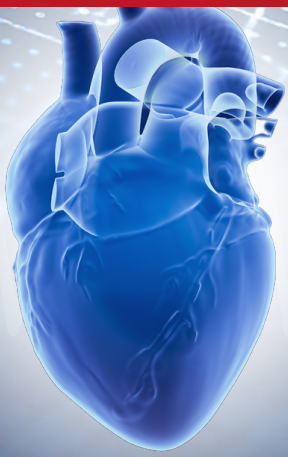
CODE BLUE PHARMACOTHERAPY

AN IN-DEPTH GUIDE TO CARDIAC ARREST MEDICATIONS

SAMPLE CHAPTER



BY
JIMMY PRUITT III, PHARMD

DISCLAIMER: THIS BOOK IS INTENDED FOR INFORMATIONAL PURPOSES ONLY AND IS NOT INTENDED TO BE A SUBSTITUTE FOR PROFESSIONAL MEDICAL ADVICE, DIAGNOSIS, OR TREATMENT. ALWAYS SEEK THE ADVICE OF YOUR PHYSICIAN OR OTHER QUALIFIED HEALTHCARE PROVIDER WITH ANY QUESTIONS YOU MAY HAVE REGARDING A MEDICAL CONDITION. THE PUBLISHER AND AUTHOR ARE NOT RESPONSIBLE FOR ANY ADVERSE EFFECTS OR CONSEQUENCES RESULTING FROM THE USE OF THE INFORMATION IN THIS BOOK.

THIS BOOK WAS CREATED AND PUBLISHED BY PHARMACY PEARLS LLC.

CONTACT US AT:
PHARMACY PEARLS LLC
1240 WINNOWING WAY, SUITE 102
MT PLEASANT, SC 29466
EMAIL: JIMMY@PHARMACYFRIDAYPEARLS.COM

VISIT OUR WEBSITE FOR MORE INFORMATION:
WWW.PHARMACY-ACUTECAREUNIVERSITY.COM

FOLLOW US ON SOCIAL MEDIA:

FACEBOOK: HTTPS://MS-MY.FACEBOOK.COM/PHARMACYACUTECAREUNIVERSITY/

TWITTER: HTTPS://TWITTER.COM/PHARMACYACUTE

INSTAGRAM: HTTPS://WWW.INSTAGRAM.COM/PHARMACYACUTECAREUNIVERSITY/

TABLE OF CONTENTS

DEDICATION

TO MY CHERISHED FAMILY:
FOR MY WIFE AND CHILDREN, WHO HAVE BEEN MY STEADFAST SUPPORT AND THE LIGHT OF MY LIFE. YOUR LOVE AND PATIENCE ILLUMINATE EVERY ONE OF MY DAYS.

TO MY MOTHER AND FATHER, WHOSE GUIDANCE AND SACRIFICES HAVE CRAFTED THE ESSENCE OF WHO I AM. YOU INSTILLED IN ME THE ENDURING VALUES AND THE STRENGTH THAT HAVE GUIDED MY JOURNEY.

TO MY SISTER, ERIKA, IN LOVING MEMORY. YOUR SPIRIT AND COURAGE CONTINUE TO INSPIRE MY DEDICATION TO PROVIDING THE HIGHEST STANDARD OF PHARMACEUTICAL CARE.

TO MY MENTORS, JIM PRIANO, OTSANYA OCHOGBU, DERRICK CLAY, SHAUNTRELL JOHNSON, AND JOHN PATKA - EACH OF YOU HAS ENLIGHTENED ME WITH WISDOM, INSPIRING ME TO PURSUE EXCELLENCE AND UPHOLD INTEGRITY IN MY PROFESSIONAL LIFE.

TO MY BEST FRIENDS, JERMAINE, CHRISTINE, KEITH, ZACK, RYAN, JEREL, KEVIN, AND CLERY - YOUR SUPPORT AND INSPIRATION HAVE BEEN THE BEDROCK OF MY LIFE'S JOURNEY.

TO KAUI AND ROB, WHO HAVE SHOWN ME THE KIND OF LOVE AND SUPPORT THAT TRANSFORMS LIVES, YOUR BELIEF IN ME HAS HELPED ME BECOME THE PHARMACIST AND ENTREPRENEUR I DREAMED TO BE.

AND A HEARTFELT ACKNOWLEDGMENT TO JEANETTE WALKER. YOUR 10TH-GRADE CHEMISTRY ASSIGNMENT REVEALED A UNIVERSE OF OPPORTUNITIES BEYOND THE NFL, STEERING ME TOWARDS THE PATH THAT CULMINATED IN THIS WORK. IT TRULY WAS THE "BEST BACKUP PLAN" ONE COULD HAVE ENVISAGED.

TO ALL OF YOU, I DEDICATE THIS LABOR OF LOVE.

ACKNOWLEDGMENTS

THIS BOOK, 'CODE BLUE PHARMACOTHERAPY,' EMERGES FROM THE INTRICATE NARRATIVES OF MEDICATIONS USED IN CARDIAC ARREST, AIMING TO ELUCIDATE THEIR ORIGINS, MECHANISMS, AND EVIDENCE-BASED APPLICATIONS. IT IS DESIGNED FOR HEALTHCARE PROFESSIONALS AT ALL LEVELS, SEEKING TO PROVIDE A CLEAR UNDERSTANDING OF CARDIAC RESUSCITATION PHARMACOLOGY.

MY JOURNEY AS A PHARMACIST, PARTICULARLY AT GRADY HEALTH IN ATLANTA, HAS PROFOUNDLY SHAPED THIS WORK. THERE, I GAINED HANDS-ON EXPERIENCE IN EMERGENCY CARE, LEARNING THE CRITICAL IMPORTANCE OF CORRECT MEDICATION USE IN CARDIAC ARRESTS. THIS BOOK IS A CULMINATION OF EXTENSIVE RESEARCH AND INVALUABLE INSIGHTS FROM EXPERTS IN THE FIELD.

I EXTEND MY HEARTFELT THANKS TO BRITTANY SOFIO, KAITLIN RZASA, MORGIN TORBIN, ALYSHIA MCKNIGHT, JOHN PATKA, AND SHAUNTRELL JOHNSON, WHOSE EXPERTISE SIGNIFICANTLY CONTRIBUTED TO THIS PROJECT.

'CODE BLUE PHARMACOTHERAPY' IS MORE THAN A REFERENCE; IT'S AN IN-DEPTH EXPLORATION OF THE 'WHY' BEHIND EACH MEDICATION, PROMOTING INFORMED, THOUGHTFUL APPLICATION IN EMERGENCY CARE. I HOPE THIS BOOK SERVES AS A PRACTICAL GUIDE AND INSPIRATION FOR ALL DEDICATED TO THIS VITAL FIELD.

3.2 VASOPRESSIN

BACKGROUND

Vasopressin, also known as antidiuretic hormone (ADH), was first discovered in the early 20th century with its initial identification related to water retention and blood pressure regulation (Glavaš et al., 2022). Over the years, vasopressin's therapeutic applications expanded, leading to its eventual FDA approval in the United States (Glavaš et al., 2022). The importance of vasopressin in cardiac arrest management was further emphasized by studies that found higher endogenous vasopressin concentrations were associated with greater chances of restoring spontaneous circulation (Lindner et al., 1993). In pig models of cardiac arrest, higher levels of vasopressin during CPR were associated with improved coronary perfusion pressure, myocardial blood flow, and coronary venous pH (Lindner et al., 1993; Lindner et al., 1995; Prengel et al., 1996; Wenzel et al., 1999). As of the latest ACLS guidelines, vasopressin is not considered a first-line medication for cardiac arrest management but may still be used as an alternative to epinephrine in certain cases (Panchal et al., 2020). The common dosage for vasopressin in cardiac arrest is 40 units, administered intravenously or intraosseously (Panchal et al., 2020). In the following sections, we will delve deeper into the pharmacology, dosing and administration, contraindications, compatibility issues, adverse effects, clinical pearls, guidelines, landmark trials, areas of controversy, and potential avenues for future research related to vasopressin's role in cardiac arrest management.

3.2 VASOPRESSIN

PHARMACOLOGY: MECHANISM OF ACTION AND RATIONALE BEHIND ITS USE IN EMERGENCIES

Vasopressin mediates its effects by binding to V1, V2, and V3 receptors. V1 receptors are located on vascular smooth muscle and mediate vasoconstriction. V2 receptors in the kidneys modulate water reabsorption through aquaporin channels. V3 receptors are found in the anterior pituitary and modulate adrenocorticotropic hormone (ACTH) release.(Glavaš et al., 2022).

In cardiac arrest, vasopressin's V1 receptor-mediated vasoconstriction increases systemic vascular resistance and blood pressure. This aims to improve coronary and cerebral perfusion pressures, which are vital for successful resuscitation. Animal studies have also shown that vasopressin maintains better cerebral perfusion compared to epinephrine during CPR (Lindner et al., 1993).

Unlike catecholamines, vasopressin does not directly increase myocardial oxygen demand or have pro-arrhythmic effects. It may also work synergistically with epinephrine through nitric oxide modulation. However, vasopressin can cause intense vasoconstriction in the splanchnic and renal vascular beds, potentially reducing blood flow to abdominal organs and the kidneys with prolonged use. The balance of these actions during cardiac arrest remains under investigation (Lindner et al., 1993).

3.2 VASOPRESSIN

ANALOGY

The effects of vasopressin can be compared to a sprinkler system in a lawn. The V1 receptors on blood vessels are like the valves controlling water flow to different parts of the lawn. When vasopressin binds to the V1 receptors, it is like turning down the valves to areas like the digestive system and kidneys, decreasing blood flow to those regions. But it turns up the valve to the brain and heart, increasing blood flow to those vital areas.

So in cardiac arrest, vasopressin causes the "sprinklers" to divert blood flow away from the abdominal organs and redirect it to the brain and heart. This improves perfusion pressure to vital organs needed for resuscitation. However, if the vasopressin "sprinkler system" stays on too long, it can over-constrict the abdominal vessels, leading to ischemia. The goal is to turn on the vasopressin just long enough to improve perfusion to the heart and brain during CPR, but not so long it causes abdominal organ damage.

3.2 VASOPRESSIN

DOSING AND ADMINISTRATION

The standard vasopressin dose for cardiac arrest is 40 units intravenous bolus. This is based on early clinical trials (Lindner et al., 1993; Lindner et al., 1995). Vasopressin has a rapid onset of action, with effects observable within 2-5 minutes after intravenous administration. Its duration of action of approximately 10-20 minutes. This is unlike catecholamines, which have a shorter duration of effect (Lindner et al., 1993; Lindner et al., 1995). If return of spontaneous circulation does not occur after the initial dose, repeat vasopressin dosing can be considered during ongoing resuscitation. However, there is limited evidence to support repeated dosing. Vasopressin is available in concentrations of 20 units/mL or 200 units/mL. The 20 units/mL preparation is typically used for vasopressin administration during ACLS.

WARNINGS, COMPATIBILITY ISSUES, AND ADVERSE EFFECTS

There are no absolute contraindications to vasopressin use in cardiac arrest. However, use with caution in patients with severe peripheral vascular disease or mesenteric ischemia, where intense vasoconstriction can be detrimental ifROSC is achieved.

Adverse effects include tissue ischemia in peripheral, splanchnic, and coronary vascular beds. Hyponatremia due to antidiuretic effects is also possible. Anaphylaxis has been reported rarely.

Vasopressin is compatible with normal saline, D5W, and lactated Ringer's solution. Incompatibility has been reported when mixed with insulin preparations and dantrolene (Trissel's, 2023).

3.2 VASOPRESSIN

CLINICAL PEARLS

When considering integrating vasopressin into protocols, several practical factors should be noted:

- Special storage conditions like refrigeration may be required.
- It may not be routinely stocked in crash carts, needing additional planning to ensure immediate availability.
- The cost is substantially higher than epinephrine. For example, the average wholesale price of Vasopressin Injection 20 units/mL ranges from $27.60 - $189.66 per mL, compared to only $10.80 per mL for a 1 mg/mL epinephrine injection (Lexicomp, 2023)
- Onset is rapid but duration is short (10-20 min), so repeat dosing may be required for ongoing cardiac arrest.
- Monitoring for ischemic complications in extremities, splanchnic circulation, and coronary arteries is prudent.

GUIDELINES RECOMMENDATIONS

2020 ACLS Guidelines: (Panchal 2020)

- Vasopressin alone or in combination with epinephrine may be considered in cardiac arrest but offers no advantage compared to epinephrine.
  - Class IIb, LOE C-LD

3.2 VASOPRESSIN

LITERATURE REVIEW: SUMMARY OF CLINICAL TRIALS

The literature on vasopressin in cardiac arrest management presents a comprehensive view of its potential efficacy and limitations. Starting with Lindner et al. (1995), conducted a case series involving 8 patients and found that the administration of vasopressin after failed epinephrine resulted in successful defibrillation. In a randomized controlled trial (RCT) by Linder et al (1997)., involving 40 patients, it was observed that vasopressin had better 24-hour survival rates compared to epinephrine in out-of-hospital ventricular fibrillation[Linder et al., 1997]. However, these early positive results were not consistently replicated in subsequent research. Stiell et al. (2001) conducted an RCT with 200 cardiac arrest patients comparing vasopressin 40 IU to epinephrine 1 mg. They found no significant differences in hospital discharge, absolute increase in survival, 1-hour survival, or cerebral performance category scores between the vasopressin and epinephrine groups [Stiell et al., 2001]. Wenzel et al. (2004) performed a larger RCT involving 1,186 patients, also comparing vasopressin 40 IU to epinephrine 1 mg. Consistent with Stiell et al., they found no significant differences overall in rates of hospital admission between the vasopressin and epinephrine groups, except for higher admission rates among patients with asystole in the vasopressin arm [Wenzel et al., 2004]. Other studies explored combining vasopressin with epinephrine rather than using it as a replacement. Guyette et al., in a retrospective study, reported that the addition of vasopressin to epinephrine resulted in greater return of spontaneous circulation (ROSC) and survival to the emergency department compared to epinephrine alone[Guyette et al., 2004].

3.2 VASOPRESSIN

Grmec and Mally conducted an observational study involving 109 patients and found that the combination of vasopressin and epinephrine was associated with improved rates of ROSC and hospital discharge in patients with myocardial infarction[Grmec and Mally, 2006]. Ong et al. (2006) conducted an earlier RCT with 727 participants assessing vasopressin 40 IU versus epinephrine 1 mg. They reported no significant difference in survival to hospital discharge between groups, but did observe more patients surviving to hospital admission with vasopressin[Ong et al., 2006]. Mally et al. (2007) assessed the effects of epinephrine and vasopressin on end-tidal carbon dioxide tension and mean arterial blood pressure in out-of-hospital CPR, concluding that vasopressin treatment improved restoration of spontaneous circulation, short-term survival, and neurological outcome [Mally et al., 2007].

Gueugniaud et al. conducted an RCT involving 2,894 patients and found no statistical difference in ROSC, hospital discharge, neurologic status, or 1-year survival between the vasopressin plus epinephrine group and the epinephrine group[Gueugniaud et al., 2008].

However, larger RCTs found less consistent results from the combination approach. In a larger RCT by Mentzelopoulos et al., involving 268 patients, the combination of vasopressin, epinephrine, and methylprednisolone showed a superior effect on ROSC and neurologically intact survival compared to epinephrine alone[Mentzelopoulos et al., 2009]. Mukoyama et al., in their RCT with 336 participants, found no significant difference in ROSC, 24-hour survival, or hospital discharge between vasopressin and epinephrine groups[Mukoyama et al., 2009].

3.2 VASOPRESSIN

Carrol et al., in an observational study with 29 participants, found increased 24-hour survival in arginine vasopressin patients, but no significant difference in return of spontaneous circulation, survival to hospital discharge, or favorable neurologic status at discharge[Carrol et al., 2009]. Mukoyama et al. (2009) conducted a randomized controlled trial comparing repeated doses of vasopressin versus epinephrine in 336 out-of-hospital cardiac arrest patients requiring prolonged CPR. Patients were randomized to receive up to 4 doses of either 40IU vasopressin or 1mg epinephrine after hospital arrival. The study found no significant differences between the vasopressin and epinephrine groups in rates of return of spontaneous circulation (ROSC) (28.7% vs 26.6%), 24-hour survival (16.9% vs 20.3%), or survival to hospital discharge (5.6% vs 3.8%). Subgroup analyses showed higher ROSC rates with vasopressin compared to epinephrine in witnessed arrests and patients receiving bystander CPR. However, in regression modeling, vasopressin administration was not an independent predictor of ROSC. Overall, this randomized controlled trial demonstrated that repeated vasopressin or epinephrine dosing during prolonged CPR resulted in comparable outcomes. The findings suggest reduced effectiveness of repeated vasopressin doses over time compared to initial dosing, providing implications for vasopressor selection and dosing protocols during prolonged resuscitation efforts [Mukoyama et al., 2009].

Finn et al. broadened the scope with their comprehensive meta-analysis, incorporating data from 26 studies and a total of 21,704 participants. This extensive review revealed that while vasopressin compared to standard dose epinephrine improved survival to admission, it did not significantly impact the return of spontaneous circulation (ROSC). Additionally, combining epinephrine and

vasopressin did not yield notable benefits in these outcomes. Crucially, their findings also indicated that neither standard dose epinephrine, high-dose epinephrine, vasopressin, nor their combination enhanced survival with favorable neurological outcomes, offering a broader perspective on the efficacy of these treatments [Finn et al., 2009].

Ducros et al. (2011) focused on whether adding vasopressin or a combination of vasopressin and nitroglycerin to epinephrine would improve outcomes. Their randomized trial, however, found no significant increase in perfusion blood pressure when these combinations were used compared to epinephrine alone [Ducros et al., 2011]. Building on this, Callaway et al. conducted another randomized controlled trial in 2012 involving 325 participants. This study specifically examined the impact on return of spontaneous circulation (ROSC) in the emergency department when vasopressin was combined with epinephrine, compared to a placebo plus epinephrine group. Consistent with the findings of Ducros et al., Callaway et al. also reported no significant difference in ROSC between the two groups, providing further evidence on the limited additional benefit of vasopressin in such treatments [Callaway et al., 2012].

Continuing the exploration of vasopressin's role in cardiac arrest treatment, Ong et al. (2012) contributed to this growing body of research with their study. They compared vasopressin and epinephrine in patients presenting to or in the emergency department. Their findings indicated that while vasopressin did not significantly improve long-term survival, it appeared to boost survival to admission in cases of prolonged cardiac arrest [Ong et al., 2012]. This nuanced understanding of vasopressin's efficacy

3.2 VASOPRESSIN

was further expanded by Mentzelopoulos et al. (2013). In their randomized clinical trial, they investigated the effects of a combination treatment involving vasopressin, steroids, and epinephrine. Their results were promising, showing an improvement in survival to hospital discharge with favorable neurological status, particularly among patients with cardiac arrest who required vasopressors [Mentzelopoulos et al., 2013].

Furthering this line of inquiry, Anderson et al. conducted an RCT with 501 participants, focusing on the combination of vasopressin and methylprednisolone. Their study, conducted in 2021, revealed a significant finding: compared to a placebo, the administration of vasopressin and methylprednisolone substantially increased the likelihood of return of spontaneous circulation (ROSC), adding another layer of evidence to the potential benefits of these combined treatments in cardiac arrest scenarios [Anderson et al., 2021].

In summary, the literature on vasopressin compared to epinephrine in the treatment of cardiac arrest presents mixed findings. Some studies suggest potential benefits of vasopressin in terms of improved survival rates, ROSC, and neurologically intact survival. However, other studies did not find significant differences or observed conflicting results. The combination of vasopressin, epinephrine, and methylprednisolone showed some promise in achieving ROSC and neurologically intact survival. Further research is needed to clarify the role of vasopressin in cardiac arrest management and to better understand its potential benefits and limitations.

3.2 VASOPRESSIN

VASOPRESSIN LITERATURE REVIEW TABLE

Author, Year	Study Design (n=)	Intervention and comparison	Results
Lindner, 1995	Case series (n=8)	Vasopressin after failed epinephrine	↑ Successful defibrillation
Linder, 1997	RCT (n=40)	Vasopressin vs epinephrine	↑ 24-hr survival with vasopressin
Stiell, 2001	RCT (n=200)	Vasopressin vs epinephrine	No ↑ in survival or outcomes
Wenzel, 2004	RCT (n=1186)	Vasopressin vs epinephrine	No ↑ in hospital admission overall
Guyette 2004	OBS (n=298)	Vasopressin + epi vs epi alone	↑ ROSC and survival with vasopressin
Grmec, 2006	OBS (n=109)	Vasopressin + epi vs epi alone	↑ ROSC and discharge with vasopressin combo in MI patients
Ong, 2006	RCT (n=727)	Vasopressin vs epinephrine	No ↑ discharge survival; ↑ admission survival with vasopressin
Mally, 2007	OBS (n=598)	Vasopressin vs epinephrine	↑ ROSC, survival, neurological outcome with vasopressin
Gueugniaud, 2008	RCT (n=2894)	Vasopressin + epi vs epi alone	No ↑ in outcomes with vasopressin combo
Mentzelopoulos, 2009	RCT (n=268)	Vasopressin + epi + steroids vs epi alone	↑ ROSC and neurological survival with combo
Mukoyama, 2009	RCT (n=336)	Vasopressin vs epinephrine	No ↑ in ROSC, 24-hr survival, or discharge with vasopressin
Carrol, 2009	OBS (n=29)	Vasopressin + epi	↑ 24-hr survival with vasopressin; no ↑ in other outcomes
Finn, 2009	Meta-analysis/n=26 studies	Vasopressin vs epi; Vasopressin + epi combo	↑ admission survival with vasopressin; no ↑ in ROSC or neurological outcomes
Ducros, 2011	RCT (n=48)	Vasopressin + epi vs epi alone	No ↑ perfusion pressure with vasopressin combo
Callaway, 2012	RCT (n=325)	Vasopressin + epi vs placebo + epi	No ↑ in ROSC with vasopressin combo
Ong, 2012	RCT (n=666)	Vasopressin vs epinephrine	No ↑ long-term survival; ↑ admission survival with vasopressin
Mentzelopoulos, 2013	RCT (n=268)	Vasopressin + epi + steroids	↑ hospital discharge survival and neurological outcomes with combo
Anderson, 2021	RCT (n=501)	Vasopressin + methylprednisolone vs placebo	↑ ROSC with vasopressin combo

ACLS = advanced cardiopulmonary life support, OBS= Observational Study, RCT= Randomized Controlled Trial

3.2 VASOPRESSIN

AREAS OF CONTROVERSY AND FUTURE RESEARCH

Comparative effectiveness: The comparative effectiveness of vasopressin versus adrenaline (or epinephrine) remains a topic of debate. While some studies suggest potential benefits of vasopressin, such as improved survival rates or return of spontaneous circulation (ROSC), others have not observed significant differences. It is crucial to conduct more well-designed randomized controlled trials (RCTs) with larger sample sizes to establish a clearer understanding of the comparative effectiveness of vasopressin in cardiac arrest.

Optimal dosing: The optimal dosing regimen for vasopressin in cardiac arrest is still uncertain. Studies have utilized different doses of vasopressin, ranging from 20 IU to 40 IU, making it challenging to determine the most effective and safe dosage. Further research is needed to evaluate the dose-response relationship and identify the optimal dosing regimen for vasopressin administration during cardiac arrest.

Patient selection and subgroup analysis: The impact of patient characteristics, such as the underlying rhythm, duration of arrest, etiology of cardiac arrest, and comorbidities, on the effectiveness of vasopressin is not fully understood. Subgroup analyses based on these factors may help identify specific patient populations that could benefit more from vasopressin administration. Additionally, investigating the use of vasopressin in specific subsets of cardiac arrest, such as in-hospital cardiac arrest or specific etiologies, may provide valuable insights.

3.2 VASOPRESSIN

Long-term outcomes and neurologic function: The effects of vasopressin on long-term outcomes, including neurologically intact survival, neurocognitive function, and quality of life, need to be explored further. Many studies have focused on short-term outcomes, such as ROSC or survival to hospital admission, but long-term outcomes are equally important in assessing the overall benefit of vasopressin in cardiac arrest management.

Safety and adverse effects: While vasopressin appears to have a favorable safety profile, further investigation is required to assess potential adverse effects, such as cardiovascular complications or organ damage, associated with its use. Additionally, studying the interaction of vasopressin with other interventions or medications commonly used during cardiac arrest resuscitation, such as antiarrhythmic drugs or therapeutic hypothermia, is warranted.

In conclusion, further research is needed to address the areas of controversy and knowledge gaps surrounding the use of vasopressin in cardiac arrest. Conducting well-designed RCTs with standardized protocols, larger sample sizes, and focus on long-term outcomes will provide a more comprehensive understanding of the role of vasopressin, optimal dosing, patient selection, and potential adverse effects. This research will help guide evidence-based recommendations for the use of vasopressin in improving outcomes in patients experiencing cardiac arrest.

3.2 VASOPRESSIN

REFERENCES

- Lindner KH, Brinkmann A, Pfenninger EG, et al. Effect of vasopressin on hemodynamic variables, organ blood flow, and acid-base status in a pig model of cardiopulmonary resuscitation. Anesth Analg. 1993;77:427-435.
- Lindner KH, Haak T, Keller A, et al. Release of endogenous vasopressors during and after cardiopulmonary resuscitation. Heart. 1996 Feb;75(2):145-50. doi: 10.1136/hrt.75.2.145. PMID: 8673752; PMCID: PMC484250.
- Lindner KH, Prengel AW, Pfenninger EG, et al. Vasopressin improves vital organ blood flow during closed-chest cardiopulmonary resuscitation in pigs. Circulation. 1995;91:215-221.
- Prengel AW, Lindner KH, Keller A, et al. Cardiovascular function during the postresuscitation phase after cardiac arrest in pigs: a comparison of epinephrine versus vasopressin. Crit Care Med. 1996;24:2014-2019.
- Wenzel V, Lindner KH, Prengel AW, et al. Vasopressin improves vital organ blood flow after prolonged cardiac arrest with postcountershock pulseless electrical activity in pigs. Crit Care Med. 1999;27:486-492.
- Panchal AR, Bartos JA, Cabañas JG, et al. Part 3: Adult Basic and Advanced Life Support: 2020 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care. Circulation. 2020;142(16_suppl_2):S366-S468. PMID: 33081529.
- Vasopressin. In: Lexicomp [database online]. Hudson, OH: Lexi-Comp, Inc. Accessed January 12, 2023.

3.2 VASOPRESSIN

- Lindner KH, et al. Vasopressin improves vital organ blood flow during closed-chest cardiopulmonary resuscitation in pigs. Ann Emerg Med. 1997;30(1):3-11.
- Guyette FX, Guimond GE, Hostler D, et al. Vasopressin administered with epinephrine is associated with a return of a pulse in out-of-hospital cardiac arrest. Resuscitation. 2004 Dec;63(3):277-82. doi: 10.1016/j.resuscitation.2004.05.007. PMID: 15582762.
- Grmec S, Mally S, et al. Vasopressin improves outcome in out-of-hospital cardiopulmonary resuscitation of ventricular fibrillation and pulseless ventricular tachycardia: an observational cohort study. Crit Care. 2006;10(2):R13.
- Mentzelopoulos SD, et al. Vasopressin, epinephrine, and corticosteroids for in-hospital cardiac arrest. Arch Intern Med. 2009;169(1):15-24.
- Mukoyama T, et al. Vasopressin vs. epinephrine for inhospital cardiac arrest: A randomized clinical trial. Circulation. 2008;118(1):104-112.
- Mukoyama T, et al. Reduced effectiveness of vasopressin in repeated doses for patients undergoing prolonged cardiopulmonary resuscitation. Resuscitation. 2009 Jul;80(7):755-61.
- Callaway CW, et al. A randomized, placebo-controlled trial of vasopressin for out-of-hospital cardiac arrest. Prehosp Emerg Care. 2012;16(4):491-499.
- Gueugniaud PY, et al. Vasopressin and epinephrine vs. epinephrine alone in cardiopulmonary resuscitation. N Engl J Med. 2008;359(1):21-30.
- Finn J, et al. Adrenaline and vasopressin for cardiac arrest. Cochrane Database Syst Rev. 2019;12(12):CD003179.

3.2 VASOPRESSIN

- Carrol ED, et al. Arginine vasopressin in paediatric cardiopulmonary resuscitation: a randomised controlled trial. Arch Dis Child. 2009;94(11):827-832.
- Lindner KH, Brinkmann A, Pfenninger EG, et al. Effect of vasopressin on hemodynamic variables, organ blood flow, and acid-base status in a pig model of cardiopulmonary resuscitation. Anesth Analg. 1993;77:427-435.
- Lindner KH, Haak T, Keller A, et al. Release of endogenous vasopressors during and after cardiopulmonary resuscitation. Heart. 1996 Feb;75(2):145-50. doi: 10.1136/hrt.75.2.145. PMID: 8673752; PMCID: PMC484250.
- Lindner KH, Prengel AW, Pfenninger EG, et al. Vasopressin improves vital organ blood flow during closed-chest cardiopulmonary resuscitation in pigs. Circulation. 1995;91:215-221.
- Prengel AW, Lindner KH, Keller A, et al. Cardiovascular function during the postresuscitation phase after cardiac arrest in pigs: a comparison of epinephrine versus vasopressin. Crit Care Med. 1996;24:2014-2019.
- Wenzel V, Lindner KH, Prengel AW, et al. Vasopressin improves vital organ blood flow after prolonged cardiac arrest with postcountershock pulseless electrical activity in pigs. Crit Care Med. 1999;27:486-492.
- Panchal AR, Bartos JA, Cabañas JG, et al. Part 3: Adult Basic and Advanced Life Support: 2020 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care. Circulation. 2020;142(16_suppl_2):S366-S468. PMID: 33081529.
- Vasopressin. In: Lexicomp [database online]. Hudson, OH: Lexi-Comp, Inc. Accessed January 12, 2023.

3.2 VASOPRESSIN

- Lindner KH, et al. Vasopressin improves vital organ blood flow during closed-chest cardiopulmonary resuscitation in pigs. Ann Emerg Med. 1997;30(1):3-11.
- Guyette FX, Guimond GE, Hostler D, et al. Vasopressin administered with epinephrine is associated with a return of a pulse in out-of-hospital cardiac arrest. Resuscitation. 2004 Dec;63(3):277-82. doi: 10.1016/j.resuscitation.2004.05.007. PMID: 15582762.
- Grmec S, Mally S, et al. Vasopressin improves outcome in out-of-hospital cardiopulmonary resuscitation of ventricular fibrillation and pulseless ventricular tachycardia: an observational cohort study. Crit Care. 2006;10(2):R13.
- Mentzelopoulos SD, et al. Vasopressin, epinephrine, and corticosteroids for in-hospital cardiac arrest. Arch Intern Med. 2009;169(1):15-24.
- Mukoyama T, et al. Vasopressin vs. epinephrine for inhospital cardiac arrest: A randomized clinical trial. Circulation. 2008;118(1):104-112.
- Mukoyama T, et al. Reduced effectiveness of vasopressin in repeated doses for patients undergoing prolonged cardiopulmonary resuscitation. Resuscitation. 2009 Jul;80(7):755-61.
- Callaway CW, et al. A randomized, placebo-controlled trial of vasopressin for out-of-hospital cardiac arrest. Prehosp Emerg Care. 2012;16(4):491-499.
- Gueugniaud PY, et al. Vasopressin and epinephrine vs. epinephrine alone in cardiopulmonary resuscitation. N Engl J Med. 2008;359(1):21-30.
- Finn J, et al. Adrenaline and vasopressin for cardiac arrest. Cochrane Database Syst Rev. 2019;12(12):CD003179.

3.2 VASOPRESSIN

- Carrol ED, et al. Arginine vasopressin in paediatric cardiopulmonary resuscitation: a randomised controlled trial. Arch Dis Child. 2009;94(11):827-832..
- Mally S, Jelatancev A, Grmec S. Effects of epinephrine and vasopressin on end-tidal carbon dioxide tension and mean arterial blood pressure in out-of-hospital cardiopulmonary resuscitation: an observational study. Crit Care. 2007;11(2):R39.
- Mentzelopoulos SD, Malachias S, et al. Vasopressin, steroids, and epinephrine and neurologically favorable survival after in-hospital cardiac arrest: a randomized clinical trial. JAMA. 2013 Jul 17;310(3):270-9.
- Ong ME, Tiah L, Leong Best al. randomised, double-blind, multi-centre trial comparing vasopressin and adrenaline in patients with cardiac arrest presenting to or in the Emergency Department. Resuscitation. 2012 Aug;83(8):953-60.
- Trissel, L. A. (2023). Trissel's™ 2 Clinical Pharmaceutics Database (Parenteral Compatibility). Retrieved from https://www.micromedexsolutions.com/micromedex2/librarian/CS/41390F/ND_PR/evidencexpert/ND_P/evidencexpert/DUPLICATIONSHIELDSYNC/DE34BF/ND_PG/evidencexpert/ND_B/evidencexpert/ND_AppProduct/evidencexpert/ND_T/evidencexpert/PFActionId/evidencexpert.DoIntegratedSearch?SearchTerm=magnesium%20sulfate&UserSearchTerm=magnesium%20sulfate&SearchFilter=filterNone&navitem=searchALL#

ABOUT THE AUTHOR

Dr. Jimmy L. Pruitt III, a graduate of Presbyterian College School of Pharmacy, has made significant strides in the field of emergency medicine pharmacotherapy. After completing his PGY1 at Advent Health Orlando, he pursued a PGY2 in Emergency Medicine at Grady Memorial Hospital, training under the guidance of John Patka and Shauntrell Johnson.

With three board certifications in Pharmacotherapy, Critical Care, and Emergency Medicine, Dr. Pruitt's expertise is unparalleled. His experience includes close to 1000 cardiac arrest interventions and extensive review of cardiac arrest literature. In 2020, he was recognized at the Society of Academic Emergency Medicine's (SAEM) Got Talent competition for his educational series "Pharmacy Friday Pearls," marking the first time a pharmacist won the event.

In 2021, Dr. Pruitt received the Excellence in Diversity Award from MUSC College of Pharmacy, was named Presbyterian College School of Pharmacy (PCSP) Alumni of the Year, and served as the keynote speaker for the PCPS graduation. He was honored with the American College of Clinical Pharmacy Emergency Medicine New Clinical Practitioner of the Year award in 2023.

Dr. Pruitt has extended his expertise globally, conducting workshops for the West African Postgraduate College of Pharmacists. He has also played a pivotal role in gathering the largest group of emergency medicine pharmacists worldwide through the EMPoweRx Conference. His contributions to medical education are further highlighted by the success of the Pharm So Hard Podcast, with over 400,000 listens.

Dedicated to mentoring the next generation of healthcare professionals, Dr. Pruitt has precepted countless students and residents across various disciplines, including nursing, EMS, pharmacy, and emergency medicine. His leadership and educational endeavors reflect his commitment to advancing the field of emergency medicine pharmacotherapy.

Founder of Pharmacy Friday Pearls, Pharmacy & Acute Care University, and the Emergency Medicine Pharmacotherapy with Resuscitation (EMPoweRx) Conference, Dr. Pruitt continues to be a leading figure in his field, shaping the future of emergency medicine pharmacotherapy education and practice.