



# PERIOPERATIVE MANAGEMENT OF HYPERTENSION IN NEUROSURGERY

Neuro-anesthesia Quiz # 79

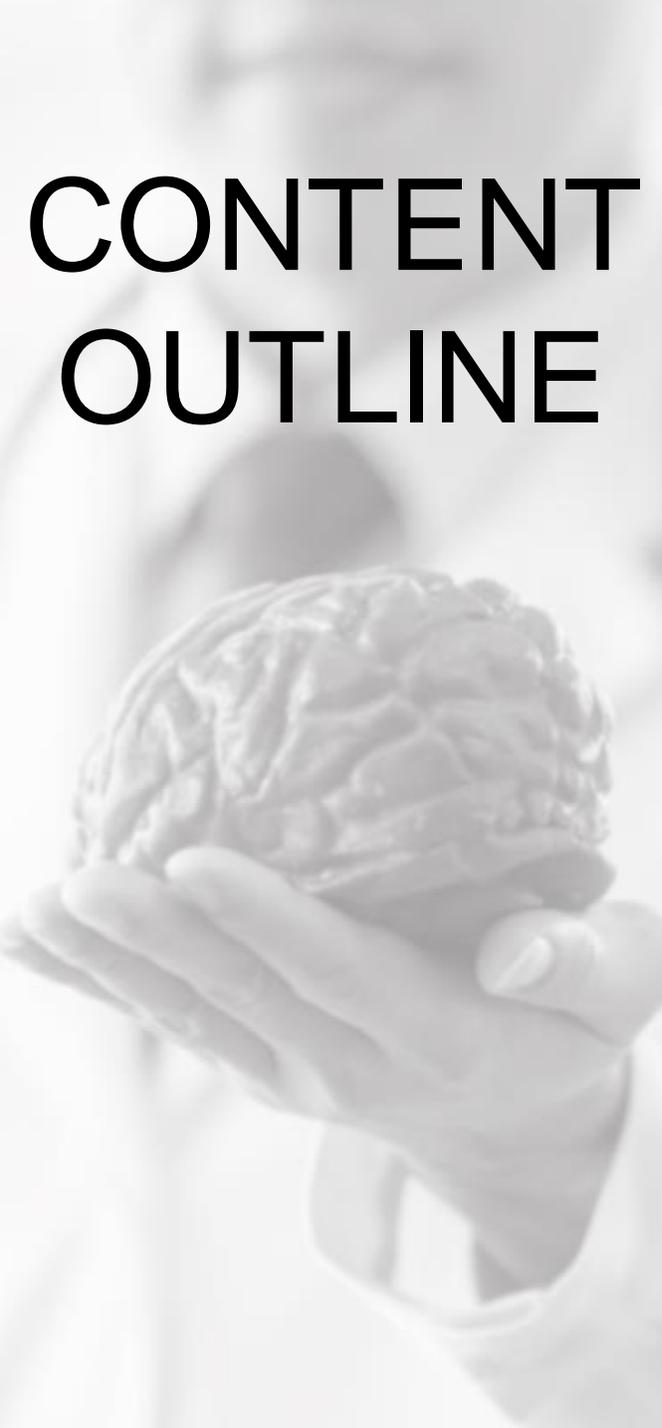
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# CONTENT OUTLINE

A grayscale image of a hand holding a human brain, positioned on the left side of the slide.

Please click on any of the following links to proceed to that question/topic.

[Question 1: Post-craniotomy hypertension](#)

[Question 2: Labetalol in craniotomy](#)

[Question 3: Nicardipine in Post- craniotomy hypertension](#)

[Question 4: Hypertension in DBS](#)

[Question 5: Management of hypertension for cerebral aneurysms](#)

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# QUESTION 1

A 40y male has a BP of 180/ 110 in the PACU after a posterior fossa craniotomy for tumor resection. Which of the following statements is **INCORRECT** about post-craniotomy emergence hypertension (PCEH)?

Please click on any of the following links to proceed to that question/topic.

[A. Pre- existing hypertension is a risk factor for PCEH](#)

[B. Alteration in cerebral autoregulation may be responsible for PCEH](#)

[C. Inadequate pain control is a key contributor of PCEH](#)

[D. Any rise in BP should be treated promptly with anti-hypertensives](#)

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# Sorry! Incorrect.

## EXPLANATION

A. Pre- existing hypertension is a risk factor for PCEH

This statement is correct

Patients with a previous diagnosis of hypertension have a significantly increased risk for PCEH and for requiring treatment with anti-hypertensives .

In a prospective study of Neurosurgical patients admitted to the ICU, Perez et al concluded that PCH was seen more often in patients with a prior history of hypertension (31.5%, 13.7% of the entire cohort), compared to those without a prior history of hypertension (12.7%, 7.0% of the entire cohort  $p < .001$ ). Pre-existing medical conditions such as hypertension and diabetes can lead to a higher rate of post-operative hypertension and post-operative complications, hence should be monitored carefully in the post-op period.

*Claudia A. Perez, et al, Elevated blood pressure after craniotomy: A prospective observational study, Journal of Critical Care, Volume 60, 2020, Pages 235-240, ISSN 0883-9441, <https://doi.org/10.1016/j.jcrc.2020.08.013>*

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# Sorry! Incorrect.

## EXPLANATION

B. Alteration in cerebral autoregulation may be responsible for PCEH

This statement is correct

Cerebral autoregulation is a negative feedback loop mechanism that counteracts the mean ABP increase by increasing vascular tone and narrowing the vessels diameter (thus increasing resistance of vessels) that brings CBF to the original level. Conversely, a decrease of ABP results in CBF diminishing whereas the regulatory mechanism decreases the vascular tone leading to vessels dilation to rebalance CBF.

Alterations of cerebral autoregulation and disruption of the cerebrovascular reflex in hypertensive patients may result in a disruption of the negative feedback loop, which induces an increase in blood pressure, and may have a higher incidence of postoperative hypertension because of an increased sensitivity to this phenomenon.

*Gal TJ, Cooperman LH. Hypertension in the immediate postoperative period. Br J Anaesth 1975; 47: 70–4.*

*Lyubashina Olga A., Mamontov Oleg V., Volynsky Maxim A., Zaytsev Valeriy V., Kamshilin Alexei A. Contactless Assessment of Cerebral Autoregulation by Photoplethysmographic Imaging at Green Illumination Frontiers in Neuroscience 13 :2019 ,1235*

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# Great Job!! Correct.

## EXPLANATION

C. Inadequate pain control is a key contributor of PCEH

This statement is INCORRECT

Pain does not appear to be an important factor in the development of postoperative hypertension. Although scalp infiltration with bupivacaine and epinephrine at end of craniotomy relieved postoperative pain, it did not have an influence on PCEH

*Bloomfield EL, Schubert A, Secic M, et al. The influence of scalp infiltration with bupivacaine on hemodynamics and postoperative pain in adult patients undergoing craniotomy. Anesth Analg 1998; 87: 579–82.*

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# Sorry! Incorrect.

## EXPLANATION

D. Any rise in BP should be treated promptly with anti-hypertensives

This statement is correct

Hypertension during emergence increases the tendency to bleed at the operative site by disrupting the hemostatic plugs, blocking autoregulation, and thus allowing the cerebral blood volume to increase leading to cerebral edema as the blood pressure increases.

Preemptive treatment with antihypertensives is, therefore, imperative to avert these potentially life-threatening events

*I.H. Kalfas, J.R. Little Postoperative hemorrhage: a survey of 4992 intracranial procedures Neurosurgery, 23 (1988), pp. 343-347*

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## QUESTION 2

During emergence after a bi-coronal frontal craniotomy, patient becomes tachycardic and hypertensive. IV labetalol is given. Which of the following statements is **INCORRECT** about labetalol?

Please click on any of the following links to proceed to that question/topic.

[A. Labetalol helps in preventing bleeding at emergence](#)

[B. Labetalol can worsen cerebral edema by increasing CBF](#)

[C. Labetalol counteracts the adverse effects of vasodilators](#)

[D. Labetalol has a prolonged duration of action](#)

# Sorry! Incorrect.

## EXPLANATION

A. Labetalol helps in preventing bleeding at emergence

This statement is correct

Post craniotomy hypertension increases the tendency to bleed at the operative site by disrupting the hemostatic plugs, blocking autoregulation, and thus allowing the cerebral blood volume to increase as the blood pressure increases.

Labetalol may help reduce the risk of bleeding by lowering blood pressure without influencing cerebral blood flow or cerebral autoregulation.

*K.S. Olsen, L.B. Svendsen, F.S. Larsen, O.B. Paulson Effect of labetalol on cerebral blood flow, oxygen metabolism and autoregulation in healthy humans Br J Anaesth, 75 (1) (1995), pp. 51-54*

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# Great Job!! Correct.

## EXPLANATION

**B. Labetalol can worsen cerebral edema by increasing CBF**

This statement is incorrect

Vasogenic edema usually occurs with PCEH due to the the leakage of fluid through a blood-brain barrier damaged by surgical intervention Labetalol does not influence cerebral blood flow, cerebral autoregulation or cerebral metabolic oxygen consumption, hence would reduce the risk of edema.

- *K.S. Olsen, L.B. Svendsen, F.S. Larsen, O.B. Paulson Effect of labetalol on cerebral blood flow, oxygen metabolism and autoregulation in healthy humans Br J Anaesth, 75 (1) (1995), pp. 51-54*

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# Sorry! Incorrect.

## EXPLANATION

C. Labetalol counteracts the adverse effects of vasodilators

This statement is correct

Labetalol, with its alpha and beta blockade can counteract the adverse effects of tachycardia, preventing increase in cerebral blood flow leading to increased ICP associated with vasodilators like nicardipine. This also allows reduced doses of vasodilators to prevent the PCEH.

*W. Frank Peacock, et al, A systematic review of nicardipine vs labetalol for the management of hypertensive crises, The American Journal of Emergency Medicine, Volume 30, Issue 6, 2012, Pages 981-993, ISSN 0735-6757,*

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# Sorry! Incorrect.

## EXPLANATION

D. Labetalol has a prolonged duration of action

This statement is correct

In contrast to agents possessing only beta-blocking activity, labetalol immediately and significantly lowers SVR by blocking alpha-adrenergic receptors with direct vasodilation, without significant reflex effects on HR because of its beta-adrenoceptor blockade. After an intravenous injection of labetalol, the hypotensive effect begins within 2 to 5 minutes and the full antihypertensive effect is apparent within 5-10 minutes. The plasma half-life of labetalol is approximately 3.5 to 4-5 hours, but the pharmacologic effect of this agent outlasts its plasma half-life.

*W. Frank Peacock, et al, A systematic review of nicardipine vs labetalol for the management of hypertensive crises, The American Journal of Emergency Medicine, Volume 30, Issue 6, 2012, Pages 981-993, ISSN 0735-6757,*

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## QUESTION 3

Which of the following statements is **INCORRECT** about using nicardipine in a morbidly obese patient after a right frontal meningioma resection?

Please click on any of the following links to proceed to that question/topic.

[A. Nicardipine has a favorable myocardial oxygen balance](#)

[B. There is an increased risk of cerebral edema](#)

[C. Nicardipine is very difficult to titrate](#)

[D. Nicardipine should be titrated carefully in hypoxic patients](#)

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# Sorry! Incorrect.

## EXPLANATION

A. Nicardipine has a favorable myocardial oxygen balance

This statement is correct

Morbidly obese patients like this patient are at increased risk of congestive heart failure which may complicate this patient's postoperative course.

A therapeutic benefit of nicardipine is to increase both stroke volume and coronary blood flow with a favorable effect on myocardial oxygen balance. This property is beneficial in patients with coronary artery disease and systolic heart failure.

Lambert CR, Hill JA, Nichols WW, et al. Coronary and systemic hemodynamic effects of nicardipine. *Am J Cardiol.* 1985;**55**:652–6

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# Sorry! Incorrect.

## EXPLANATION

B. There is an increased risk of cerebral edema

This statement is correct

Nicardipine causes a dose-dependent cerebral vasodilation and inhibition of autoregulation and as a result may lead to vasogenic edema consequent to the leakage of fluid through a blood-brain barrier damaged by surgical intervention.

*Kross, Ronald A. MD\*; Ferri, Enrico MD†; Leung, Denis PhD\*; Pratila, Margaret MD\*; Broad, Clara MD\*; Veronesi, Michele MD†; Melendez, Jose A. MD\* A Comparative Study Between a Calcium Channel Blocker (Nicardipine) and a Combined  $\alpha$ - $\beta$ -Blocker (Labetalol) for the Control of Emergence Hypertension During Craniotomy for Tumor Surgery, Anesthesia & Analgesia: October 2000 - Volume 91 - Issue 4 - p 904-909 doi: 10.1097/00000539-200010000-00024*

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# Great Job!! Correct.

## EXPLANATION

C. Nicardipine is very difficult to titrate.

This statement is incorrect

Intravenous nicardipine is rapidly distributed, binds strongly to plasma proteins, and is extensively metabolized, primarily in the liver. It has a rapid onset of action and a half-life of 40 to 60 minutes, making its administration easier to titrate when compared to antihypertensives like labetalol.

- *Bebawy et al Nicardipine is superior to esmolol for the management of post craniotomy emergence hypertension, a randomized open-label study. A and A, 2015;120(1):186-192)*
- *Varon J, Marik PE. The diagnosis and management of hypertensive crises. Chest. 2000;118:214–27.*

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# Sorry! Incorrect.

## EXPLANATION

D. Nicardipine should be titrated carefully in hypoxic patients

This statement is correct

Nicardipine is known to inhibit hypoxic pulmonary vasoconstriction. Nicardipine can induce a decrease in Pao<sub>2</sub> by altering ventilation/perfusion mismatch. The effects on arterial oxygenation depend on the relative influences of an increase in venous admixture resulting from the pulmonary vascular effects of the calcium entry blockers and the increase in mixed venous oxygen tension associated with the increase in cardiac output. The decrease in Pao<sub>2</sub> is prevented by improving oxygenation in hypoxemic patients.

*J.L. Vincent, G. Berlot, J.C. Presier, et al. Intravenous nicardipine in the treatment of postoperative arterial hypertension*

*J Cardiothorac Vasc Anesth, 11 (1997), pp. 160-164*

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## QUESTION 4

An 80-year-old patient is scheduled for right sided DBS electrode placement under MAC anesthesia. All are true about hypertension in DBS surgery, **EXCEPT?**

Please click on any of the following links to proceed to that question/topic.

[A. Patients may have pre-existing hypertension](#)

[B. Patients should hold their anti-hypertensive medications](#)

[C. Maintain blood pressure below 140 mm Hg systolic](#)

[D. Hypertension can occur during electrode placement](#)

# Sorry! Incorrect.

## EXPLANATION

### A. Patients may have pre-existing hypertension

This statement is correct

Patients with neurological diseases including PD, multiple sclerosis (MS), and essential tremors may present with autonomic dysfunction which may contribute to hemodynamic perturbations including wide swings in blood pressure and hypertension. There are other contributing factors including side effects of anti-Parkinson medications, anxiety, pain, and pre-existing hypertension, diabetes.

*Nicholson G, Pereira AC, Hall GM Parkinson's disease and anaesthesia. Br J Anaesth. 2002 Dec; 89(6):904-16.*

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# Great Job!! Correct.

## EXPLANATION

**B. Patients should hold their anti-hypertensive medications**

This statement is incorrect

DBS procedures are associated with significant cardiovascular instabilities leading to complications during and after the surgery. Chowdhary et al found that only the pre-operative blood pressure (SBP, DBP, and MAP) was shown to be significantly associated with this intraoperative risk. Hence it is recommended for the blood pressure in these patients to be stabilized in the peri-operative period and the patients should not be asked to hold their medications.

*Chowdhury, Tumul et al. "Hemodynamic Perturbations in Deep Brain Stimulation Surgery: First Detailed Description." *Frontiers in neuroscience* vol. 11 477. 28 Aug. 2017, doi:10.3389/fnins.2017.00477*

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# Sorry! Incorrect.

## EXPLANATION

C: Maintain blood pressure below 140 mm Hg systolic

This statement is correct

One of the most serious sequelae of DBS surgery is intracranial bleeding. Its incidence can vary from 0.5 to 5%. Age and hypertension were identified as risk factors for bleeding. Hence the BP should be maintained below 140 mm Hg with drugs like labetalol, nicardipine or clevidipine.

*Jain S, Ton TG, Perera S, Zheng Y, Stein PK, Thacker E, Strotmeyer ES, Newman AB, Longstreth WT Jr. Cardiovascular physiology in premotor Parkinson's disease: a neuroepidemiologic study. Mov Disord. 2012 Jul; 27(8):988-95.*

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# Sorry! Incorrect.

## EXPLANATION

### D. Hypertension can occur during electrode placement

This statement is correct

Procedures involving electrode placement and nuclei stimulation are commonly associated with hypertensive episodes. Patients may have an intracranial bleed while having hypertensive episodes during both the electrode placement and the stimulation phases. The mechanism of hypertension is not clearly understood yet; however, in a small case series the precise stimulation of periaqueductal gray matter incited cardiovascular changes including BP and HR changes. Similarly, the STN stimulation may also cause hemodynamic perturbations that include a rise in HR (25 bpm) and BP (20 mm Hg).

*Green AL, Hyam JA, Williams C, Wang S, Shlugman D, Stein JF, Paterson DJ, Aziz TZ. Intra-operative deep brain stimulation of the periaqueductal grey matter modulates blood pressure and heart rate variability in humans. Neuromodulation. 2010 Jul;13(3):174-81. doi: 10.1111/j.1525-1403.2010.00274.x. Epub 2010 Feb 9. PMID: 21992829.*

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## QUESTION 5

Which of the following statements is **INCORRECT** regarding perioperative management of hypertension in a cerebral aneurysm?

Please click on any of the following links to proceed to that question/topic.

[A. In an unsecured ruptured aneurysm systolic blood pressure should be below 160 mmHg](#)

[B. Periods of increased blood pressure is required during temporary clipping](#)

[C. Calcium channel blockers along with a beta blocker has good control of BP](#)

[D. Nitroprusside is best used in the postoperative period](#)

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# Sorry! Incorrect.

## EXPLANATION

A. In an unsecured ruptured aneurysm systolic blood pressure should be below 160 mmHg

This statement is correct.

The American Heart Association/American Stroke Association and the Neurocritical Care guidelines suggest keeping the mean arterial blood pressure below 110 mm Hg or systolic blood pressure below 160 mm Hg (or both) in the presence of ruptured unsecured aneurysm. The European guidelines suggest keeping the systolic blood pressure below 180 mm Hg. These parameters could change after aneurysm treatment per surgeon discretion.

*de Oliveira Manoel, A.L., Goffi, A., Marotta, T.R. et al. The critical care management of poor-grade subarachnoid haemorrhage. Crit Care 20, 21 (2016). <https://doi.org/10.1186/s13054-016-1193-9>*

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# Sorry! Incorrect.

## EXPLANATION

B. Periods of increased blood pressure is required during temporary clipping

This statement is correct.

In order to facilitate accurate placement of a permanent clip , a temporary clip is often placed on the parent vessel , which can cause a reduction in brain tissue oxygen tension and an increase in carbon dioxide tension. To prevent this and to ensure perfusion through collateral channels during temporary clipping, the blood pressure should be raised 10% to 20% or higher above the patient's baseline with coordination with the neuromonitoring personnel and neurosurgeon. Once the aneurysm is successfully secured, blood pressure can be normalized.

*Deepak Sharma; Perioperative Management of Aneurysmal Subarachnoid Hemorrhage: A Narrative Review. Anesthesiology 2020; 133:1283–1305 doi:*

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# Sorry! Incorrect.

## EXPLANATION

C. Calcium channel blockers along with a beta blocker has good control of BP

This statement is correct

The initial treatment goal in the context of aneurysm pathology is to decrease the pulsatile load or the aneurysmal wall stress (a function of change in pressure with time [ $dP/dT$ ]) with  $\beta$ -blockade. In the acute setting, only after effective control of  $dP/dT$  should one initiate vasodilator therapy. The sole use of a vasodilator may decrease blood pressure, but without  $\beta$ -blockade, the pulsatile load will actually increase, potentially leading to adverse events including bleeding.

*Khoynezhad A, Plestis KA. Managing emergency hypertension in aortic dissection and aortic aneurysm surgery. J Card Surg. 2006;21(suppl 1):S3-7*

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# Great Job!! Correct.

## EXPLANATION

### D. Nitroprusside is best used in the postoperative period

This statement is incorrect

Nitroprusside does help in preventing a rise in blood pressure, but it also increases CBF and hence may cause rebleeding and aneurysmal rupture

Intra-arterial nitroprusside has been shown to treat delayed cerebral vasospasm as an NO donor but may lead to an acute increase in cerebral blood flow after administration and worsening hyperemia and edema.

*Joshua B. Bederson, M.D. et al, Acute Vasoconstriction after Subarachnoid Hemorrhage, Neurosurgery, Volume 42, Issue 2, February 1998, Pages 352-362, <https://doi.org/10.1097/00006123-199802000-00091>*

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