AEMP Newsletter Summer 2026

Academy of Emergency Medicine Pharmacists

President's Message

There's a strange kind of quiet that settles over a residency program in late June. The schedule's still fairly full, the pager still goes off, but something has noticeably changed. The residents who spent the last year(s) learning to handle codes, critique evidence, and hold their ground in a resuscitation bay are about to become the specialists other people call. It happens in a matter of days. And a new class is about to walk in not even knowing where the bathrooms are yet.

As an RPD, I think about that transition a lot. This one felt a bit deeper, maybe because I spent this past year watching AEMP go through its own version of the same thing.

After transitioning from an interest group to SAEM's 9th academy in 2025, AEMP achieved tremendous growth over the past year. SAEM26 drew nearly 4,000 attendees this year, its largest turnout ever, and AEMP's own programming ran as an expanded two-day track within the larger four-day conference, bringing together more than 165 EM pharmacists, residents, and students. It was great to see so many of you at the networking events, cheering on the Ketameanies dodgeball team, and supporting your colleagues. The educational lineup featured a keynote panel of legacy EM pharmacists, year-in-review highlights, pro-con debates, innovative practice ideas, and PGY2 EM residents delivering auto-advancing IGNITE! presentations, along with a jam-packed research session. Somewhere in the middle of it all, a torrential downpour caused the ceiling to start hemorrhaging water during a session while the speakers kept going without missing a line. Someone just walked over, dropped a trash can under the leak, and sat back down. If that doesn't sum up an EM conference, I'm not sure what does. We'd love to see you next year, so be sure to mark your calendars for **May 18-21, 2027,** because **AEMP is heading to San Francisco.**

This was also the first year we held formal academy elections for the Executive Committee, which is now more streamlined, down from 14 to 7 members. Two of those seven are brand new roles. Alli and Anne will serve as our inaugural Members at Large, acting as the bridge between our committees and the EC while strengthening our ties to our members and SAEM's broader organizational structure. Alongside this, we welcomed a new group of Committee Chairs transitioning in from their roles as chair-elect to keep AEMP's momentum moving forward. It's a smaller table, but an inherently more connected one, built by people stepping into new roles for the first time. That's the same transition happening right now in EM residency programs across the country.

A few words to the graduating PGY2 EM residency class of 2026:

You're going to feel like an imposter for a while. That's normal. You were a resident just a few months ago, so you don't owe yourself total confidence on day one as an EM clinician. Put the pressure down. Spend this early stretch building real relationships with your team, and lean on your coworkers more than you think you're allowed to. You're going to spend the rest of your career getting better at your craft, and there's no version of this job where you arrive and stop learning.

You may also notice the pace feels slower, even a little boring, compared to residency, where you juggled a million things by design. Let it be boring for a while. Don't go looking for more to carry just because you're used to carrying a lot. The opportunities will find you again sooner than you'd guess. Appreciate this quieter window instead of rushing past it.

To the new class walking in this July:

It's going to be a lot, and some days it'll feel like too much. Stay curious anyway. Ask the question even when you're worried it sounds obvious; the people around you asked it too, not that long ago. Get comfortable stepping outside what feels safe, because that's where most of the real learning happens. Recognize the small wins: the RSI plan you recommend without having to look up dosing, the feedback you implement successfully, and tightening up your patient workups. Those count.

And get comfortable with things not going according to plan. Emergency medicine does not run on schedule. Ceilings leak mid-session. Conference rooms fill past capacity. This job is about adapting well, not following a plan to the letter.

AEMP has been adapting too, in its own way.

We're continuing to push for a real seat at the table. That means moving closer to having more EM pharmacist authors on guidelines, more of us on EM journal editorial boards, and increased collaboration with the organizations already shaping this space. We also want AEMP to be a place where members feel free to just try something. Some of our best ideas have come from someone who simply decided to act on one. We're investing in the education and research infrastructure that turns those ideas into lasting work, expanding student involvement through RxSTAT, deepening resident and fellow engagement in our committees, and sharpening our professional development resources along the way. Lastly, we're creating new ways to recognize and award more of our own members and leaders.

If any of this sounds appealing, great. Engage other members on the new ConnectED listserv, sign up for one of the Education Committee's webinars, be a mentee or mentor, or join a committee. You don't need to run a committee your first year to improve AEMP. Start with one thing!

I've said this before, but I'll scream it from the rooftops again here: this organization is for everyone. "Academic" may be in our name, but AEMP is built on pharmacists from every background and setting, including community hospitals, academic centers, students, new practitioners, and seasoned clinicians alike. If you care about EM pharmacy, you have a home here.

That's true for the residents starting this month. It's just as true for an organization figuring out its next chapter with a smaller EC and new people at the table.

Thank you for being part of this, through the leaks, the growth, and everything in between. It's a privilege to follow Megan as President, and I look forward to working with you all over the next year.

Sincerely,

Kyle DeWitt, PharmD, BCEMP
President, Academy of Emergency Medicine Pharmacists

AEMP Leadership

Executive Committee

President: Kyle DeWitt (kylemdewitt@gmail.com)
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Juana Rabulinski (SAEM governance)
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Marquita Norman, MD (SAEM Board Liaison)

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Chair Elect: Christina Tang (ctang3@northwell.edu)

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Faculty Liasions: Kevin Mercer (kevin.mercer@ymail.com), Deepika Sivakumar (dsivakumar@mgh.harvard.edu)

Committee Updates

Treasurer's Report

Contact: Evan Zahn (evan.d.zahn@gmail.com)

I am excited to be taking on the role of Treasurer for the AEMP Executive Committee and to share updates on our financial status to increase engagement and awareness among our members.

Now that AEMP is an academy, our 2026 budget is $10,000, with an additional, one-time $10,000 continuing education (CE) allocation from the SAEM Board of Directors, for a total of $20,000. We continue to explore opportunities to use these funds in ways that provide the greatest benefit to our members.

Recent and upcoming expenses include CE programming related to AEMP/SAEM26, a networking event at AEMP/SAEM26, and an ENLS certification opportunity for members. After these and several other minor expenses are finalized, we anticipate having approximately $8,000 remaining to support CE initiatives and other member-focused activities for the remainder of 2026, so please reach out if you have any proposals that would benefit a large proportion of AEMP members!

Communication Committee Updates

Chair: Alyssa McQueen (Alyssa.Mcqueen@uchealth.com)

Open Leadership Positions!

Charge 2 (Pulse Article): 1 Junior Lead Position & 1 Trainee Lead Position

Charge 3 (AEMP Newsletter): 2 Junior Lead Positions & 1 Trainee Lead Position

Get Involved!

Email Alyssa to volunteer to make infographics, author Pulse articles, journal club reviews, and more!

Scan the QR code to volunteer to help with a newsletter!

Follow us on social media!
IG & X: @SAEM_AEMP
Bluesky: @saemaemp.bsky.social

Join us on Linkedin:
Academcy of Emergency Medicine Pharmacists (AEMP) - Society for Academic Emergency Medicine

Career Development Committee Updates

Chair: Kyle Schuchter (kyle.schuchter@osumc.edu)

If you are interested in signing up as a mentee through the AEMP Mentorship Program, please use the link HERE. If you have any questions about the AEMP Mentorship Program, please email Kyle Schuchter (kyle.schuchter@osumc.edu) and Elise Metts (elise.metts@uky.edu).

We will soon begin planning our next Career Development Committee webinar for the Fall. If you have any suggestions on topics you would like to see, please email Kyle Schuchter (kyle.schuchter@osumc.edu) and Iram Nasreen (iram.nasreen@ynhh.org).

We are looking for individuals to help with various existing charges, including the following: mentorship program, career development education/webinar coordination, preceptor/RPD resources, and career paths in EM. We also have a robust list of exciting potential new career development focused charges for the upcoming year we are looking for assistance with. If interested in helping with any of the existing charges above or exploring potential new charges, please reach out to Kyle Schuchter (kyle.schuchter@osumc.edu).

JOURNAL CLUB CORNER

A Comparison of High Dose Versus Low Dose Intranasal Midazolam for Sedation in the Pediatric Emergency Department

Written by: Cecilia Schowe, PharmD, MS, BCPS, BCEMP
Edited By: Nicholas Pugliese, PharmD, BCCCP

Abbreviations: INM: Intranasal midazolam; PED: Pediatric emergency department; GABA: γ-aminobutyric acid

Background[1,2]

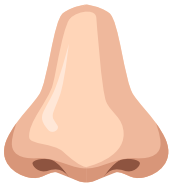
- Minimal sedation is commonly utilized in the PED for minor procedures. Sedation can help immobilize patients without the need for restraints decreasing stress patients and healthcare providers
- Midazolam, a $GABA_A$ positive allosteric modulator, is commonly utilized due to its sedative, anxiolytic, and amnestic effects and its ability to be administered via a mucosal atomizer device
- Current literature recommends a wide dosing range from 0.2 to 0.5 mg/kg.

Yealy et al (1992)[1]	• Retrospective chart review • Pediatrics requiring INM for laceration repair • Lower rates of adequate sedation when utilizing 0.2-0.29 mg/kg (27%) compared to when using 0.3-0.39 mg/kg (80%) or 0.4-0.5 mg/kg (100%) with no increased risk of side effects
Tsze et al (2025)[2]	• Prospective, randomized, open-label, single-center trial • Pediatrics requiring INM for laceration repair • Higher rates of sedation utilizing the Pediatric Sedation State Scale were found with the 0.4 and 0.5 mg/kg dosing

- Given the wide dosing range in literature from 0.2 to 0.5 mg/kg without clear recommendation, the aim of this study was to evaluate the impact of low versus high dose INM on the need for additional rescue sedation.

Study Design[3]

- Single center, retrospective cohort analysis
- Patients were identified using documented administration of INM in the pediatric emergency department from August 2022 to August 2023
- Dosing:
  - Patients were grouped according to low dose vs high dose with max 10 mg
    - Low group defined as 0.2 mg/kg with 10% rounding (0.18-0.22 mg/kg)
    - High dose group defined as 0.5 mg/kg with 10% rounding (0.45-0.55 mg/kg)
  - There was no formal protocol for dose selection and was at the discretion of the physician



Inclusion Criteria	Exclusion Criteria
Age < 18 years old ≤ 22 kg Located in the PED Determined by physician to be appropriate candidates for minimal sedation by INM*	Documentation of INM for seizures or behavioral agitation Wards of the state, pregnant, or in police custody Admission to the hospital Receipt of sedation medication (opioids, low dose ketamine) 2 hours prior or within 5 minutes of INM administration

*Defined as a drug-induced state in which patients respond normally to verbal commands and may have impaired cognition and coordination, but maintains unaffected ventilatory and cardiovascular function, with only 1 medication administered

Outcomes[5]

- Primary Outcome:
  - Composite measure of additional sedation medication administration following the initial INM dose; definition as any additional dose between 5-15 min, 15-45 min, or physician documentation of sedation failure
- Secondary Outcomes:
  - PED length of stay
  - Time from PED presentation to initial INM dose
  - Time from initial INM dose to PED discharge
  - Any documented adverse drug reaction

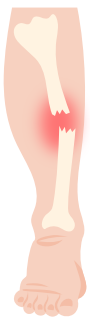
Statistical Analysis[3]

- Categorical data was compared using Chi squared, Fisher's Extract Test, and univariate logistic regression
- Continuous data was compared using independent t-test and Mann Whitney U
- Statistical tests were interpreted using two-sided alpha of 0.05 and considered significant if the $p<0.05$

Results[3]

Study Sample

- Enrollment
  - 336 patients identified (most common exclusion criteria: weight >22 kg (n=99), dose outside of defined group (n=50))
  - 161 (47.8%) patients included



- Baseline Characteristics

Baseline Characteristic	Low dose (n=25)	High dose (n=136)	*P* value
Age, month	33.2 ± 22.22	37.7 ± 20.1	0.43
Weight, kg	13.8 ± 4.3	14.6 ± 3.9	0.39
Male, n (%)	15 (60)	77 (56.5)	0.75
Initial INM dose, mg	2.8 ± 0.8	7.2 ± 1.9	<0.0001
Initial INM dose, mg/kg	0.2 ± 0.01	0.5 ± 0.02	<0.001
Analgesics given in PED, n (%)	5 (20)	36 (26.5)	0.5
Procedure, n (%)			
Imaging	4 (16)	26 (19.1)	0.83
Lac Repair	13 (52)	82 (60.3)	0.71
Other*	8 (32)	28 (20.3)	0.44
Time to repeat dose (≤ 45 mins)	26.2 ± 5.4	23.3 ± 10.8	0.58

*Bladder catheterization, IV-line placement, fracture/dislocation, foreign body removal, incision/drainage, nasogastric tube placement, visualization of the eye, or bladder prolapse reduction

- Key Results

Outcome	Low dose (n=25)	High dose (n=136)	*P* value
Primary Composite Outcome, n (%)	7 (28)	10 (7.4)	0.01
5-15 min	0 (0)	2 (1.5)	1
>15-45 min	6 (24)	2 (1.5)	<0.001
Provider-documented sedation failure	3 (12)	6 (4.4)	0.15
Secondary:			
PED length of stay, hour	4.9 ± 2.6	4.8 ± 2.4	0.91
Time from arrival to initial INM, hour	3.1 ± 2.6	2.6 ± 1.7	0.08
Time from initial INM to discharge, hour	3.2 ± 5.0	2.2 ± 1.9	0.62
Reported adverse effects, n (%)	0 (0)	1 (0.7)*	1

*Dystonia and allergic reaction which resolved with IV diphenhydramine

Discussion & Conclusions[3]

- Author's Conclusion
  - Patients receiving high dose INM administered for minimal sedation were less likely to need additional sedatives and did not increase risk for adverse events or longer PED length of stay
- Strengths
  - Exclusion of additional medications before and during procedure (2 hours prior and 5 min post INM) allowing for true assessment of INM effectiveness
  - Results consistent with previous literature suggesting high dose INM provides efficacy without increasing adverse events
- Limitations
  - Small sample size with single center design
  - Utilization of medical records for start time of procedure potentially influencing time interval between INM administration and procedure initiation
  - No data regarding dose or timing or additional analgesics (ie fentanyl) which could affect INM effectiveness
  - Adverse event reporting relies on documentation in the medical chart, potentially leading to under-reporting
  - Unable to identify optimal dose for various individual types of procedures
  - Selection of dose to utilize was up to the discretion of the treating physician and may have introduced bias

Applicability to Practice

- Results of this trial help to solidify the safety and efficacy of high-dose INM in pediatric patients undergoing procedures that require minimal sedation without increasing risk of adverse events. The use of a higher one-time dose allows providers to finish procedures and disposition patients more quickly.

Clots & Chaos: Mastering the Pulmonary Embolism Guideline Updates

Written by: Liz Uttaro, PharmD, BCEMP
Edited by: Judah Brown, PharmD, BCCCP, BCEMP & Lyuba Benin PharmD Candidate

Background[1]

Pulmonary embolism (PE) is an occlusion in the pulmonary vasculature resulting in impaired blood flow and gas exchange distal to the occlusion. PEs usually result from thrombosis in a deep vein of the lower limb, although they can result from other thrombosis within the body. Thrombosis typically occurs due to 3 factors, often referred to as the Virchow triad – venous stasis, hypercoagulability, and endothelial injury. Risk factors of clot formation include presence of vascular devices, trauma, older age, immobilization, cancer, pregnancy, and estrogen containing oral contraceptives.

Patients with PE may present within a range of completely asymptomatic to hemodynamic collapse. Diagnosis remains challenging due to the non-specific symptoms such as chest pain, dyspnea, syncope, and tachycardia. The current estimated incidence of PE is 60-120 cases per 100,000 population per year. The current estimated in-hospital mortality is 14% accompanied by a 90-day mortality of 20%. Prompt diagnosis and treatment is paramount to increase positive patient outcomes.

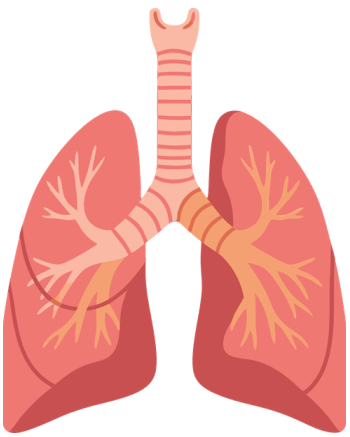
Diagnosis[2] of PE starts with an in-depth history of present illness and use of a clinical probability of PE validated tool such as the Wells score or revised Geneva score. If clinical probability is 15-50%, a D-dimer test and YEARS criteria assessment is recommended. PE diagnosis may be excluded if 0 YEARS criteria are met and D-dimer is <1000 ng/mL as well as if >/=1 YEARS criteria and D-dimer <500 ng/mL. If neither of these criteria exclude PE, it is recommended to perform diagnostic imaging. If the clinical probability of PE is >50%, it is recommended to skip the D-dimer and YEARS criteria and perform diagnostic imaging off the bat. Patients who undergo diagnostic imaging and have a positive computed tomography pulmonary angiography (CTPA) or high probability ventilation/perfusion (V/Q) scan, the diagnosis of PE is confirmed.

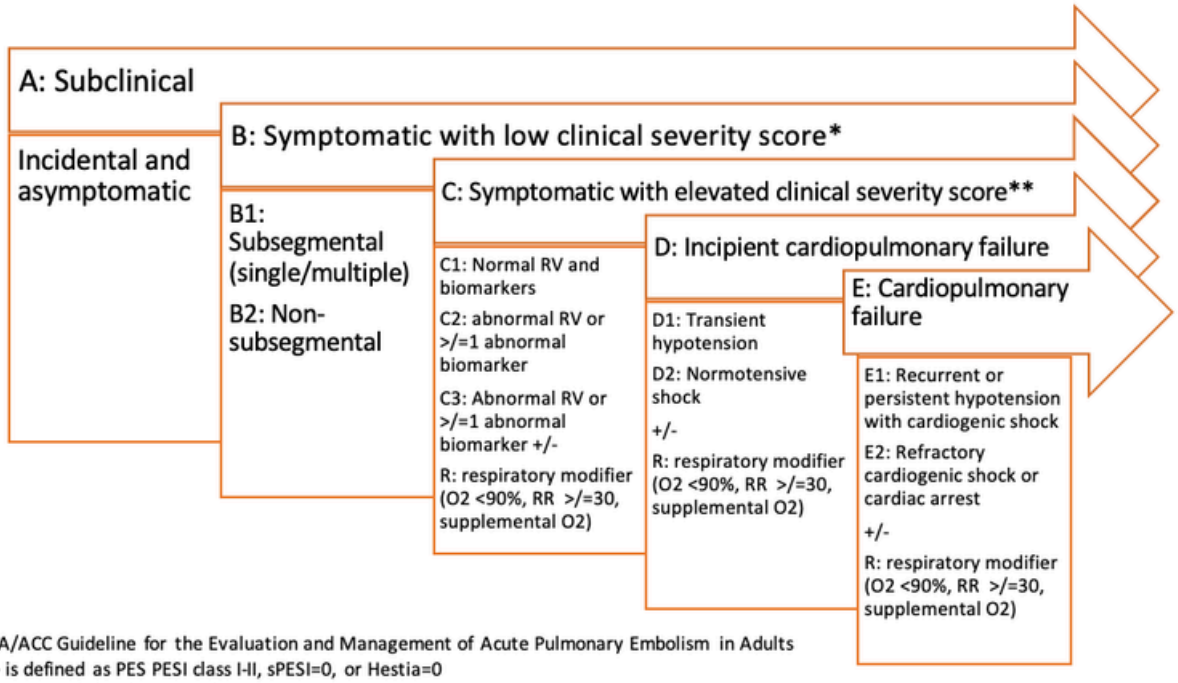
What's New?[2]

In early 2026, the American Heart Association (AHA) and American College of Cardiology (ACC) Joint Committee released the Guideline for the Evaluation and Management of Acute Pulmonary Embolism in Adults. The new guidelines focus on a new risk categorization schema and associated treatments within those categories. The new risk categories provide a framework for assessing likelihood of further decompensation and associated mortality. Most treatment modalities remain the same as previous but with further delineation of agent choice and further category breakdown.

<u>Progression of Acute PE Risk Categorization Schema</u>

- In 2011, the AHA scientific statement regarding PE risk categorization used the risk categories of low risk, submassive, and massive. The definitions of these were based on hemodynamic function as well as right ventricle (RV) function.
- In 2019, the ESC acute pulmonary embolism risk scheme changed the risk categories to low risk, intermediate low-risk, intermediate high-risk, and high risk. The definitions were based on Pulmonary Embolism Severity Index (PESI)/Simplified Pulmonary Embolism Severity Index (sPESI) risk scores, RV function, positive troponins, and hemodynamic instability.
- The 2026 AHA/ACC PE guideline introduces the new Acute PE Clinical Categories A-E. Each category has a broad definition with subcategories within. Patients can transition from one category to another as their clinical course changes through evaluation and treatment.





Adapted from the 2026 AHA/ACC Guideline for the Evaluation and Management of Acute Pulmonary Embolism in Adults
*Low clinical severity score is defined as PES PESI class I-II, sPESI=0, or Hestia=0
**elevated clinical severity score is defined as PESI class III-V, sPESI ≥1, Hestia ≥1

<table>
<tr><th colspan="6">Initial Assessment and Management Using Acute PE Clinical Categories[2]</th></tr>
<tr><th>Category A & B</th><th>Category C</th><th>Category D1</th><th>Category D2</th><th>Category E1</th><th>Category E2</th></tr>
<tr><td>Initiate a direct oral anticoagulant (DOAC) (1)
Examples: apixaban, rivaroxaban</td><td colspan="4">Initiate low molecular weight heparin (LMWH) (1)
Example: enoxaparin</td><td>Initiate LMWH or unfractionated heparin (UFH) (1)</td></tr>
<tr><td rowspan="6">Use Hestia, PESI, and/or sPESI to assess short term risk (1)
Consider using a decision tool to identify suitability for outpatient treatment (2a)</td><td colspan="4">Evaluate RV size and function with CT and/or echo (1)</td><td></td></tr>
<tr><td colspan="5">Measure lactate (1)</td></tr>
<tr><td colspan="5">Multidisciplinary Pulmonary Embolism Response Team (PERT) assessment to guide clinical management (1)</td></tr>
<tr><td rowspan="3">Measure at least 1 cardiac biomarker (1)
Use validated risk scores to identify higher-risk patients (2a)</td><td></td><td>Evaluate for normotensive shock (2a)</td><td></td><td>VA-ECMO (2a)
Systemic thrombolysis if acceptable bleeding risk (2a)</td></tr>
<tr><td colspan="3">Systemic thrombolysis (if acceptable bleeding risk), catheter directed thrombolysis (CDL), or mechanical thrombectomy (MT) in appropriate cases (2b)</td><td></td></tr>
<tr><td></td><td colspan="3">Vasopressor and/or inotropic therapy (1)</td></tr>
</table>

Key Pharmacotherapy Takeaways[2]

- Patients eligible for oral anticoagulation (category A & B), should preferentially receive a DOAC over vitamin K antagonists to prevent recurrent VTE and reduce major bleeding events (class 1 recommendation).
    - A meta-analysis of 65 real world studies evaluating nonvitamin K oral anticoagulants in patients with VTE found rivaroxaban and apixaban were associated with lower major bleeding events and lower risk of recurrent VTE compared to standard of care.[3]
- Patients in categories C1-E1, LMWH is recommended over UFH to reduce recurrent VTE and major bleeding events
    - In 2017, a meta-analysis evaluated the efficacy and safety of LMWH compared to UFH in the initial treatment of those with VTE. This meta-analysis included 29 studies and found LMWH reduced occurrence of major bleeding, recurrent thrombosis, and reduced thrombus size compared to UFH[4]. There was no reported difference in mortality between the two agents concluding LMWH is as efficacious and safer in VTE treatment compared to UFH.
    - LMWH's do not require routine lab monitoring compared to UFH due to predictable pharmacokinetics and is dosed once or twice daily. LMWH is made in prefilled, ready to administer syringes allowing for ease of administration outpatient.
- Patients in cardiogenic shock (categories D2-E2), vasopressors and/or inotropes are recommended to increase cardiac output and improve systemic perfusion
    - Indirect evidence in animal models and humans with a diagnosis of cardiogenic shock, norepinephrine is the vasopressor of choice and will increase systemic vascular resistance (SVR) with modest inotropic effects.
- Patients with acute PE in categories A1-C2, systemic thrombolysis is likely to cause more harm than benefit with increased risk of major bleeding events and intracranial hemorrhage.
    - A meta-analysis combining 16 trials evaluating systemic thrombolysis for acute PE treatment found systemic thrombolysis reduced all-cause mortality, however, had significantly more major bleeding, including intracranial hemorrhage (ICH)[10]. Therefore, patients with lower risk of mortality in risk categories A1-C2, systemic thrombolysis is not recommended.
    - 3 thrombolytic agents are currently FDA approved for treatment of PE – streptokinase, urokinase, and rt-PA (alteplase)
        - A total of 224 patients compiled from 4 randomized controlled trials compared systemic thrombolysis to anticoagulation with heparin alone found systemic thrombolysis improved pulmonary obstruction along with a reduction in RV dilation[5-8].
    - Another meta-analysis evaluating systemic thrombolysis for acute pulmonary embolism causing hemodynamic decompensation found patients receiving systemic thrombolysis had a significant reduction in the combined endpoint of all-cause death and clinical deterioration requiring rescue treatment[9].
    - Tenecteplase is not currently FDA approved for use in PE but has been used off-label and investigated in several clinical trials
        - In 2014, the PEITHO trial compared tenecteplase plus heparin vs. heparin alone in intermediate risk pulmonary embolism patients. Patients had to have RV dysfunction and abnormal biomarkers equating to the new groups C2 and C3. The tenecteplase group did prevent further hemodynamic decompensation in these patients however, significantly increased the risk of major bleeding and hemorrhage[12].
    - Alteplase is the thrombolytic of choice with standard dosing of 100 mg IV over 2 hours
        - There is a scant amount of high-quality evidence regarding optimal dosing in acute PE
        - Emerging evidence is starting to show lower dose alteplase (25-50 mg) may be as efficacious as the standard 100 mg dose and reduces the risk of major bleeding events.
            - A pilot study was conducted in 2023 evaluating the use of low-dose extended infusion alteplase in patients experiencing massive pulmonary embolism. A total of 37 patients were included and received 25 mg of alteplase infused over 6 hours and may have received a repeat dose if instability continued. Patients were found to have significantly decreased mean pulmonary artery systolic pressure and significantly decreased right/left ventricle diameter post-low dose alteplase with no increase in mortality. The authors found low-dose extended infusion alteplase for massive PE is safe and effective in patients with massive PE[11].

Scan me for references!

Appendix A: PESI Score and Interpretation

Predictors	Points
Age (year)	1 point/year
Male sex	+10
Cancer	+30
Heart failure	+10
Chronic lung disease	+10
Pulse ≥110 BPM	+20
SBP <100 mmHg	+30
RR ≥ 30/min	+20
Temp <36°C	+20
Altered mental status	+60
Arterial O2 saturation <90%	+20

PESI Interpretation		
Score	**Class**	**30-day Mortality**
≤65	I	1.1%
66-85	II	3.1%
86-105	III	6.5%
106-125	IV	10.4%
>125	V	24.5%

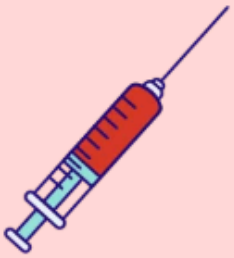
Appendix B:

Dosing Recommendations of Selected Pharmacologic Therapies in Acute PE	
DOACS	
Agent	**Dose**
Apixaban	10 mg by mouth BID x7 days followed by 5 mg by mouth BID
Rivaroxaban	15 mg by mouth BID x21 days followed by 20 mg once daily CrCl <30 mL/min: avoid use
Vitamin K Antagonists	
Agent	**Dose**
Warfarin	Initial 2.5-5 mg by mouth daily with target INR of 2-3 along with enoxaparin bridge until therapeutic
LMWH	
Agent	**Dose**
Enoxaparin	1 mg/kg subcutaneous q12h or 1.5 mg/kg subcutaneous q24h CrCl <30 mL/min: 1 mg/kg subcutaneous q24h
Dalteparin	200 units/kg subcutaneously once daily or 100 units/kg subcutaneously twice daily for 1 month, followed by 150 units/kg subcutaneously once daily CrCl <30 mL/min: avoid use
UFH	
Agent	**Dose**
UFH	80 unit/kg bolus (max 10,000 units) followed by 18 units/kg/hr (max initial infusion rate of 2,000 units/hr)
Systemic Thrombolysis	
Agent	**Dose**
Alteplase	Full dose: 100 mg IV infused over 2 hours Low dose: 0.6 mg/kg (max 50 mg) over 2 hours (consider if >65 years old, high bleeding risk, or weight <55 kg) Cardiac arrest: 50 mg IV bolus over 2 minutes, repeat 50 mg IV bolus after 15 minutes if ROSC still not achieved
Tenecteplase	Weight-based IV push over 5 seconds <60 kg: 30 mg 60-69 kg: 35 mg 70-79 kg: 40 mg 80-89 kg: 45 mg 90 kg or greater: 50 mg
Catheter Directed Thrombolysis	
Agent	**Dose**
Alteplase	Optimal regimen has not been fully defined, may be institution and/or type of catheter dependent Summary of dosing based off trials: 0.5 -2 mg/hr intracatheter continued for 2-15 hours

Academy of Emergency Medicine Pharmacists

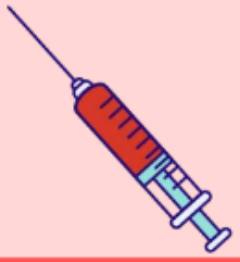
EM Pharmacotherapy Crossword

Created by: Alli Cowett, PharmD, BCEMP



CLUES

AEMP
Academy of Emergency Medicine Pharmacists



Across

[3] I am considered a "dirty" drug because at different dose ranges, I target different receptors

[6] Patients with a heart transplant require dose adjustment for this cardiac medication

[8] Think of me to treat an uncomplicated cystitis in a patient with history of MDRO, a simple one and done option

[10] I am an oral third cousin that if used then you need to check if the patient is on concomitant PPI because my absorption decreases

[11] I am actually protective to the patient if co-ingested in an acetaminophen overdose

[14] I can be administered topically, orally, or intravenously based on the site of bleed

[15] Calculate a shock index in the trauma bay and if ≥ 0.9, I may pose an increased risk of hypotension when used as the sedative during RSI

[16] Combine me with apixaban and I put the patient at a higher risk of bleeding when compared to metoprolol

Down

[1] An overdose of me may mimic brain death

[2] I may be the reason you need to give methylene blue to a patient if they are G6PD deficient

[4] Consider using me as a third line agent for status epilepticus but not in patients with a prolonged PR interval

[5] I may seem harmless because they sell me at gas stations but true dependence can occur

[7] My MOA on GABA receptors is two-fold

[9] The answer to another clue can be used to reverse a bleed caused by me

[12] I don't actually resolve migraines but rather prevent reoccurrence

[13] I'm a cream you can apply for patients with cannabinoid hyperemesis

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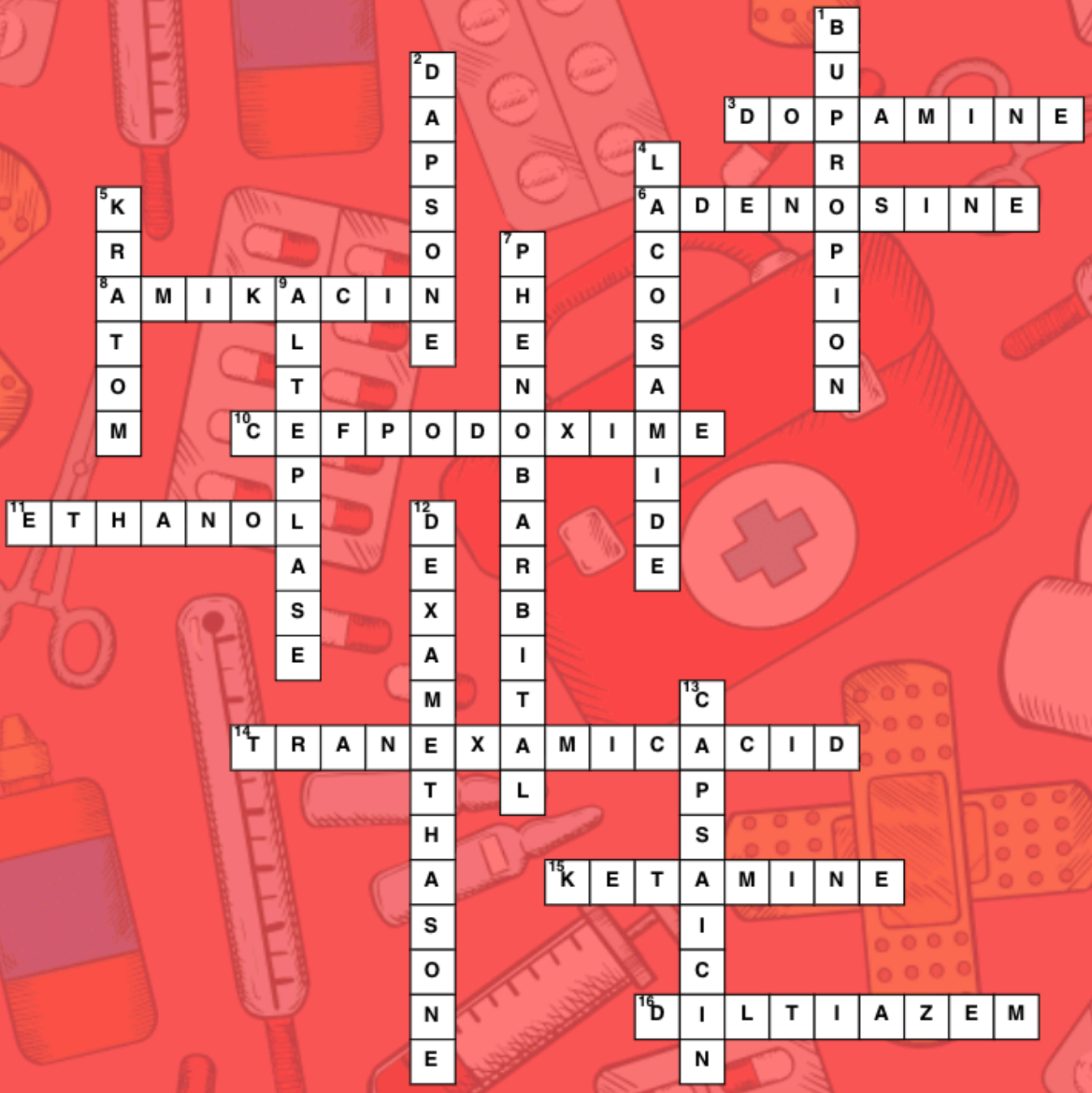
Solution

Academy of Emergency Medicine Pharmacists



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