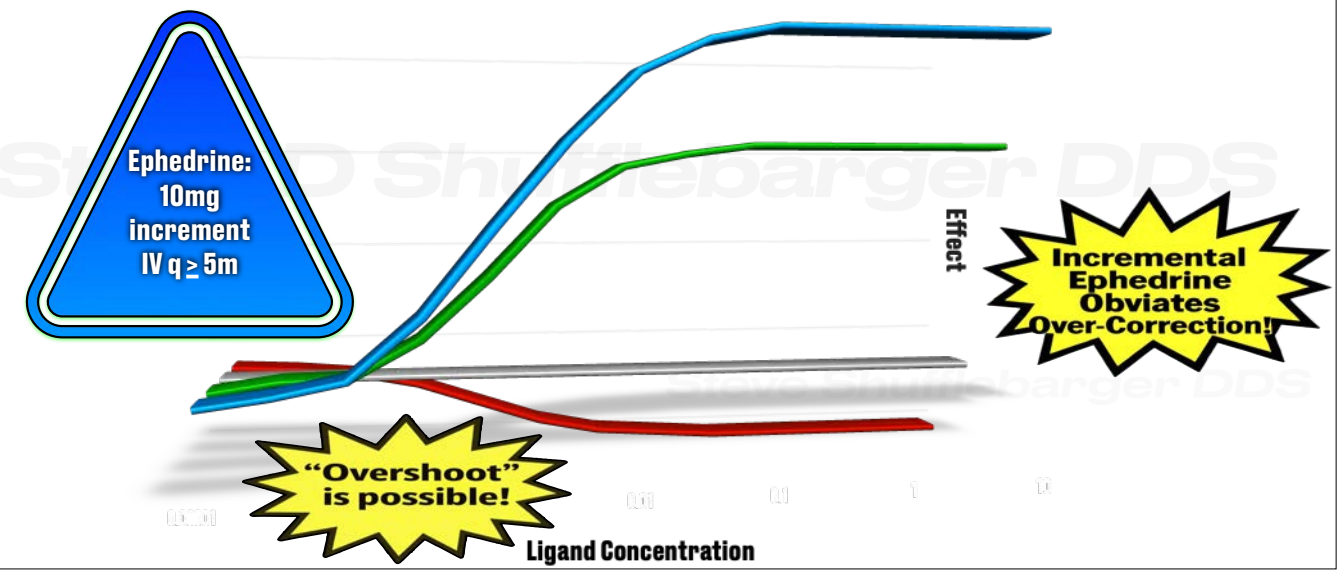
Amphetamines are CNS stimulants that increase DA, NE, and 5-HT (lesser extent) in synaptic clefts via several mechanisms AND they inhibit MAO metabolism of these same neurotransmitters.

Chemistry 101

Martin D, Le JK. Amphetamine. [Updated 2023 Jul 31]. In: StatPearls. Treasure Island (FL): NIH StatPearls Publishing; 2025 Jan

PharmacoDynamics: Ligand actions on Receptors

Opposite Action Drugs aren't quite the same as Neutral Antagonists



Hemodynamic Stability Key points

1. Measure a STANDING not seated BP measurement prior to discharge (especially when using DMET)
2. Standing MAP ≥ 65 is recommended as discharge criteria regardless of baseline BP.
3. Measure a "Baseline BP" while your patient is relaxed but NOT somnolent
4. Consider using the above baseline BP when comparing for Discharge Criteria
5. Pay attention to hydration status in Fasted Patients
6. IV Fluids aren't just useful to deliver drugs to your patient
7. Don't overreact to potential erroneous BP readings.
8. Use proper cuff size, ensure patient is still, and arm is at heart level
9. Consider ATLS pulse <-> pressure relationships when unsure of BP accuracy and remeasure.
10. Consider the patient's condition not just the number when assessing perfusion status

Questions?

HeyDrSteve@gmail.com

Optimizing the utility of Dexmedetomidine in daily practice

Steve D Shufflebarger DDS

Miami Valley Hospital Dept Medical Education

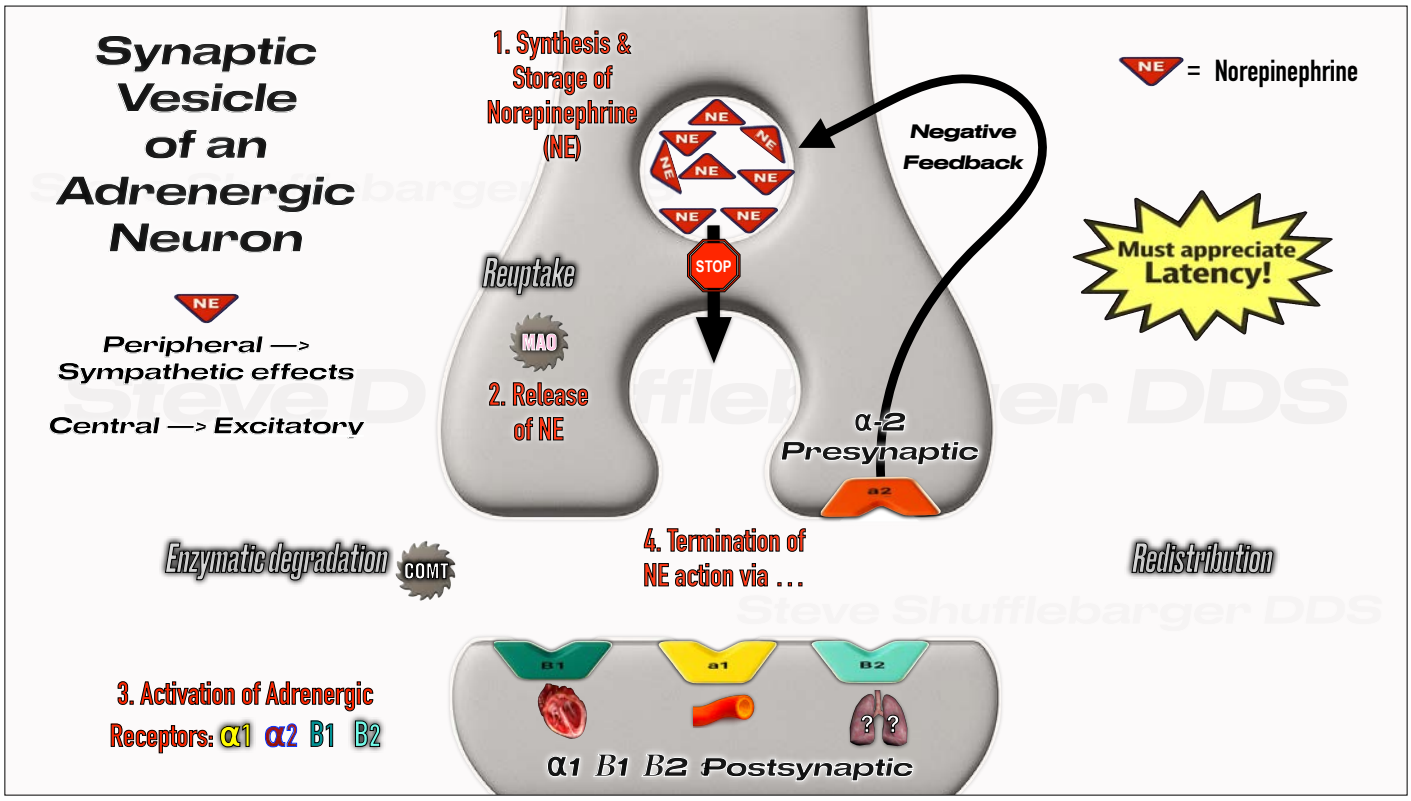
DMET Challenges, Strengths, Controversies, and Quirks

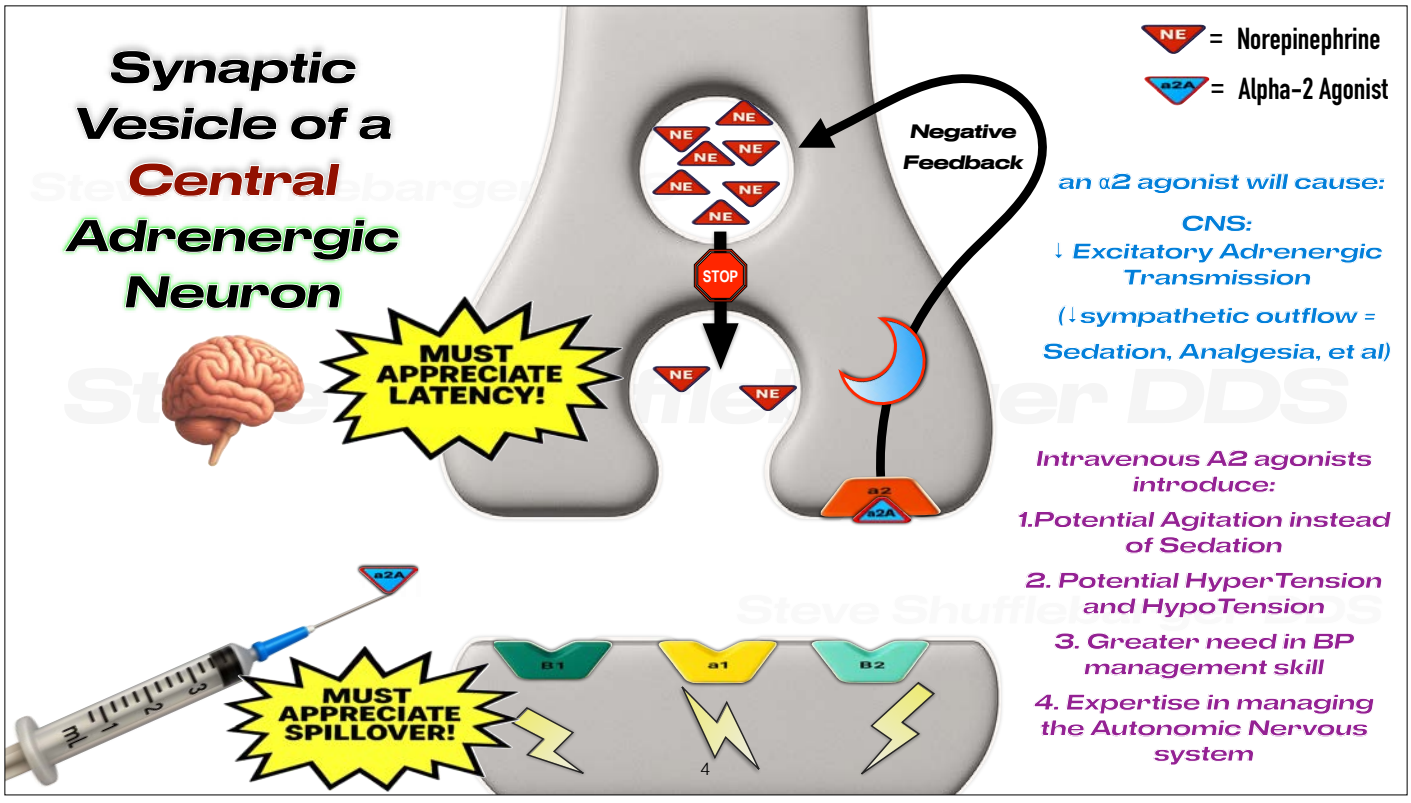
Downsides:

1. Must be given slowly
2. Can produce "Opposite Effects" when not Paced properly
3. Complications / Limitations of Use revolve around CV (not Airway and Breathing)
4. Paradox: IV DMET has a shorter Dist 1/2 life than Midazolam, but a slower onset than Midazolam
4. DMET is an indirect acting drug
5. DMET has no precise Alpha-2 antagonist approved for human use

Upsides:

1. DMET exhibits almost NO Respiratory Depressant effects
2. DMET produces excellent, predictable sedation in many "difficult to sedate" patients
3. DMET has numerous available "Opposite Action" drugs

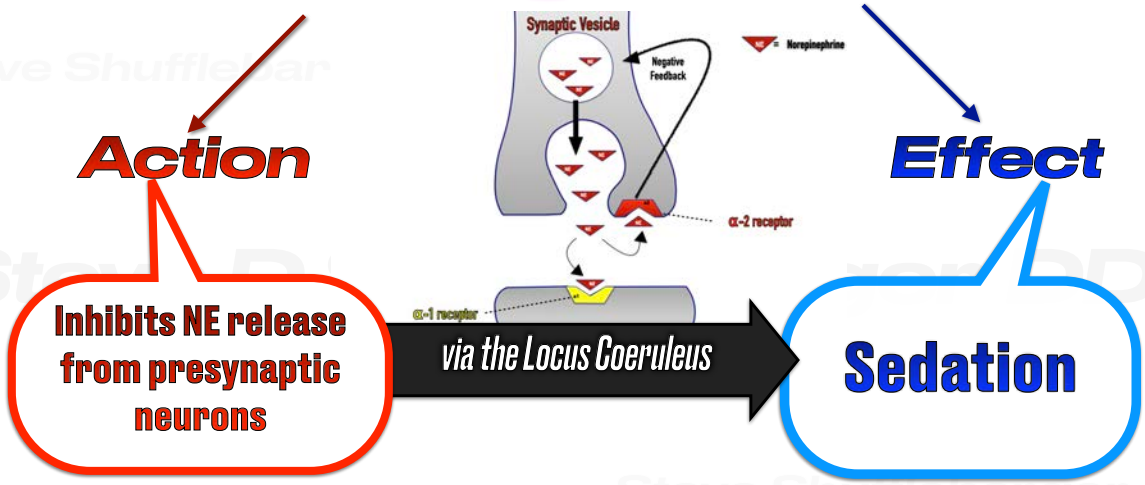

Synaptic Vesicle of an Adrenergic Neuron
NE
Peripheral —> Sympathetic effects
Central —> Excitatory
1. Synthesis & Storage of Norepinephrine (NE)
NE = Norepinephrine
Negative Feedback
Must appreciate Latency!
Reuptake
STOP
MAO
2. Release of NE
α-2 Presynaptic
a2
Enzymatic degradation
COMT
4. Termination of NE action via . . .
Redistribution
3. Activation of Adrenergic Receptors: α1 α2 B1 B2
B1
a1
B2
α1 B1 B2 Postsynaptic


Synaptic Vesicle of a Central Adrenergic Neuron
NE = Norepinephrine
a2A = Alpha-2 Agonist
Negative Feedback
an α2 agonist will cause:
CNS:
↓ Excitatory Adrenergic Transmission
(↓sympathetic outflow = Sedation, Analgesia, et al)
MUST APPRECIATE LATENCY!
STOP
a2
a2A
Intravenous A2 agonists introduce:
1.Potential Agitation instead of Sedation
2. Potential HyperTension and HypoTension
3. Greater need in BP management skill
4. Expertise in managing the Autonomic Nervous system
MUST APPRECIATE SPILLOVER!
B1
a1
B2

Pharmacologic basis of Sedation

Receptor	Ligand	Central Effect(s)
GABA	GABA	- Inhibit
Mμ, Kappa	Opioid	- Inhibit
DA	Dopamine	+ / - Excite / Inhibit
Na+ channels	Na+	+ Excite
NMDA	Glutamate	+ Excite
H1	Histamine	+ Excite
H2		+ Excite
Muscarinic	Ach	+ Excite
ß1	Epi / NE	+ Excite
ß2		+ Excite
å1		+ Excite
å2		- Inhibit

α-2 Agonists



α2 Agonists (use) an Endogenous Sleep-promoting Pathway to (produce)..Sedation

Nelson et al, Anesthesiology 2003, Vol.98, 428-436

α2 Agonists: Clinically effective Sedation. (yet,)..patients are easily + uniquely rousable

Ogawa, DDS,et al Anesth Prog. 2008 Fall; 55(3): 82–88.

Understanding drug SAFETY

Therapeutic Index (TI)

The therapeutic index is a <u>Quantitative</u> measurement of the relative safety of a drug.

LD_{50} = median Lethal dose

ED_{50} = median Effective dose

$$\textbf{TI (therapeutic Index)} = \frac{LD_{50}}{ED_{50}}$$

It is a comparison of the amount of a therapeutic agent that causes the therapeutic effect to the amount that causes toxicity.

α-2 agonists TI

CASE SUMMARIES: *3 patients accidentally .. overdosed (with) dexmedetomidine (DMET):*

1. *intraop (192 µg over 20 min)*
2. *post op 4 and 2 (rather than 0.4 and 0.2 µg/kg/h)*
3. *post op 0.5 µg/kg/min (intended 0.5 µg/kg/hour).*

Hemodynamic(s) (were) stable in all 3 patients. The most notable sign was oversedation diagnosed ...clinically or (via) BIS.

In all 3 cases, oversedation resolved ≤ 1 hr of d/c DMET.
There were no other sequelae, and ..each patient's (remaining) hospital course was unremarkable.

Victor SB Jorden, et al ; Dexmedetomidine Overdose in the Perioperative Setting : Ann Pharmacother 2004;38:803-7.

α-2 agonists TI

DISCUSSION: As of this writing, DMET dosing in excess of the label .. has been reported, but accidental DMET overdose .. has not

Excessive sedation was the only significant finding in all 3 (OD) patients.

DMET's short redistribution half-life of 6m should (aid) rapid resolution of oversedation (via) OD if .. duration of infusion (was ≤8 h).

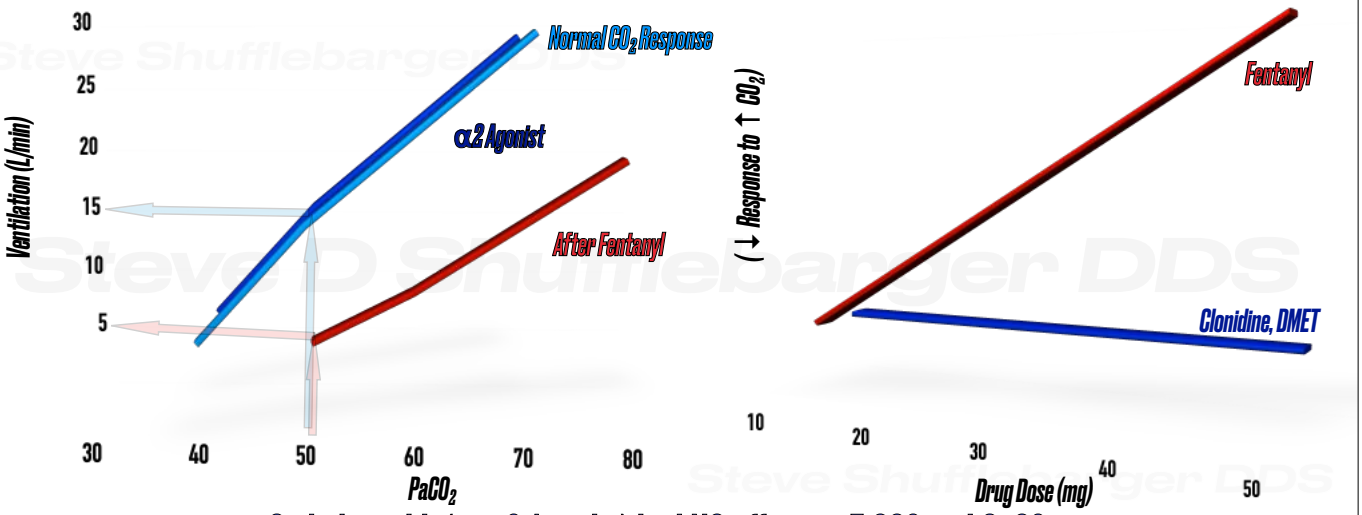
While these patients ..were hemodynamically stable, DMET may (cause) hemodynamic changes either (due to) sympatholysis at normal dose or vasoconstriction at ↑ recommended doses.

The absence of.. HTN (post) ↑ DMET concentrations suggests that DMET-induced HTN may be multifactorial, not (just from) ↑ plasma concentration

we discussed

Victor SB Jorden, et al ; Dexmedetomidine Overdose in the Perioperative Setting : Ann Pharmacother 2004;38:803-7.

One Reason A2as are So SAFE



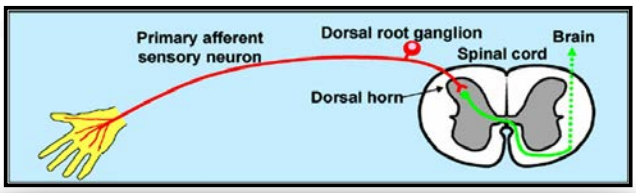
Sedation with (an α2 Agonist) had NO effect on EtCO2 and SpO2

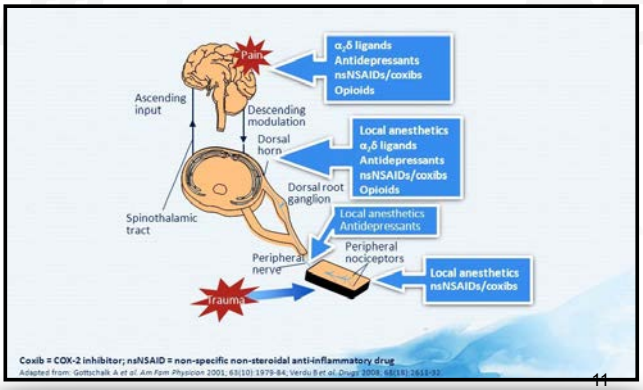
Ogawa, DDS et al.: Anesth Prog. 2008 Fall; 55(3): 82–88.

(α2 Agonists) produce..excellent sedation without compromising airway(s) or depressing respiration

J A Giovannitti, jr, DMD et al : Anesth Prog. 2015 Spring; 62(1): 31–38.

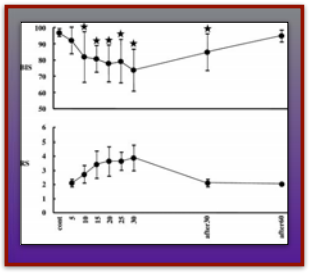
Analgesia via A2as





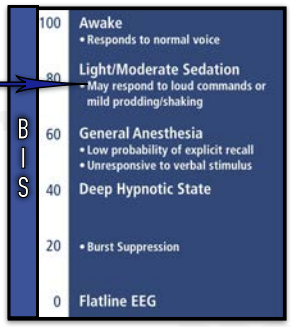
primary sedative vs. adjunct

Anterograde Amnesia?



Ramsay Sedation Scale

1 : Patient is anxious and agitated or restless, or both.
2 : Patient is cooperative, oriented, and tranquil.
3 : Patient responds to command only.
4 : A brisk response to a light glabella tap or a loud auditory stimulus.
5 : A sluggish response to a light glabella tap or a loud auditory stimulus.
6 : No response to a light glabella tap or a loud auditory stimulus.



27 G needle prick at 21 min

31% of patients recalled the prick

Ogawa, DDS,et al Anesth Prog. 2008 Fall; 55(3): 82–88.

α-2 Agonists

"Due to pharmacodynamic interactions, a reduction in dosage of (DMET) or other concomitant anesthetics, sedatives, hypnotics, or opioids may be required when co-administered."

Adding an Alpha 2 Agonist to a sedation regimen reduces:

Opioid requirements by 50-75% and BNZ requirements by upwards of 80%

Giovannitti et al, Anesthesia Progress 62:31-38 2015

Midazolam with (α2 Agonists) was Synergistic

Greater synergy occurred at lower levels of sedation.

Giovannitti et al, Anesthesia Progress 62:31-38 2015

Precedex inj.: drug label package insert. 2021

α-2A - "Starter Doses"

*suitable "at home" pre op healthy adult doses

Clonidine tabs		Dexmedetomidine
PO OR SL	Dynamics	IV
1	Alpha-1 receptor affinity	1
220	Alpha-2 receptor affinity	1620
	Kinetics	
—	Distribution Half Life ($T_{1/2\alpha}$)	6 min
12-16 hr	Elimination Half Life ($T_{1/2\beta}$)	2 hr
60-90 min	Peak Serum Level (PO)	—
?	Duration (PO)	—
75-95%	Oral Bioavailabilty	—
0.1*, 0.2*, 0.3, 0.4mg	PO Dosage (for sedation)	—
—	IV Increments (slow push!)	5-10 mcg/min
$0.17 per 0.1mg	$$ per Increment	$0.25-0.50

DMET Dosing Guidance

Label: Initiation of Adult procedural Sedation

1 mcg/kg infused over 10 min

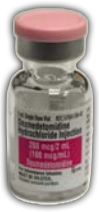
Then, wait 5m for full effect!

Shufflebarger: initial MOD technique

0.25 mcg/kg infused over 5-10 min

Label: Maintenance of Adult procedural Sedation

0.2 mcg/kg/hr - 1 mcg/kg/hr.



Generally start with 0.6 mcg/kg/hr and adjust ↑ or ↓ prn

Over age 65?

Initiate with 1/2 normal dose and reduce maintenance doses as well.

Hepatic Impaired adults and elderly

a dose reduction should be considered

Initiation or Maintainance?

Precedex inj.: drug label package insert. 2021

DMET Dosing and Administration

Dosage adjustments

(DMET) dosing should be individualized.

Precedex inj.: drug label package insert. 2021

Titrated prn

(DMET) dosing should be titrated to desired clinical response.

Precedex inj.: drug label package insert. 2021

Slow, methodical pace

(DMET) should be administered (via) a controlled infusion

Precedex inj.: drug label package insert. 2021

Dilute all brands DMET for IV use

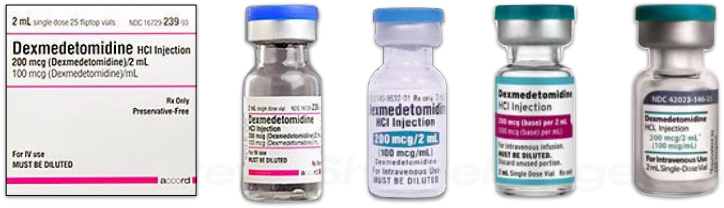
Hospira received approval to make and sell Precedex in 1999. Multiple patents have successively expired since. In 2014 the first approvals to make generic DMET were awarded to 2 companies.

while still available as branded "precedex" 20 manufacturers are now able to manufacture DMET

Precedex

sold in boxes of 25 SDVs

The price is dramatically lower for generics

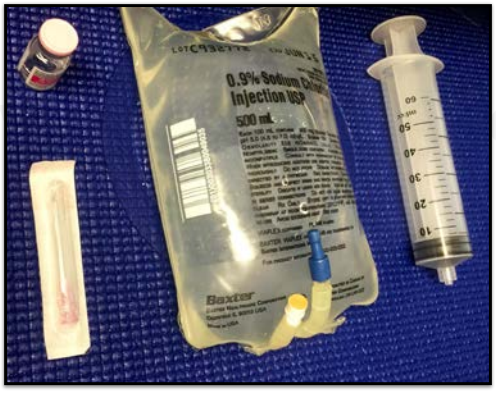


Supplied as 200mcg/2mL (100mcg/mL) SDV (far too concentrated a to push 0.25 mcg/kg slowly)

DMET dilution for Bolus

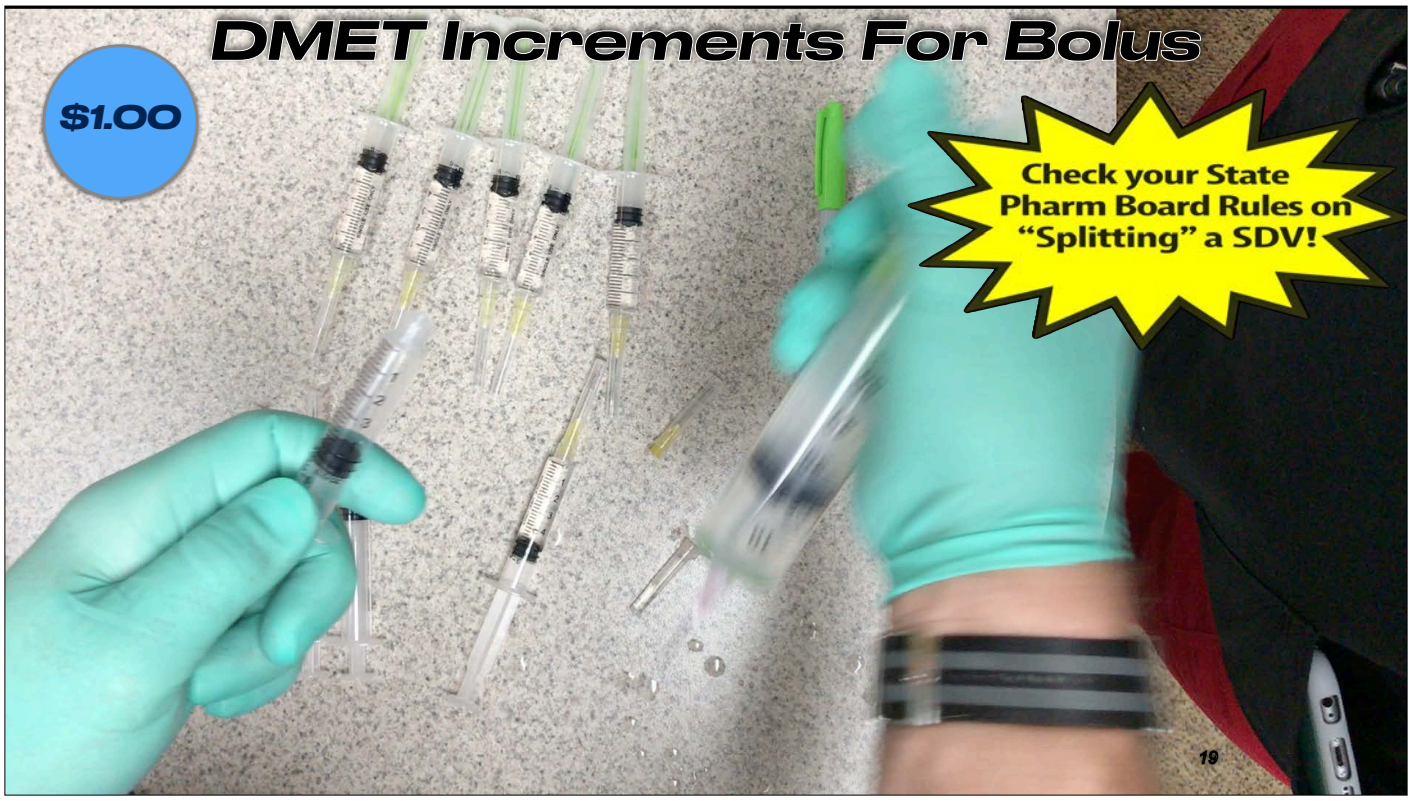
Typically supplied as 200mcg/2mL SDV

Must be diluted with 0.9% NaCl to 4mcg/mL



can be used for Drip or increments

To prepare the infusion, add 2 ml of dexmedetomidine to 48 ml of 0.9% sodium chloride injection to a total volume of 50 ml. Shake gently to mix. The final dilution will then contain 4 mcg/ml.



diluted DMET Stability

48 hr stability (at room temp in daylight) after diluted with 0.9% NaCl to 4 mcg/mL

One study evaluated the potential adsorption and stability of dexmedetomidine hydrochloride diluted to 4 mcg/ml with 0.9% sodium chloride and then stored in various syringes, detailed in the table below.[4] The syringes were stored in daylight at ambient room temperature. Samples were obtained from each syringe at baseline, 1 hour, 4 hours, 8 hours, 24 hours, and 48 hours, and the concentration of dexmedetomidine base was determined via the HPLC method.

Description of Polypropylene (PP) Syringes Tested for Adsorption/Stability*

Manufacturer	Volume	Ref. No.	Barrel Material	Plunger-end Composition
Becton-Dickinson (B-D) Luer Lok	60 ml	309663	PP	Neoprene rubber
Becton-Dickinson (B-D) Plastipak	50 ml	8090114	PP	Neoprene rubber
IVAC Medical Systems	50 ml	30602N	PP	Medical grade rubber, siliconized coating
Braun Perfusor	50 ml	87228810	PP	Natural rubber

*Ref. No. indicates reference number; PP, polypropylene.

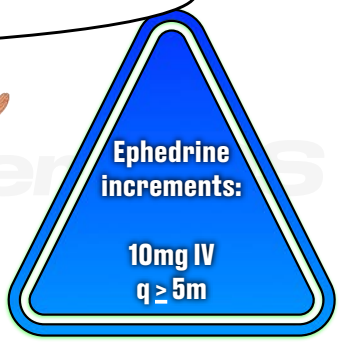
The results obtained indicated that there was no significant adsorption/instability of dexmedetomidine (from a dexmedetomidine 4 mcg/ml base solution) onto any of the syringes tested when stored for up to 48 hours at ambient room temperature.

48 hr sterility after diluted with 0.9% NaCl to 4 mcg/mL??

α-2 Agonists
Adverse / Side effects?

In 2025....it's not just "All about the Airway" anymore

- **Cardiovascular effects**
  - ↓ HR
  - ↓ MAP



Typically, HR and BP remain clinically acceptable

Ogawa, DDS,et al: Anesth Prog. 2008 Fall; 55(3): 82–88.

"Reversal"
multiple α2 Antagonist drugs exist

alpha2-adrenergic receptor Antagonists:
Yohimbine, Atipamezole and Tolazolineare
are used in veterinary medicine as
Reversal Agents for the sedative effects of alpha2-agonists

ANTISEDAN is an α2-Antagonist that competitively inhibits α2-adrenergic receptors

ANTISEDAN (atipamezole) is indicated for the reversal of the sedative and analgesic effects of (DMET) ..and..reduces time the dog is sedated

there is NO specific Alpha-2 antagonist drug is approved for human use

Selectivity of atipamezole, yohimbine and tolazoline for alpha-2 adrenergic receptor subtypes: implications for clinical reversal of alpha-2 adrenergic receptor mediated sedation in sheep.J Vet Pharmacol Ther 1998 Oct;21(5):342-7

α-2 agonists TI

Pediatric patient accidentally .. overdosed (with) Dexmedetomidine (DMET):

> 60-120X Pace ≅9X Dose

1. A Clerical error results in a 100 µg bolus given to an 11 kg 3 yr old
2. This case occurred in the critical care setting while treating pyogenic meningitis, not during sedation/anesthesia.

Hemodynamic instability noted. HR 65, BP 70/40, SpO^2 85%, RR 8-10/m, constricted pupils with normal eye reflex.
Bradypnea, Bradycardia, Hypotension, Deep Hypnosis and Miosis was noted.

He was successfully managed with O^2, NS bolus, and Epi Infusion.

He did not respond to painful stimuli until 3hr and became oriented at 7hr post OD.

Nath SS, et al:. Dexmedetomidine Overdosage: An unusual presentation. Indian J Anaesth. 2013 May;57(3):289-91

DMET Challenges, Strengths, Controversies, and Quirks

Utilization:

Administer DMET Early

titrate "right now drugs" off of the base of DMET

"Shrink" your Incremental Dose of sedatives if a DMET sedative 'base' has been built

Incorporate latency into your workflow

DMET Key Points:

Inexpensive and Safe when used properly

Must attention to Maintaining BP!

biggest downside —> substantially longer induction and recovery

DMET Challenges, Strengths, Controversies, and Quirks

Things to Avoid:

Administering DMET After other "fast onset" Sedatives

Starting Surgery too soon after DMET administration

Administering normal doses/increments of other sedatives when initially adding DMET to your workflow

DMET Key Points:

Excellent Sedative

Requires Patience to use

Must fully comprehend DMET mechanism of Action

Behaves Very differently than conventional Sedation Agents

Questions?

HeyDrSteve@gmail.com

OHIO ANESTHESIA RULES UPDATE - 2026

EFFECTIVE
JANUARY 1, 2024

- Joel M. Weaver DDS, PhD
- Dentist Anesthesiologist Specialist
- Diplomate American Dental Board of Anesthesiology
- Professor Emeritus
- The Ohio State University

DISCLOSURES

- I have nothing to disclose
- I have no conflicts of interest
- I am not a lawyer
- I do not speak for the OSDB
- My interpretations of the rules are my own
- Be certain to review the rules and interpret them for yourself

OHIO STATE DENTAL BOARD

- Regularly reviews & revises, if necessary, all rules
- 2019 Anesthesia Rules Revision Committee formed
- Dentist Anesthesiologist
- Oral and Maxillofacial Surgeon
- Pediatric Dentist
- Periodontist
- General Practioner Oral Implantologist moderate sedationist
- OSDB staff

DRAFT OF REVISIONS COMPLETED 1/1/2020

- Draft ready for OSDB to review
- Then Covid-19 epidemic cancelled everything
- Eventually open public hearings took place in March 23, 2023
- Eventually passed Joint Committee on Agency Rule Review (JCARR)

JOINT COMITTEE ON AGENCY RULE REVIEW (JCARR)

- 5 Stete Representatives and 5 State Senators
- Can recommend to invalidate part or all of a rule if :
- Rule exceeds statutory authority
- Rule conflicts with legislative intent or current/proposed rule
- Rule more strict / burdensome than federal law or rule requires
- Rule agency fails to provide accurate summary or

Rule agency fails to provide fiscal/business impact analysis

MAJOR CHANGES IN NEW OHIO ANESTHESIA RULES

- Replaced inaccurate wording (e.g. ACGME-accredited residency)
- Modernized definitions (e.g. *moderate sedation*, not conscious sedation)
- ***Requires documentation of medical emergency management drills***
- ***Requires*** current ***BLS-HCP & ACLS*** for permited dentist
- ***Requires BLS-HCP*** for the person ***assisting*** the permitted dentist

MORE MAJOR CHANGES

- Children are high sedation risks except for nitrous oxide-oxygen minimal sedation
- Uncooperative behavior can lead to oversedation
- Oxygen reserve less than adults
- Hypoxemia occurs quickly– tachycardia--bradycardia -- cardiac arrest
- Thus, **pediatric endorsement** now required for DS-GA (age < 8) & Mod Sed (< 13)
- Now consistent with updated OMFS pediatric anes training CODA requirement :
- ***"The graduating resident must be trained to competence in the delivery of***
- ***GA-DS to patients of at least 8 years of age and older"***
- {age <8 yrs = "training" (not competence) in Behav. Management, N2O, Sed, GA}

CLARIFY REPORTS OF ADVERSE OCCURENCES

- ***All Ohio dentists*** must notify OSDB within 72 hrs hrs of ***knowledge*** of an adverse occurrence and submit a complete written report within 30 days of any adverse occurence requiring ***hospital admission*** within 24 hrs of treatment or any ***mortality*** which occurred as a direct result of treatment in an out-patient dental facility.
- NOT JUST ANESTHESIA RELATED, … ANY EVENT RELATED TO DENTAL TREATMENT
- Failure to comply when mortality or adverse occurrence is related to moderate sedation, deep sedation, or general anesthesia may result in restriction, suspension, or revocation of permits and/or other disciplinary action.

MINIMAL SEDATION

- A minimally depressed level of consciousness
- Retains pt's ability to independently & continuously maintain open airway
- Respond normally to tactile stimulation and verbal command
- Cognitive function and coordination may be modestly impaired
- Ventilatory and cardiovascular functions are unaffected.

MODERATE SEDATION

- Pts respond purposefully to verbal commands, either alone or by light tactile stim
- No interventions are required to maintain a patent airway
- Spontaneous ventilation is adequate.
- Cardiovascular function is usually maintained.
- Drugs and/or techniques should carry a margin of safety wide enough to render unintended loss of consciousness unlikely.

DEEP SEDATION

- Drug-induced depression of consciousness where pts cannot easily be aroused
- Respond purposefully following repeated or painful stimulation.
- Ability to independently maintain ventilatory function may be impaired.
- Patients may require assistance in maintaining a patent airway
- Spontaneous ventilation may be inadequate.
- Cardiovascular function is usually maintained

GENERAL ANESTHESIA

- Drug-induced loss of consciousness where pts not arousable even by painful stimulation
- The ability to independently maintain ventilatory function is often impaired.
- Patients often require assistance in maintaining a patent airway
- May need positive pressure vent due to depressed spon vent/neuromuscular function
- Cardiovascular function may be impaired.

ANY OHIO DENTIST CAN ADMINISTER MINIMAL SEDATION FOR THOSE 13 YRS AND OLDER -- NO PERMIT REQUIRED WITHIN THE FOLLOWING RULES:

- Patients ***thirteen years*** of age or older may receive enteral minimal sedation, limited to a single dose of a single drug at not more than the maximum recommended dose on the FDA-approved labeling indicated for unmonitored home use, with or without nitrous oxide-oxygen minimal sedation and local anesthesia
- Patients of ***any age*** may receive nitrous oxide-oxygen inhalation for minimal sedation

EXAMPLES OF MINIMAL ORAL SEDATION BASED ON FDA-APPROVED LABELING

- Halcion (Triazolam) "A dose of 0.5 mg should not be exceeded."
- "Geriatric and/or debilitated patients…A dose of 0.25 mg should not be exceeded"

- Ambien (Zolpidem) " The total dose should not exceed 10 mg daily"
- "Elderly or debilitated patients… recommended dose… is 5 mg daily"

- Xanax (Alprazolam) " Treatment of patients with anxiety should be… 0.25 to 0.5 mg…"
- Elderly/debilitated patients…"the usual starting dose is 0.25 mg…"

COMPREHENSIVE MODERATE SEDATION TRAINING

Predoctoral or advanced dental education program accredited by the CODA

in moderate sedation commensurate with ADA 2016 "Guidelines

for Teaching Pain Control and Sedation to Dentists and Dental Students"

or

C.E. course approved by ADA or AGD C.E. provider in moderate sedation and commensurate with ADA 2016 "Guidelines for Teaching Pain Control and Sedation to Dentists and Dental Students"

Course must assess and document competency in all aspects of moderate sedation

DRUGS REQUIRING G.A. TRAINING & G.A. PERMIT

- Drugs with a narrow margin for maintaining consciousness including, but not limited to
- potent volatile inhalation anesthetic agents,
- ultra-short acting barbiturates (methohexital (Brevital)
- propofol
- ketamine, and
- similarly acting drugs, or quantity of agent(s), or techniques, or any combination thereof that would likely render a patient deeply sedated, generally anesthetized
- Precedex (dexmedetomidine) is NOT specifically included but is debatable

WHO CAN PROVIDE MINIMAL OR MODERATE SEDATION TO CHILD YOUNGER THAN 13 YEARS OTHER THAN NITROUS OXIDE-OXYGEN MINIMAL SEDATION ?

- A dentist with a moderate sedation permit with a pediatric endorsement, or
- Provisional moderate sedation privileges with a pediatric endorsement, or
- A general anesthesia permit, or
- Provisional general anesthesia privileges.

- *"The moderate sedation provider must be prepared to manage a level of anesthesia deeper than intended as it is not always possible to predict how a given patient will respond to anesthesia."*

PEDIATRIC MODERATE SEDATION TRAINING

- Adv. dental edu. program in pediatric dentistry accredited by the CODA

or

- Equivalent C.E. program in pediatric moderate sedation for children <13

or

General Anesthesia permitted dentist

C.E. PEDIATRIC MOD. SED. TRAINING REQUIREMENTS

- Includes: 60 hours didactic instruction in ped mod sedation
- ***Written*** documentation of ***competency*** in ped mod sedation
- 25 mod. ped. sed. by any route with non-inhalation sedative(s) (with or without N2O)
- Only one course participant earns credit for each patient sedation
- Individual patient sedated no more than once / day
- 25 additional sed experiences by individual or group participation or with human sedation/anesthesia simulation or a combination and
- 4 weeks of hosp anes rotation includes preop eval, risk mangemement, pharm, venipuncture, adv airway placement, monitoring, recognition, emerg manage

Written certification by the program director of ***competency*** in:

Rescuing ped pts from level deeper than intended and drug reversal

BIENNIAL MODERATE SEDATION PERMIT RENEWAL

- No renewal fee
- Current BLS-HCP
- Current ACLS (PALS if moderate sedation for peds)
- Minimum of 6 hrs C.E. in prevention & management of sed emerg
- ***NOTE : BLS HCP plus both ACLS and 6 hrs C.E.***

PERMIT RENEWAL= DOCUMENT ALL EMERGENCY DRILLS

- Document quarterly emerg drills, names, drills
- Do all drills within the year, every year
- (a) Hypotensive, hypertensive, bradycardic and tachycardic emergencies;
- (b) Loss ETCO2 tracing > airway manage & cause, e.g. overdose, secretions,
- (i) Hypoventilation progressing to respiratory arrest;
- (ii) Soft tissue or foreign body obstruction of the airway;
- (iii) Laryngospasm; and
- (iv) Bronchospasm;

PERMIT RENEWAL= DOCUMENT ALL EMERGENCY DRILLS

- (c) Unexpected decline consciousness level > over-sedation, stroke, seizure, hypoxia,
- street drug use, anaphylaxis, etc.
- (d) Recognition and management of chest pain progressing to cardiac arrest;
- (e) In offices where mod sed less than quarterly or the first time, do all scenarios
- immediately preceding the adm of moderate sedation for an actual patient
-
- Have reviewed laws and rules governing administration of moderate sedation
- Have verified all licensed/registered mod sed personnel are current/active

MOD. SED. PERMIT RENEWAL FOR C.E.-TRAINED DENTISTS

- (G) Licensed dentists who did not complete a comprehensive predoctoral or advanced dental education program accredited by the U.S. department of education as defined in …this rule who hold a current permit and have been using or employing sedation prior to adoption of this rule and who desire to continue to use or employ sedation, *shall within one year of the effective date of this rule* submit an attestation to the board along with evidence demonstrating competency of successful administration of sedation to a minimum of twenty patients in the three years preceding attestation. Evidence of competency shall be commensurate with each intended patient age range whether ages <13 (20 cases) or ages 13+ (20 cases) or both (40 cases).

GENERAL ANESTHESIA PERMIT HOLDERS

- Do not need to obtain a Moderate Sedation Permit

EMPLOYING OR USING A CRNA

- Only sedation-trained-permitted Ohio dentist can ***supervise*** CRNA for Mod Sed
- CRNA can only provide sedation depth that dentist is permitted to administer
- CRNA can only administer drugs that supervising dentist can personally administer
- CRNA must meet std of sedation care that the dentist would have to meet

FOR MODERATE SEDATION IN ANOTHER OFFICE ?

- Permit holder must notify OSDB within 10 business days of new office where moderate sedation is provided

MODERATE SEDATION TEAM

- During moderate sedation, the following TWO persons must be physically present in the room and caring exclusively for the patient:
- A moderate sedation provider; and
- A person currently certified in BLS-HCP
- Assists moderate sedation provider
- Shares the patient monitoring and documentation duties
- Assists moderate sedation provider in an emergency

RECOVERY FROM MODERATE SEDATION

- Moderate sedation provider must remain at dental facility until patient d/c
- Assuming patient continues to respond appropriately to verbal command after the procedure has ended, further recovery until ready for d/c must be monitored by a person described in rule as BLS-HCP- monitoring- assistant

EMPLOYING A MODERATE SEDATION PROVIDER

- Dentist who employs or works with moderate sedation provider must:
- (1) Ensure that anesthesia provider meets requirements defined in rules
- (2) Ensure that facility meets requirements set forth in this rule
- (3) Be current BLS-HCP and able to assist mod sed provider in emergency
- (Interpretation: Permitted dentist must be in the room even if delegating operative procedure to another provider)

DEEP SEDATION/GENERAL ANESTHESIA TRAINING

- An advanced dental education CODA-accredited program
- which affords appropriate training to competently administer DS/GA
- or

Grandfathered training commensurate with ADA "Guidelines for Teaching the Comprehensive Control of Pain and Anxiety at the Advanced Education Level (Part 2)" in effect at the start of their training prior to CODA accreditation of dental anesthesia residencies (2008) (one year training prior to July 1, 1993, and two years thereafter)

Board shall issue applicant dentist provisional GA privileges valid for up to one year pending successful completion of the clinical onsite evaluation

DEEP SEDATION/GENERAL ANESTHESIA INSPECTION FOR MOBILE ANESTHESIA PROVIDER

- For a mobile or portable facility, one inspection of that “mobile facility” shall be conducted in office of an Ohio dentist.
- Must provide written list to OSDB of monitors, emergency equipment, and other materials the mobile anesthesia provider agrees to have available at all times.

BIENNIAL GA PERMIT RENEWAL

- OSDB renews permit (N/C) biennially at time of dental license renewal
- Permit holder attests to the OSDB that the permit holder:
- (1) Maintains successful completion of ***BLS-HCP and ACLS*** (and PALS <age 8)
- (2***) 6 hr OSDB-approved CE*** on manage/prevention DS/GA emergencies
- (3) Has performed emergency drills at least quarterly during the biennium, ***documenting in log*** the date, nature of simulated emergencies and names & roles of all participants

LOG OF SIMULATED EMERGENCIES

 # 1 Recognize & manage chest pain progressing to cardiac arrest

 # 2 Hypotensive, hypertensive, bradycardic and tachycardic emergencies

 # 3 Recognize and manage loss of capnograph tracing requiring appropriate management of airway and of underlying cause, e.g. anesthetic overdose, secretions, etc. due to:

- (i) Hypoventilation that progresses to respiratory arrest;
- (ii) Soft tissue or foreign body obstruction of the airway;
- (iii) Laryngospasm; and
- (iv) Bronchospasm

 # 4 Unexpected decline in level of consciousness & consider multiple possible etiologies, e.g. oversedation, stroke, street drug use, hypoxia, anaphylaxis, etc

LOG OF SIMULATED EMERGENCIES

- Offices where DS/GA is performed less than quarterly, or for the first time:
- Do emergency drills for all above scenarios immediately preceding DS/GA for an actual patient
- ALSO, ATTESTATION THAT RENEWAL PERMIT HOLDER :
- Has reviewed DS/GA laws and rules
- Has verified all licensed/registered personnel involved in the administration of DS/GA maintain current, active, licensure or registration.

SUPERVISION OF CRNA

- Dentist may supervise licensed CRNA for any anesthetic procedures for which the dentist is qualified by permit.
- The permitted dentist must provide direct, personal, on-site supervision of the CRNA throughout the entire anesthesia time

NOTIFICATION TO OSDB OF NEW GA LOCATION

- Must notify OSDB within 10 days of new GA office location

EMPLOYING/USING DS/GA PROVIDER

- Dentist employing/using a DS/GA provider must:
- (1) Ensure GA provider meets the requirements defined in the rules
- (2) Ensure that the facility meets the requirements defined in the rules
- (3) Be currently certified in BLS-HCP
- (4) Able to assist GA provider in an emergency if needed.

DEEP SEDATION/GENERAL ANESTHESIA TEAM

- DS/GA for a pt 8 yrs or over, 3 persons must be physically present in room
- All must be caring exclusively for pt
- (1) A general anesthesia provider
- (2) Either of the following:
- (a) Ohio-licensed dentist with current BLS-HCP or ACLS who can assist the anes provider in an emergency if needed; or
- (b) Person with current BLS-HCP, experienced in pt monitoring and documentation & solely dedicated to pt monitoring and documentation and, if needed, assisting GA provider in an emergency; and
- (3) One individual whose duties may include assisting with dental procedures

DS/GA TEAM FOR CHILDREN UNDER AGE 8

- **3 persons must be physically in room & exclusively caring for pt.**
- ***(1)*** Either (a) GA provider with current PALS who exclusively administers and monitors the anesthetic and is not otherwise involved with the dental procedure OR (b) GA provider with current PALS who is involved in dental procedure and who maintains log of performing DS/GA for a minimum of 20 cases / year or a total of 40 cases in children <8 yrs during preceding twenty-four months
- ***(2)*** Either (a) Ohio-licensed dentist with current PALS able to provide emergency assistance to GA provider if needed; or (b) Person with current BLS-HCP, experienced in pt monitoring and documentation & solely dedicated to pt monitoring and documentation and, if needed, assisting GA provider in an emergency and
- ***(3)*** One individual whose duties may include assisting with dental procedures.

POST-ANESTHETIC MONITORING

- Once pt regains consciousness & responds appropriately to light tactile stimulation or verbal command, patient's recovery must be monitored by an individual experienced in patient monitoring and documentation and is certified in BLS-HCP.

SUPERVISION OF RECOVERY FROM DS/GA

- GA provider must remain in dental facility until pt regains consciousness & D/C
- When conscious and responding appropriately to light tactile stimulation or verbal command, further recovery until ready for discharge must be monitored by BLS-HCP monitoring person able to assist in emergency as described in rules.

ADVERSE OCCURRENCES REPORTING TO OSDB

- Notify OSDB within 72 hrs of ***knowledge*** of M&M >hosp admit within 24 hrs of tx
- if event is a direct result of treatment in an outpatient dental facility
- Submit a complete written report within 30 days
- NOT JUST ANESTHESIA RELATED, … ANY EVENT RELATED TO DENTAL TREATMENT
- Failure to comply when mortality or adverse occuence is related to moderate sedation, deep sedation, or general anesthesia may result in restriction, suspension, or revocation of permits and/or other disciplinary action.

NEW RULES ARE A STEP FORWARD TO IMPROVING THE MARGIN OF SAFETY FOR ANESTHESIA IN DENTAL OFFICES

ASDA-AAOMS-AAP MODEL STATE SEDATION/GENERAL ANESTHESIA RULES PUBLISHED IN 2023

Ohio rules similar to ASDA-AAOMS-AAP Model Sedation/General Anesthesia Rules

Ohio Rules do not require an office permit to use a medical anesthesiologist

A major benefit of anesthesia specialty recognition

American Society of Dentist Anesthesiologists (ASDA) spearheaded this document

Endorsed by ASDA, AAOMS and American Academy of Periodontology

AAPD relies on their sed document with American Academy of Pediatrics (peds MDs) which require the anesthesia provider to not be involved in the operation

THANK YOU FOR YOUR ATTENTION