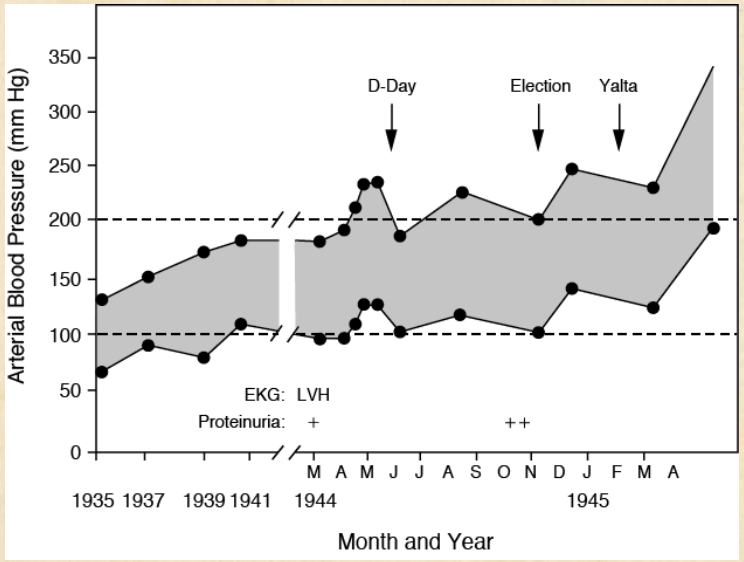
Неотложные состояния при артериальной гипертонии и преэклампсии

Профессор
Е.М. Шифман

Гипертензия беременных



The NEW ENGLAND JOURNAL of MEDICINE

OCCASIONAL NOTES

THIS DAY 50 YEARS AGO

THE headlines of April 13, 1945, stunned the nation and the world. Franklin D. Roosevelt, 32nd president of the United States, had died in Warm Springs, Georgia, the day before. Presumably, he had been in excellent health, there was no indication of imminent danger, and as Admiral Ross McIntire, the president's personal physician, asserted, the cerebral hemorrhage "came out of the clear sky" (Fig. 1).[1] Steve Early, press secretary for the White House, stated officially that "the President was given a thorough examination by seven or eight physicians, including some of the most eminent in the country, and was pronounced organically sound in every way."[1]

However, scrutiny of Roosevelt's history and physical findings (Fig. 2) reveals that these headlines either were a smoke screen or reflected the ignorance of some of the president's attending physicians. As recorded in the personal notes of Dr. Howard G. Bruenn,[2] the cardiologist who cared for Roosevelt during the last year of his life, FDR's blood pressure was 136/78 mm Hg in 1935, 162/98 mm Hg two years later, and 188/105 mm Hg by 1941. By March 1944, target-organ disease was evident — left ventricular hypertrophy on an electrocardiogram, cardiac enlargement on chest film, and proteinuria. Shortly before the invasion of Normandy, FDR's recorded blood pressure reached 226/118 mm Hg (Fig. 2). Throughout the balance of 1944, the president's blood pressure remained high; it was recorded as being over 200/100 mm Hg at the time of his reelection in November 1944. Before the Yalta conference in February 1945, Dr. Bruenn recorded values of 260/150 mm Hg. On the morning of April 12, 1945, while being sketched by Nicholas Robbins, a New York artist, FDR reported a "terrific" occipital headache[3] and lost consciousness immediately afterward. Fifteen minutes later, Dr. Bruenn recorded a blood pressure of more than 300 mm Hg systolic and 190 mm Hg diastolic. The president was pronounced dead at 3:35 p.m.

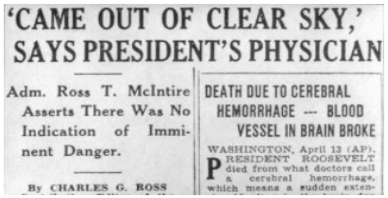
Even from these sparse clinical notes, it is obvious that over a period of only 10 years, FDR had progressively severe hypertension that ultimately entered a malignant phase, leading to a fatal cerebral hemorrhage. During his 1944 radio addresses, short-windedness was occasionally audible, probably reflecting some degree of congestive heart failure. Unfortunately, the president's original chart, which was kept in a safe at the U.S. Naval Hospital in Bethesda, Maryland, vanished immediately after his death, never to be found again. Thus, the only available data are Dr. Bruenn's notes.

In retrospect, it seems unlikely that FDR had essential hypertension. It is unusual for this disorder to appear for the first time at the age of 54 (Roosevelt's age in 1936) and to progress to a malignant phase in less than 10 years. Some form of renovascular disease more readily accounts for this sequence of events or may at least have accelerated the course of essential hypertension. The president was a heavy smoker, and smoking has been identified as a powerful risk factor for renovascular hypertension. Although no autopsy was performed, the embalmers noted that "the arteries were so severely clogged with plaques that the pump [serving to inject formaldehyde] strained and stopped."[4] Indeed, the embalmers had to inject successively the carotids, then the axillaries, and finally the femoral arteries.[4] Thus, there is no doubt that FDR had quite severe and extensive arteriosclerotic disease, and it seems likely that renovascular hypertension, alone or superimposed on essential hypertension, accelerated his death. Because of the severe arteriosclerotic disease, some degree of pseudohypertension may also have contributed to the extremely high blood-pressure values.[5]

The fact that as late as 1945 hypertension was not considered a disease of major clinical consequence should not come as a surprise. It was still viewed by the majority of physicians as "essential" to force blood through sclerotic arteries to the target organs. In fact, Dr. Paul Dudley White noted in his famous 1931 textbook on heart disease,

> The treatment of the hypertension itself is a difficult and almost hopeless task in the present state of our knowledge, and in fact for aught we know . . . the hypertension may be an important compensatory mechanism which should not be tampered with, even were it certain that we could control it.[6]

Given this view, it is possible that some of FDR's physicians may have misjudged the severity of his condition and that the news reports attesting to his good health may not have been merely fabricated for political reasons. Although Dr. Bruenn (a very capable cardiologist) followed FDR closely during the last year of his life, Admiral McIntire (an ear, nose, and throat specialist) relayed all reports to the media. Asked for a "definite statement" on the president's health, McIntire said, "His present health is excellent. I can say that unquali-



'CAME OUT OF CLEAR SKY,' SAYS PRESIDENT'S PHYSICIAN

Adm. Ross T. McIntire Asserts There Was No Indication of Imminent Danger.

By CHARLES G. ROSS

DEATH DUE TO CEREBRAL HEMORRHAGE — BLOOD VESSEL IN BRAIN BROKE

WASHINGTON, April 13 (AP). PRESIDENT ROOSEVELT died from what doctors call a cerebral hemorrhage, which means a sudden exten-

Figure 1. Headlines of the *St. Louis Post-Dispatch*, April 13, 1945.
Reprinted with the permission of the *St. Louis Post-Dispatch*.

Messerli F. H. This day 50 years ago. N Engl J Med. 1995 Apr 13;332(15):1038–1039.

A British Medical Association Lecture

ON

THE SIGNIFICANCE OF A RAISED BLOOD PRESSURE*

BY

JOHN HAY, M.D., F.R.C.P.

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My subject is one of very general interest and also of considerable practical importance, if for no other reason than that a large number of our patients at or over middle age present a raised blood pressure. No one can now afford to be indifferent to the problems associated with variations in blood pressure, for a high pressure is an abnormality which always demands investigation, supervision, and careful treatment. There is a danger that patients may take the variations in their blood pressure

The Diastolic Pressure

In Great Britain the diastolic pressure is usually taken as that point at which there is a sudden marked diminution in the intensity of the sounds on auscultation of the brachial artery—normally about 70 to 80 mm. Hg. An increase in diastolic pressure signifies that with each systole a greater expenditure of energy is required to force open the aortic valves. The permanent load on the heart and arteries is greater than normal. The result is an increase in the size and power of the left ventricle, and it is this strain which may be ultimately responsible for the cardiac failure. The end-result of persistent increase in the diastolic pressure is cardiac defeat. The diastolic pressure is increased by any cause which augments peripheral resistance, either vaso-constriction or actual pathological changes in the arterioles, and it is so intimately related to the elasticity of the arterial walls that it is worth while to refer to this in a little more detail.

"Наибольшая опасность для человека с высоким давлением кроется в выявлении последнего, поскольку потом какой-нибудь дурак уверенно попытается и снизит его."

- *Hay, Brit. Med. J. 1931*

Дерзкие кардиологи...

GLIMPSES

A Glimpse at Dr William Evans (1895–1988)

'Willie' Evans was a great teacher. Early in life he realized the importance of teaching in medical education and he drew up a list of requirements of a good lecture that should always be fully prepared. As a result, he was a very popular lecturer.

He visited America in 1954 and, among other places, lectured in Evanston, Illinois, and gave the Garrish Milliken Lecture in Philadelphia. He also lectured in London, Scotland, Ireland, Stockholm, Copenhagen, Brussels, Paris, Rome and Montreal, to name but a few places (Figures 1 and 2). He visited Australia and received the Sydney Gold Medal for work in cardiology in 1954. He told amusing stories of his travels in his 1964 autobiography *Journey to Harley Street*.[1]

He was also a natural research worker and gained pleasure in putting his thoughts on paper. He wrote five books and some 100 scientific publications. Sometimes these were written with a colleague and they covered the whole of cardiology, including electrocardiography, auscultation and cardioscopy. I shall always be grateful to him for starting me on writing professional papers. He showed great courage in opposing the view, generally accepted, that anticoagulants were indicated in cardiac infarction – he called them rat poisons – but he was not always right! He believed that hypertension was harmless, cardiac catheterisation would soon be abandoned and the electrocardiogram was always abnormal with cardiac pain. However, with Clifford Hoyle he was the first to use the controlled trial on the Comparative Value of Drugs used in the

Figure 1 Dr William Evans; last teaching session in the Bearsted Lecture Theatre, The London Hospital, 1973 (reproduced courtesy of The Royal London Hospital Archives)

Figure 2 Dr Williams Evans, last teaching session in the Bearsted Lecture Theatre, The London Hospital, administers Trasitrin to Dr Richard Bomford, 1973 (reproduced courtesy of The Royal London Hospital Archives)

Continuous treatment of Angina Pectoris and he coined the term 'The Placebo Effect'. His publications were collected and annotated in 1990 in a biography entitled *A Rare Hero – Dr William Evans* by Buddug Owen.[2] This includes various quotations of Evans that made his name well known in medical circles: 'No patient should be worse for seeing a doctor'; 'Better Health than Wealth'. He was a religious man but remained a lorer and difficult to get to know. He did not receive any honour – it is possible he refused one, for he certainly deserved one. I think that his writings were so ahead of his time that much of what he said was not accepted by his contemporaries and indeed he was not always right, which made him enemies among his colleagues. He would make statements (and teach) about ideas that were quite new and uncertain. He would say 'if that is wrong I shall soon hear about it'. Although he made it clear that he was not sure about some of these ideas, they were quoted by his juniors and this upset some of his colleagues.

Acknowledgements: I wish to thank Jonathan Evans, Archivist of The Royal London Hospital, Malcolm Towers and Josephine Viney.

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DOI: 10.1258/jmb.2009.009081

References and notes

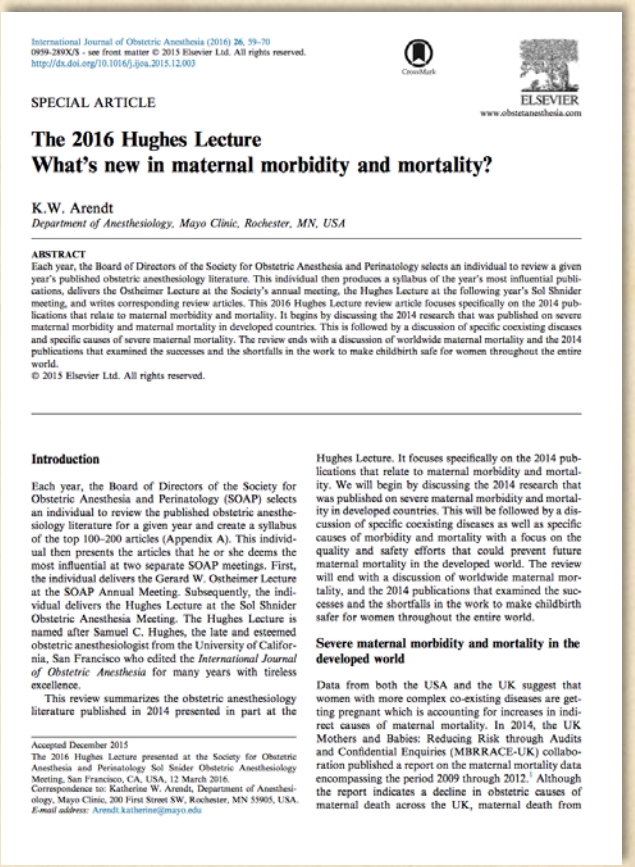
1 Evans W. *Journey to Harley Street*. London: David Rendel Ltd, 1968
2 Owen B. *A Rare Hero – Dr William Evans*. Denbigh: Gee & Sons Ltd, 1999

Уильям Эванс, шеф кардиологии,
Лондонский госпиталь, 1940
Письмо другу:

"...я не могу не презирать любого, кто переживает по поводу болезни, которая является плодом воображения. У тебя гипертония (если действительно твое давление эпизодически поднималось до 230/130), что является нормальным физиологическим состоянием, и не трансформировалось в свое время в патологическое состояние артериальной гипертонии. Поэтому, ради Бога, перестань беспокоиться о том, что не должно, но делает тебя несчастным."

У женщин с хронической артериальной гипертонией увеличивается риск:

- **Преэклампсия (уровень риска)**
  (RR 7,7; 95% CI 5,7–10,1)
- **Перинатальная смерть**
  (RR 4,2, CI 2,7–6,5)
- **Вес при рождении < 2500 г**
  (RR 3,2, CI 2,2–4,4)
- **Поступление новорожденного в отделение интенсивной терапии**
  (RR 3,2, CI 2,2–4,2)
- **Преждевременные роды < 37 недель**
  (RR 2,7, CI 1,9–3,6)
- **Кесарево сечение**
  (RR 1,3, 95% 1,1–1,5)



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http://dx.doi.org/10.1016/j.ijoa.2015.12.003

SPECIAL ARTICLE

ELSEVIER
www.obstetanesthesia.com

The 2016 Hughes Lecture
What's new in maternal morbidity and mortality?

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ABSTRACT
Each year, the Board of Directors of the Society for Obstetric Anesthesia and Perinatology selects an individual to review a given year's published obstetric anesthesiology literature. This individual then produces a syllabus of the year's most influential publications, delivers the Ostheimer Lecture at the Society's annual meeting, the Hughes Lecture at the following year's Sol Shnider meeting, and writes corresponding review articles. This 2016 Hughes Lecture review article focuses specifically on the 2014 publications that relate to maternal morbidity and mortality. It begins by discussing the 2014 research that was published on severe maternal morbidity and maternal mortality in developed countries. This is followed by a discussion of specific coexisting diseases and specific causes of severe maternal mortality. The review ends with a discussion of worldwide maternal mortality and the 2014 publications that examined the successes and the shortfalls in the work to make childbirth safe for women throughout the entire world.

Introduction

Each year, the Board of Directors of the Society for Obstetric Anesthesia and Perinatology (SOAP) selects an individual to review the published obstetric anesthesiology literature for a given year and create a syllabus of the top 100–200 articles (Appendix A). This individual then presents the articles that he or she deems the most influential at two separate SOAP meetings. First, the individual delivers the Gerard W. Ostheimer Lecture at the SOAP Annual Meeting. Subsequently, the individual delivers the Hughes Lecture at the Sol Shnider Obstetric Anesthesia Meeting. The Hughes Lecture is named after Samuel C. Hughes, the late and esteemed obstetric anesthesiologist from the University of California, San Francisco who edited the *International Journal of Obstetric Anesthesia* for many years with tireless excellence.

This review summarizes the obstetric anesthesiology literature published in 2014 presented in part at the Hughes Lecture. It focuses specifically on the 2014 publications that relate to maternal morbidity and mortality. We will begin by discussing the 2014 research that was published on severe maternal morbidity and mortality in developed countries. This will be followed by a discussion of specific coexisting diseases as well as specific causes of morbidity and mortality with a focus on the quality and safety efforts that could prevent future maternal mortality in the developed world. The review will end with a discussion of worldwide maternal mortality, and the 2014 publications that examined the successes and the shortfalls in the work to make childbirth safer for women throughout the entire world.

Severe maternal morbidity and mortality in the developed world

Data from both the USA and the UK suggest that women with more complex co-existing diseases are getting pregnant which is accounting for increases in indirect causes of maternal mortality. In 2014, the UK Mothers and Babies: Reducing Risk through Audits and Confidential Enquiries (MBRRACE-UK) collaboration published a report on the maternal mortality data encompassing the period 2009 through 2012.[1] Although the report indicates a decline in obstetric causes of maternal death across the UK, maternal death from

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The 2016 Hughes Lecture presented at the Society for Obstetric Anesthesia and Perinatology Sol Snider Obstetric Anesthesiology Meeting, San Francisco, CA, USA, 12 March 2016.
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E-mail address: Arendt.katherine@mayo.edu

Следует отметить, что в описанной чистоте присутствовала гетерогенность по исходам (r = 0,286–0,766)

K.W. Arendt. The 2016 Hughes Lecture. What's new in maternal morbidity and mortality? International Journal of Obstetric Anaesthesia 2016; 26:59–70

Гипертонический криз – терминология и определения

Внезапный подъем АД
ДАД >115–130 мм рт. ст.
Сист.АД > 180–120 мм рт. ст.

Беременность > 169/109
"важен относительный подъем"

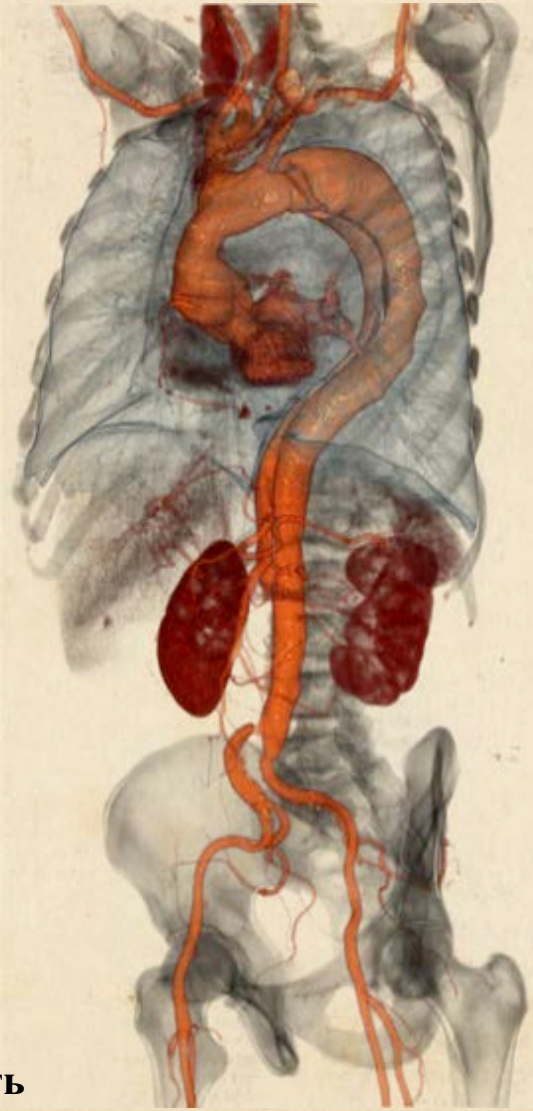
срочное состояние
при артериальной гипертонии:
значимый подъем АД без острого поражения органов
(но с высоким риском такого поражения)

экстренное состояние
при артериальной гипертонии:
острое поражение органов и систем:
ЦНС, почки, сердце.

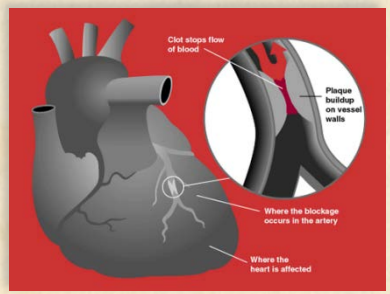
Экстренное гипертоническое состояние

Инсульт
Энцефалопатия

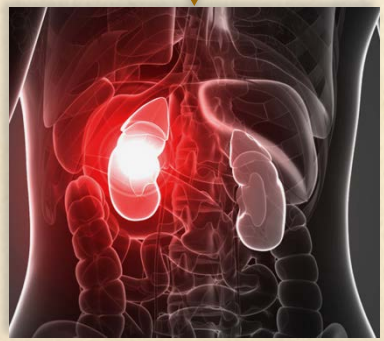
Диссекция аорты



Декомпенсированная сердечная недостаточность



Острый коронарный синдром



Острая почечная недостаточность

Пациентка в ясном сознании?

да ↓ | *нет* ↓

Наличие очаговой неврологической симптоматики

↓ *нет* → *есть* **Это новый симптом?** *да* ↓ | *нет* ↓

Есть отек на глазном дне?

↓ *нет*

Есть на глазном дне геморрагии или экссудаты?

↓ *нет* → *да* **Это новый симптом?** *да* ↓ | *нет* ↓

Есть признаки ишемии на ЭКГ?

↓ *нет* → *да* **Это новый симптом?** *да* ↓ | ↓

Большое количество эритроцитов в моче

↓ *нет*

Креатинин сыворотки повышен? → *да* **Это новый симптом?** *да* ↓ | ↓

↓ *нет*

Это не экстренное гипертоническое состояние

Это новый симптом? *да* ↓ | ↓

Это экстренное гипертоническое состояние

Препарат второй очереди — Нифедипин

- **Нифедипин никогда не следует давать под язык женщине с гипертензией. Нифедипин доступен для приёма внутрь в 3-х видах: капсулы, таблетки в высвобождением действующего вещества в течение 12 часов и в течение 24 часов. Следует внимательно свериться с инструкцией перед назначением препарата.**
- **Капсулы нифедипина (10 мг) "Дозы могут быть повторными, через 4–6 часов по необходимости. Возможно развитие глубокой гипотонии при одновременном назначении нифедипина и парентеральном введении магнезии ==> следует назначать нифедипин с осторожностью.**
- **Формы с постепенным высвобождением действующего вещества (12 часов), например, адалат-ретард, можно рассматривать как средство для длительной поддержки**

Для никардипина определен кардиопротективный эффект при отсутствии ухудшения маточно-плацентарного кровотока и состояния плода

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Hemodynamic effects of intravenous nicardipine in severely pre-eclamptic women with a hypertensive crisis

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KEYWORDS: echocardiography; hemodynamics; hypertensive crisis; nicardipine; pre-eclampsia

ABSTRACT

Objective Nicardipine permits rapid control of blood pressure in women with severe pre-eclampsia (PE) and hypertensive crisis. Our objective was to investigate its maternal and fetal hemodynamic effects.

Methods Ten severely pre-eclamptic pregnant women who required intravenous nicardipine for severe hypertension were included in this prospective observational trial. Maternal macrocirculation was assessed by transthoracic echocardiography. Maternal microcirculatory perfusion was examined sublingually with the sidestream dark field imaging technique. Fetal hemodynamics were assessed by Doppler examinations of the uteroplacental and fetal circulations. Maternal cardiac output, total vascular resistance, mitral E/A ratio and capillary heterogeneity index, uterine artery pulsatility index and fetal cerebroplacental ratio were considered primary outcomes. Paired measurements, obtained before administration of nicardipine infusion and after stabilization of blood pressure, were compared.

Results Administration of nicardipine significantly reduced the mean arterial blood pressure (median difference, 26 mmHg; P = 0.002) and total vascular resistance (median difference, 791 dynes × sec^{-1}; P = 0.002) in all included women. This induced a reflex tachycardia with consequent increase in cardiac output of 1.55 L/min (P = 0.004). There were no significant changes in the other determinants of maternal or fetal hemodynamic parameters.

Conclusions Nicardipine effectively reduces blood pressure through selective afterload reduction that triggers an increase in cardiac output, without affecting maternal diastolic function, or microcirculatory, uteroplacental or fetal perfusion. This hemodynamic response is uniform and predictable. Feto-maternal cardiovascular profiling can be achieved by combining transthoracic echocardiography with obstetric Doppler. Copyright © 2015 ISUOG. Published by John Wiley & Sons Ltd.

INTRODUCTION

A hypertensive crisis, defined as the occurrence of a systolic blood pressure (SBP) ≥ 160 mmHg and/or diastolic blood pressure (DBP) ≥ 110 mmHg in women with pre-eclampsia (PE), is a hypertensive emergency[1,2]. These women are at risk of developing complications such as cerebrovascular accidents and pulmonary edema[3–4]. Their blood pressure must be lowered rapidly without compromising the maternal or uteroplacental circulations. Nicardipine is a calcium channel blocker structurally related to nifedipine but with a distinctive pharmacological and hemodynamic profile that makes it attractive for the treatment of hypertensive emergencies in women with PE[7–10]. Its administration as intravenous form, rapid onset of action and short half-life allow easy titration against blood pressure while transplacental passage is limited (15%)[11–13]. Nicardipine induces general arterial relaxation that is more pronounced in cerebrovascular and coronary arteries[9,14,15]. The depressant action on myocardial muscle cells is less than with nifedipine and its cerebrovascular selectivity renders it more effective in preventing ischemic stroke and hypertensive brain damage than other antihypertensive drugs[16]. Results from observational and comparative trials in women with severe PE are encouraging[10,17–23]. Nicardipine seems equivalent or superior in reducing blood pressure to other intravenous drugs that are used commonly (labetalol, ketanserin, hydralazine), with excellent maternal and

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ORIGINAL PAPER

ULTRASOUND
in Obstetrics & Gynecology

Original Paper

Hemodynamic effects of intravenous nicardipine in severely pre-eclamptic women with a hypertensive crisis

J. Cornette[1,*], E. A. B. Buijs[2], J. J. Duvekot[1], E. Herzog[1], J. W. Roos-Hesselink[3], D. Rizopoulos[4], M. Meima[5] and E. A. P. Steegers[1]

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Issue

Ultrasound in Obstetrics & Gynecology

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- *J. Cornette, E. A. B. Buijs, J. J. Duvekot, E. Herzog, J. W. Roos-Hesselink, D. Rizopoulos, M. Meima and E. A. P. Steegers. Hemodynamic effects of intravenous nicardipine in severely pre-eclamptic women with a hypertensive crisis Ultrasound Obstet Gynecol 2016; 47: 89–95.*

- В когортное исследование были включены **2 292 116** беременностей, закончившихся рождением живых младенцев. Анализировались риски неонатальной гипогликемии и брадикардии, ассоциированные с использованием во время родов β-блокаторов.

- Во время **10 585** родов **(0,5%)** были использованы β-блокаторы. Самым часто назначаемым препаратом был *лабеталол* **(n=6748)**, затем следовал метопролол **(n=1485)** и *атенолол* **(n=1121)**.

- **Результаты широкого когортного исследования свидетельствуют о повышении риска неонатальной гипогликемии и брадикардии на фоне применения β-блокаторов во время родов.**

PEDIATRICS®
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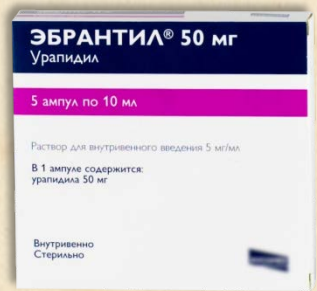
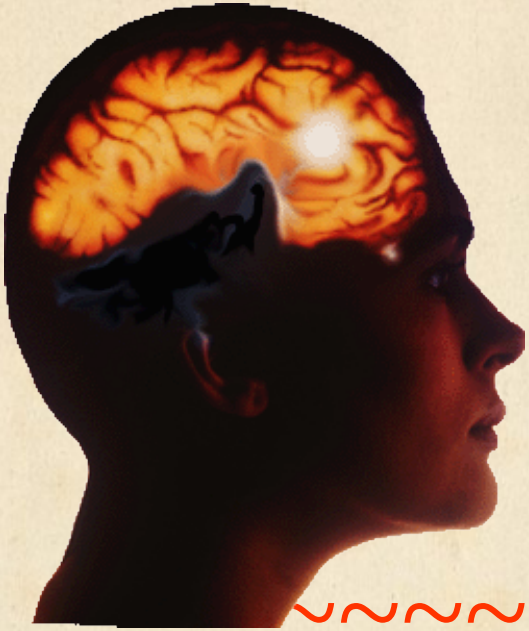
September 2016, VOLUME 138 / ISSUE 3

BrianT. Bateman, Elisabetta Patorno, Rishi J. Desai, Ellen W. Seely, Helen Mogun, Ayumi Maeda, Michael A. Fischer, Sonia Hernandez-Diaz, Krista F. Huybrechts. Late Pregnancy Blocker Exposure and Risks of Neonatal Hypoglycemia and Bradycardia. Pediatrics 2016; 138(3): e20160731

В большинстве исследований урапидил сравнивается с дигидралазином, т. к. последний в течение длительного времени (около 40 лет) в Европе был «золотым стандартом» антигипертензивной терапии при преэклампсии



Основная причина эклампсии



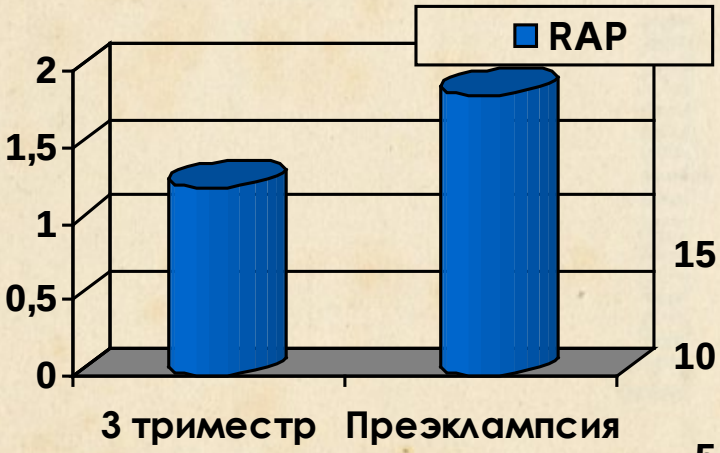
~~~~~ **спазм** ~~~~~

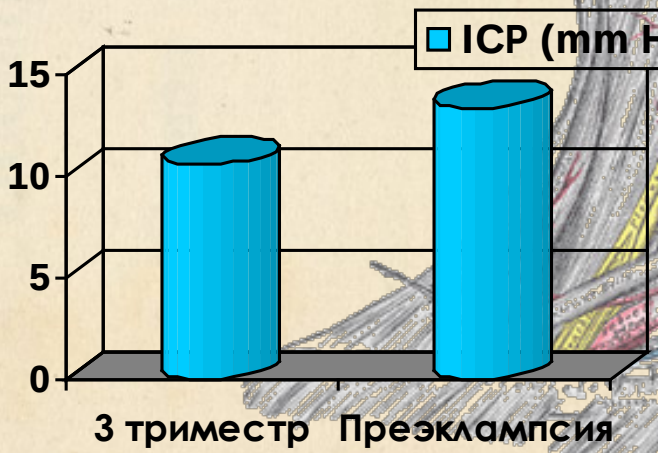
сосудов головного мозга
~~~~~

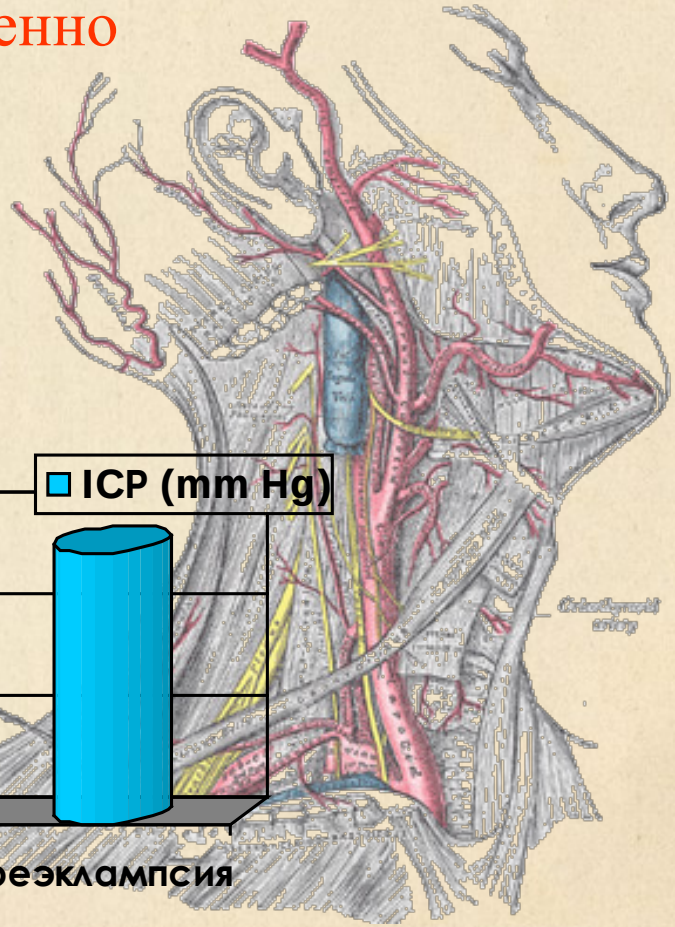
ПМ = САД – ВЧД

Результаты исследования

- уровень гидродинамического сопротивления и внутричерепного давления в группе пациентов с тяжелой преэклампсией существенно выше, чем в группе сравнения







Признаки повышенного ВЧД

- *Клинические признаки:*
  - ✓ Изменения зрачков или их асимметрия
  - ✓ Глазодвигательные нарушения
  - ✓ Отек диска зрительного нерва
  - ✓ Гемипарез
  - ✓ Слабость мышц лица
  - ✓ Новый приступ судорог
  - ✓ Сниженный уровень сознания (Расстройства сознания)
- *Радиологические признаки на КТ или МРТ:*
  - ✓ Напряжённая (натянутая) твёрдая мозговая оболочка
  - ✓ Сглаженные извилины
  - ✓ Сужение борозд
  - ✓ Стертость цистерн
  - ✓ Компрессия (или при обструкции расширение) желудочков
  - ✓ Смещение структур относительно срединой линии
  - ✓ Смещение ткани мозга из одного отдела в другой

Кровь

Оксигемоглобин → НН → НО·

Метгемоглобин

ЦН⁻ ЦН⁻ ЦН⁻ ЦН⁻ ЦН⁻

Цианиды

Цианметгемоглобин

ЦН

ЦО

Метгемоглобин

ЦО

ЦН⁻

Цитохромоксидаза

(ЦО)

Печень

Тиосульфат

ЦН⁻

Роданаза

Тиоцинат

ЦН⁻ Тиоцинатоксидаза

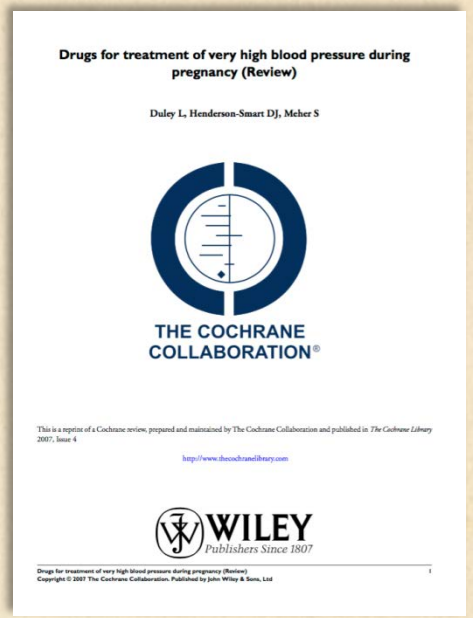
Тиоцинат

(кровь)

моча

Обзоры и мета-анализы

Drugs for treatment of very high blood pressure during pregnancy (Review)

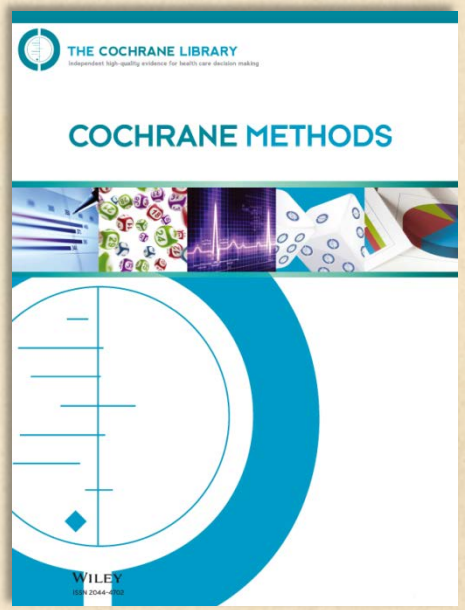


КОХРЕЙНОВСКОЕ СОТРУДНИЧЕСТВО

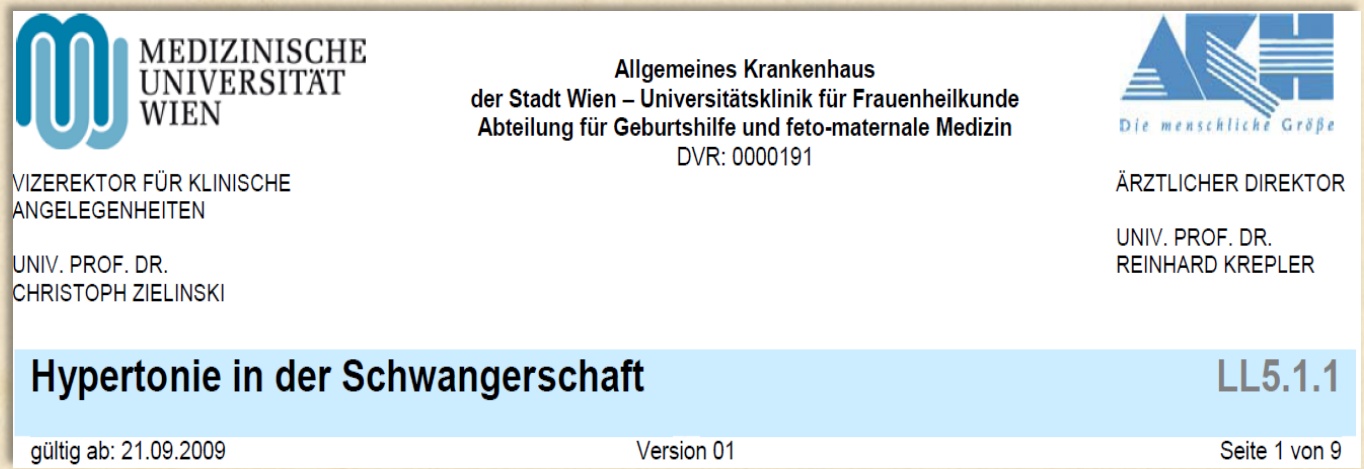
Препараты для лечения очень высокого давления при беременности (обзор)

Урапидил достоверно лучше дигидралазина по следующим конечным точкам:

- **чрезмерная гипотензия,**
- **отслойка плаценты,**
- **младенческая смертность**



Клинический протокол, Австрия



MEDIZINISCHE UNIVERSITÄT WIEN

VIZEREKTOR FÜR KLINISCHE ANGELEGENHEITEN

UNIV. PROF. DR. CHRISTOPH ZIELINSKI

Allgemeines Krankenhaus
der Stadt Wien – Universitätsklinik für Frauenheilkunde
Abteilung für Geburtshilfe und feto-maternale Medizin
DVR: 0000191

Die menschliche Größe

ÄRZTLICHER DIREKTOR

UNIV. PROF. DR. REINHARD KREPLER

Hypertonie in der Schwangerschaft LL5.1.1

gültig ab: 21.09.2009 Version 01 Seite 1 von 9

Гипертония при беременности

Антигипертензивная терапия:

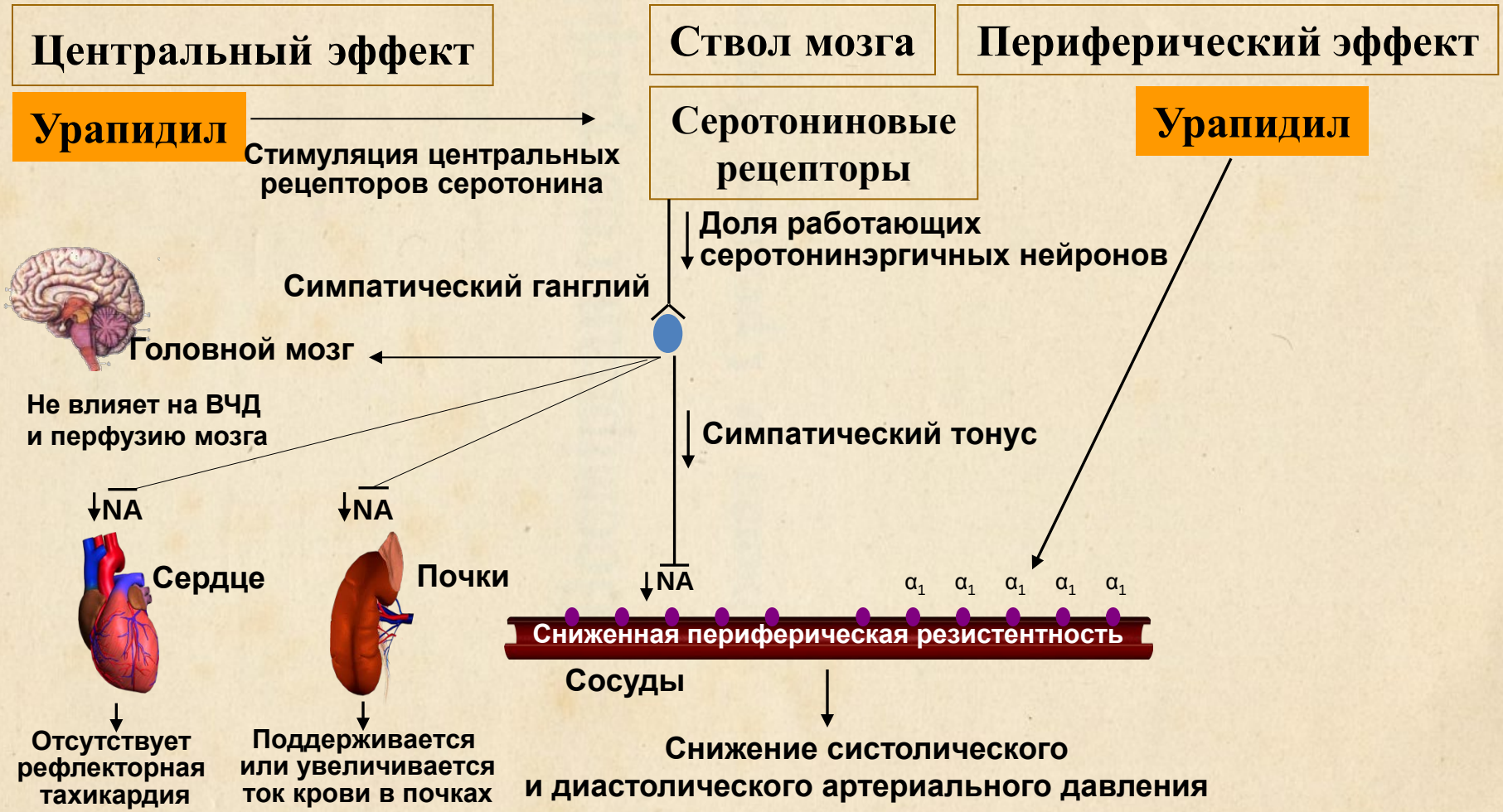
Первая линия – Эбрантил (урапидил):

- Рекомендуемый способ применения: с помощью перфузора 2 ампулы по 50 мг (10 мл) Эбрантила (урапидила HCl) на 30 мл 0,9% раствора NaCl = 50 мл
- Начальная доза: 100 мл/ч в первые 2 мин, + возможно, следующие 2 мин
- Поддерживающая доза: 5–25 мл/ч
- Максимальная доза: 50 мл/ч
- При достижении АД 170/110 мм рт. ст. – переход на пероральные препараты

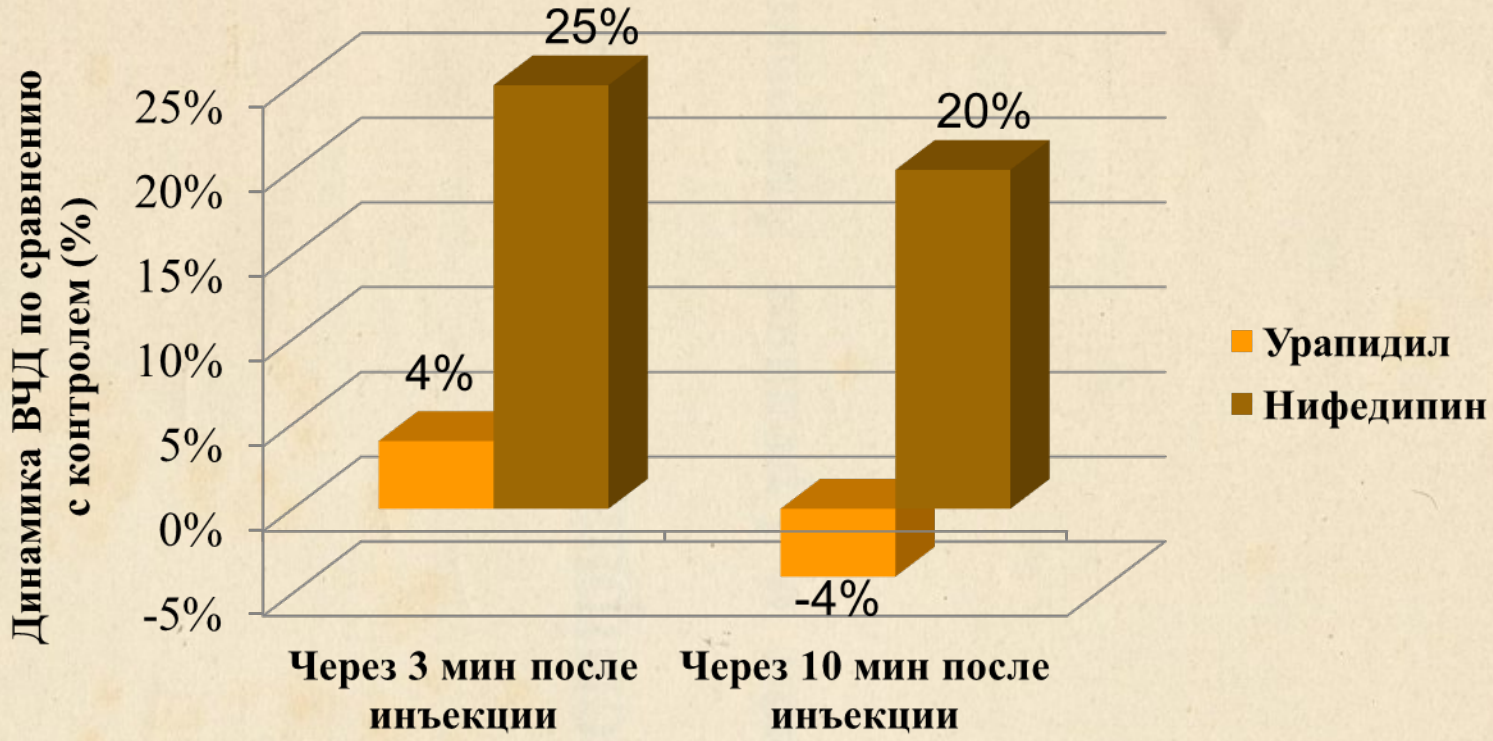
Пероральные препараты:

- После 20-й недели беременности – Эбрантил по 1 капсуле 30 мг 2 раза в день (максимальная доза – 180 мг/день)

Механизм действия Урапидила



Влияние на внутричерепное давление (ВЧД)



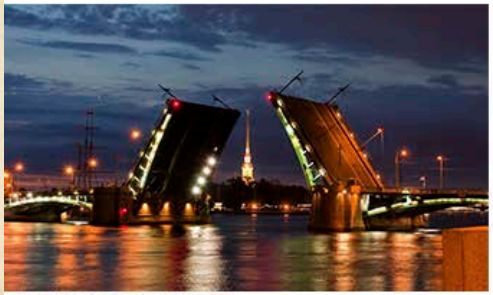
- Урапидил: в/в инъекция 2 мг/кг, затем инфузия 0,5 мг/кг
- Нифедипин: в/в инъекция 0,01 мг/кг, затем инфузия 0,002 мг/кг

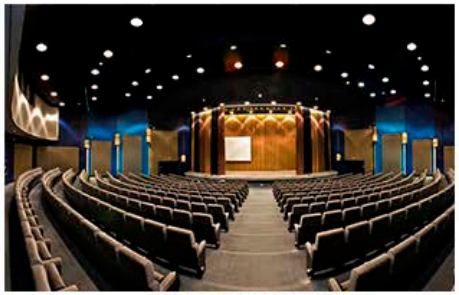
19-21 ОКТЯБРЯ 2016

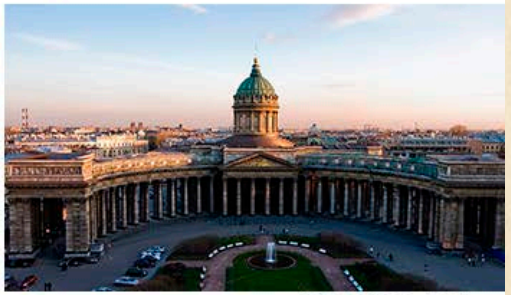
ВТОРОЙ СЪЕЗД

АССОЦИАЦИИ АКУШЕРСКИХ АНЕСТЕЗИОЛОГОВ-РЕАНИМАТОЛОГОВ

МЕСТО ПРОВЕДЕНИЯ: САНКТ-ПЕТЕРБУРГ, пл. Победы,1, ОТЕЛЬ park inn ПУЛКОВСКАЯ







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