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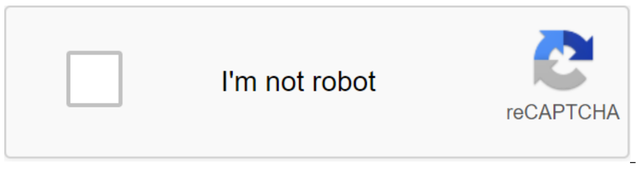

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1. Elbers PW, Ince C. Critical Disease Mechanisms - classification of microcirculatory flow anomalies in distribution shock. Crete Care 2006;10:221-221Crossref Web Science MedlineGoogle Scholar2. De Baker D, Beeston P, DevriendT J, et al. Comparison of dopamine and norepinephrine in the treatment of shock. N Engl J Med 2010;362:779-789Free full text web science MedlineGoogle Scholar3. Dellinger RP, Levi MM, Rhodes A, et al. Survival Sepsis Campaign: International Guidelines for Managing Severe Sepsis and Septic Shock, 2012. Med Intensive Care 2013;39:165-228Crossref Web of Science MedlineGoogle Scholar4. Brown SM, Lanspa MJ, Jones JP, et al. Survival after shock requires high-dose vasopressotherapy. Breast 2013;143:664-671Crossref Web Science MedlineGoogle Scholar5. Mayr FB, Yende S, Angus DC. Epidemiology of severe sepsis. Virulence 2014;5:4-11Crossref Web of Science MedlineGoogle Scholar6. Morelli A, Ertmer S, Westphal M, et al. Effect of heart rhythm control with esmolol on hemodynamic and clinical outcomes in patients with septic shock: a randomized clinical trial. JAMA 2013;310:1683-1691Crossref Web of Science MedlineGoogle Scholar7. Correa TD, Takala J, Jacob SM Angiotensin II in septic shock. Crete Care 2015;19:98-98Crossref Web Science MedlineGoogle Scholar8. Derrick JR, Anderson JR, Roland BJ. Additional use of the bio agent of the pressor, angiotensin, in the management of shock. Circulation 1962;25:263-267Crossref Web Science MedlineGoogle Scholar9. Del Greco F., Johnson, D.C. Clinical experience with angiotensin II in the treatment of shock. JAMA 1961;178:994-999Crossref Web Science MedlineGoogle Scholar10. Antonucci E, Gleason PJ, Annoni F, et al. Angiotensin II under fireproof septic shock. Shock 2017;47:560-566Crossref Web of Science MedlineGoogle Scholar11. Chawla LS, Busse L, Brasha-Mitchell E, et al. Intravenous angiotensin II for the treatment of high-efethon shock (ATHOS test): experimental study. Crete Care 2014;18:534-534Crossref Web Science MedlineGoogle Scholar12. Chawla LS, Russell JA, Bagshaw SM, et al. Angiotensin II for the treatment of high power shock 3 (ATHOS-3): protocol for Phase III, double-blind, randomized controlled trial. Crete Care Resusc 2017;19:43-49Web Science MedlineGoogle Scholar13. Food and Drug Administration. Special Protocol Assessment: A Guide to Industry. Draft guidance. May 2016 (.14. Knaus WA, Draper EA, Wagner DP, zimmerman JE. APACHE II: The severity of the disease classification system. Crete Care Med 1985;13:818-829Crossref Web Science MedlineGoogle Scholar15. Vincent JL, Moreno R, Takala J, et al. SOFA Assessment (Assessment of Organ Failure Related To Sepsis) to Describe bodies: on behalf of the Working Group on Sepsis, the European Society of Society Care medicine. Intensive Care Med 1996;22:707-710Crossref Web Science MedlineGoogle Scholar16. Bradley SE, Parker B. Hemodynamic effects of angiotonin in a normal person. J Clin Invest 1941;20:715-719Crossref MedlineGoogle Scholar17. Basso N, Terragno NA. The story of the discovery of the renin-angiotensin system. Hypertension 2001;38:1246-1249Crossref Web Science MedlineGoogle Scholar18. Thomas VL, Nielsen MS. Administration of angiotensin II in fireproof septic shock. Crete Care Med 1991;19:1084-1086Crossref Web Science MedlineGoogle Scholar19. Ray GM, Coakley JH. Severe septic shock without reacting to norepinephrine. Lancet 1995;346:1604-1604Crossref Web Science MedlineGoogle Scholar20. Yunge M, Petros A. Angiotensin for septic shock does not respond to norepinephrine. Arch Dis Baby 2000;82:388-389Crossref Web Science MedlineGoogle Scholar21. Bassi E, Park M, Azevedo LC. Therapeutic strategies for high-dose vasopressor-dependent shock. Crete Care Res Pract 2013;2013:654708-65470822. Lopez A, Lorente JA, Steingrub J, et al. Multicenter randomized, placebo-controlled, double-blind study of nitric oxide syntasis inhibitor 546C88: effect on survival in patients with septic shock. Crete Care Med 2004;32:21-30Crossref Web Science MedlineGoogle Scholar23. Inoue T, Mi Kew, Gillespie DG, Jackson EK. Cycloxygenase inhibition shows the synergistic effect of the vasoconstricted on mesangial cell growth. Eur J Pharmacol 1998;361:285-291Crossref Web of Science MedlineGoogle Scholar24. Struthers AD, Pai S, Seidelin PH, Coutie WJ, Morton JJ. Evidence in humans for post-sineaptic interaction between nor peprenaline and angiotensin II in relation to systolic but not diastolic blood pressure. J Hypertens 1987;5:671-676Crossef Web of Science MedlineGoogle Scholar Angiotensin II for the treatment of vasodilator shock. Hannah A, English SW, Wang XS, Ham K, Tumlin J, Szerlip H, Busse LW, Altaweel L, Albertson TE, McKay C, McCurdy MT, Boldt DW, Choc S, Young PJ, Krell K, Wunderink RG, Ostermann M, Murugan R, Gong MN, Panvar R, Hestback J, Favory R, Venkatesh B, Thompson BT, Bellomo R, Jensen J, Kroll Hannah A, et al n Engl J Med. 2017 Aug 3;377(5):419-430. doi: 10.1056/NEJMoa1704154. Epub 2017 May 21. N Engl J Med. 2017. PMID: 28528561 Clinical trials. @article Bellomo2017TheAT, title The Test of ATHOS-3, Angiotensin II and the Three Musketeers, by R. Bellomo and A. Hilton, Journal of Critical Care and Resuscitation: Journal of the Australian Academy of Critical Care Medicine, year {2017}, volume 19 1, pages 3-4 R. Bellomo, A. HiltonPublished 2017Medicine, Engineering and Critical Care and Resuscitation : Journal of the Australian Academy of Critical Medicine Care3 Hypotension is a defining hemodynamic marker of shock in general, and septic shock in particular, and is a defining a hemodynamic marker of shock in general. Therapeutic interventions around the world.1 The purpose of such interventions is to restore blood pressure, which is considered suffi cient to maintain perfusion of vital organs, while interventions are carried out treating the main cause of shock. In the case of septic shock or other forms of inffluoric vasofluoric vasodilated shock, interventions are used to restore adequate blood pressure... CONTINUE READINGSHOWING 1-10 of 19 REFERENCESSORT BYRelevanceMost Influential RecordsConverted: Patients with distribution shock who require high doses of vasopressors have high mortality. Angiotensin II (ATII) may be beneficial in patients who remain hypotensive despite catecholamine and vasopressin therapy. The appropriate dose of parenteral angiotensin II for shock is unknown. Methods: In total, 20 patients with distributional shock and cardiovascular consistent evaluation of organ failure score 4 were randomized by either ATII infusion (N No. 10) or placebo (N No. 10) plus standard care. THE ATII was started at a dose of 20 ng/kg/min, and was credited with the aim of maintaining an average blood pressure (MAP) of 65 mmHg. The infusion (or ATII or placebo) was continued for 6 hours and then titrated off. The main endpoint was the effect of ASI on the constant dose of norephrine needed to maintain MAP 65 mmHg. Results: ATII has led to a marked reduction in noreprenein dosing in all patients. The average dose of norradine 1 hour for the placebo cohort was 27.6 ± 29.3 micrograms per minute versus 7.4 ± 12.4 micrograms per minute for the ATII cohort (P 0.06). The most common side-effect associated with ASIA was hypertension, which occurred in 20% of patients receiving ASIA. The 30-day mortality rate for the ATII cohort and the placebo cohort was similar (50% vs. 60%, P 1.00). Conclusion: Angiotensin II is an effective means of saving vasopressor in patients with distribution shock requiring several vasopressors. The initial dose range of ATII, which appears to be suitable for patients with distribution shock is 2 to 10 ng/kg/min. Trial registration: Clinicaltrials.gov NCT01393782. Registered on July 12, 2011. From Wiki Journal Club Khanna A et al. Angiotensin II for the treatment of vasodilator shock. New Engl J Med. 2017. 377:419-30.PubMed - Full text - PDF In patients with severe vasodilator shock requiring high doses of catecholamines, does angiotensin II lead to an improvement in average blood pressure (MAP) compared to placebo? Bottom line In patients with severe vasodilator shock (MAP 55-70, despite a 0.2ug/kg/min norepinephrine or equivalent), administration of angiotensin II is associated with a 45% absolute increase in map response (defined as an increase in MAP ≥ 10mmHg or MAP qgt; 75mmHg) compared to placebo. Main shock is defined as any process leading to insufficient blood pressure to provide adequate perfusion of organs. The most common form of shock is vasodilator shock, usually usually as a shock in the conditions of peripheral vasodilation and untouched cardiac products. In addition to treating the underlying cause of vasodilator shock, catecholamine (e.g. dopamine, norepinephrine, epinephrine) and necatolamin (e.g. vasopressin) vasopressors are agents that fight vasodilator shock by causing peripheral vasocolizing. However, these agents are not equally effective in the treatment of vasodilator shock, and their use is associated with significant side effects including limb ischemia and cardiac arrhythmia. These difficulties are reflected in the fact that in patients with vasodilator shock requiring high doses of vasopressors, mortality is approaching 50% within 1 month. As a result, more effective vasopressor variants are needed. Angiotensin II is a natural hormone secreted as part of the renin-angiotensin system, leading to a powerful systemic vasoconstricting. An early study of angiotensin II in patients with vasodilator shock suggested the beneficial effects of this agent on systemic average blood pressure (MAP). A larger randomized controlled trial was needed to evaluate the effectiveness of this agent in a wider range of patients with severe vasodilator shock. The 2017 Angiotensin II for the Treatment of High-Echipeed Shock (ATHOS-3) trial of randomized 321 severe vasodilator shock patients either angiotensin II or placebo and is evaluated for the initial result of the MAP response (defined as an increase in MAP ≥ 10mmHg or MAP qgt; 75Hg without increased background vessels) in 3 hours. In ATHOS-3, 70% of patients who received angiotensin II met the criteria for a MAP response (45% absolute increase compared to placebo). There was a significant decrease in the dose of catecholamine in patients receiving angiotensin II. In 48 hours, the average improvement in cardiovascular consistent organ function evaluation (SOFA) evaluation, an overall indicator of systemic organ function, was greater in the angiotensin II group (partly due to lower catecholamine dosing, which is reflected in this assessment). Angiotensin II was well tolerated, with a lower negative event rate compared to placebo. As a research endpoint, there is a downward trend in mortality with angiotensin II, with a statistically insignificant 9% absolute reduction in mortality by 30 days. Thus, ATHOS-3 provides fairly compelling evidence that angiotensin II is safe and effective in reducing peripheral vasodilation and improving hemodynamics in severe vasodilator shock. Further research is needed to determine whether exposure to angiotensin II lead to improved morbidity and mortality in this condition. The August 2017 guidelines were not published to reflect the results of this Promising design, multicenter, double-blind, randomized controlled trial N-321 Angiotensin II (n'163) Placebo (n'158) Setting: 75 centers North America, Australia, Asia, and Europe Enrollment: May 2015 to January 2017 Duration follow-up: 28 days Analysis: Modified intention-to-treat (study drug started) Primary outcome: MAP response at 3 hours (defined as MAP increase ≥ 10mmHg or resultant MAP > 75mmHg) Population Inclusion Criteria Age ≥ 18 years Catecholamine-resistant hypotension defined as > 0.2ug/kg/min of norepinephrine or equivalent for 6-48 hours to maintain MAP 55-70mmHg Central venous access and arterial catheter present Indwelling urinary catheter present Received at least 25mL/kg of crystalloid or colloid equivalent over the previous 24-hour period and felt by treating investigator to be adequately volume resuscitated Features of high-output shock defined as 1 of the following criteria: Central venous O2 saturation > 70% and central venous pressure > 8mmHg Cardiac index > 2.3 L/min/m2 Exclusion Criteria Burn covering > 20% of total body surface area Cardiovascular SOFA score ≤ 3 Acute occlusive coronary syndrome requiring intervention On VA ECMO or previously on ECMO for less than 12 часов печеночной недостаточности с MELD оценка ≥ 30 История астмы с активным бронхоспазмом, требующих бронхолитизации (если intubated) Острая мезентерическая ишемия или история мезентерической ишемии История, наличие, или высокое подозрение на рассечение аорты или брюшной аневризмы аневризмы системный склероз, или вазоссастичное заболевание Ожидаемая продолжительность жизни < 12 часов Активное кровотечение с любой из: ожидаемая потребность (в течение 48 часов после начала исследования) для переливания > 4 единиц упакованных красных кровяных телец гемоглобина < 7g/dL или любое другое условие, которое бы противопоказание серийный анализ крови Абсолютный отсчет нейтрофила < 1000 клеток/мм3 Известная аллергия на маннитол В настоящее время участвует в другом клиническом испытании Известно, что беременна во время скрининга Базовые характеристики Все пациенты Демография: возраст 64 , мужчины 60,7%, ИМТ ≥ 30 44,3% Шок : MAP 66.3, ОЦЕНКА APACHE II 28, ScvO2 77.0, CVP 12mmHg, CI 3.1 L/min/m2, септик 80,7%, ARDS 28,4%, вазопрессин использовать 6 часов до рандомизации 69,8%, норадреналина доза 0,34 уг/ кг / мин Labs: Альбумин 2,3, MELD 22 Лекарства воздействия: ингибитор АПФ 9,3%, АРБ 6,9% Вмешательства Рандомизированные 1:1 к ангиотензину II или соответствующие плацебо Рандомизация стратифицированы в соответствии с MAP (< 65 против ≥ 65) и ОЦЕНКА APACHE II (≤ 30, от 31 до 40, или ≥ 41) Следователи, исследовательский персонал, пациенты, семьи, и спонсор были ослеплены лечения уступки Базовый MAP создан как среднее 3 определения выполнены 30, 15, и за 0 минут до начала лечения инфузии исследования, начатые со скоростью, эквивалентной 20ng ангиотензина II на кг в минуту и скорректированы в течение первых 3 часов , чтобы увеличить MAP, по крайней мере 75mmHg Максимальная Infusion infusion research angiotensin II During the adjustment of the study infusion, Standard vasopressors can only be adjusted for safety reasons If vasopressors have been adjusted, the patient has been deemed map irresponsible by MAP response determined with average triple MAP measurements performed in 2 hours 45 minutes, 3 hours, and 3 hours 15 minutes after 3 hours and 15 minutes, drug or placebo study and other vasopressors have been adjusted to maintain the target MAP 65-75 mm. Art. Angiotensin II or placebo can be adjusted for an infusion course equivalent to 1.25-40ng/kg/min angiotensin II in an hour 48, The study's infusion was discontinued in accordance with the protocol-indicated cone If the background dose of vasopressor was subsequently increased to more than 0.1ug/kg/min equivalent of norephrine or the patient's condition became unstable, the drug study could be resumed for a period of time up to 7 Days If the drug study has been discontinued within 48 hours, it may not be renewed under any circumstances By Comparison Results of Angiotensin II vs. Placebo Primary Results MAP Response (3 Hours) 114 (69.9%) 37 (23.4%) (OR 7.95, p'lt;0.001) Secondary results Average cardiovascular SOFA score (48 hours) -1.75 vs.-1.28 (p'0.01) Average change in the overall SOFA score (48 hours) 1.05 against. 1.04 p0.49 Average dose change equivalent to norarenal (3 hours) -0.03 vs. 0.03 (p'lt;0.001) All causes of death (7 days) 47 (29%) 55 (35%) (OR 0.78, 95% CI 0.53-1.16, p. 0.22) All causes of death (28 days) 75 (46%) 85 (54%) (OR 0.78, 95% CI 0.57-1.07, p. 0.12) Adverse Events Adverse Events 142 (87.1%) 145 (91.8%) Serious adverse events 99 (60.7%) 106 (67.1%) Criticism of the small size of the study limits the power to identify differences in clinical outcomes, including Mortality Short Duration Of Subsequent Limitations ability to draw conclusions regarding the long-term efficaine and safety of angiotensin II Drug Titratation Period Study may allow unintentional unblinding (which has not been evaluated) by providers due to the observed response in MAP, although this is somewhat mitigated by the MAP response in 1/4 patients receiving a placebo Funding Study supported by La Jolla Pharmaeuticals Authors with multiple industry links Further reading Reading

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