November 2007 DAC Answers

Friday, November 30, 2007

Potential complication(s) of a stellate ganglion block include:

a. recurrent nerve paralysis
b. subarachnoid block
c. brachial plexus block
d. pneumothorax
e. esophageal perforation

Answer: all: a-e

Thursday, November 29, 2007

The most important determinant of resistance to laminar gas flow through a tube is:

a. length of the tube
b. radius of the tube
c. viscosity of the gas
d. density of the gas
e. mass of the gas

Answer: b.
The laminar flow of the gas through a tube is described by the Hagen-Poiseuille law of friction: flow is directly proportional to the radius of the tube to the forth power, and pressure gradient down the tube and inversely proportional with the length of the tube and viscosity of the gas. The resistance is inversely related to the flow.
Therefore the most important determinant of the flow and resistance is the radius of the tube.

Wednesday, November 28, 2007

What are the physiologic changes of vital capacity (VC), closing capacity (CC) and functional residual capacity (FRC) during pregnancy?

During pregnancy, both vital capacity (VC) and closing capacity (CC) are minimally affected but functional residual capacity (FRC) decreases up to 20% at term; FRC returns to normal within 48 h of delivery. This decrease is principally due to a reduction in expiratory reserve volume as a result of larger than normal tidal volumes.

Tuesday, November 27, 2007

What is a “tet” spell?

A "tet spell" is a sudden hypoxic episode in a patient with Tetralogy of Fallot. It is characterized by severe tachypnea, and increased cyanosis. Untreated, it may lead to loss of consciousness,

seizures, cerebrovascular accidents and death. The tet spell result from a relative decrease in systemic vascular resistance (SVR) to pulmonary vascular resistance (PVR), resulting in a decrease in the effective pulmonary blood flow with right to left shunting through the VSD.

Monday, November 26, 2007

An intubated man is undergoing tracheostomy under general anesthesia with isoflurane, N_2O, and oxygen (50%/50%). The trachea is exposed and incised; the surgeon applies electrocautery to some superficial bleeding vessels prior to extubation and cannulation with a tracheostomy tube. The cuff of the endotracheal tube ignites, followed by the shaft of the tube. A "blowtorch" airway fire is noted.
What should be the initial management of this situation? What steps can be taken to avoid this complication?

Considerable attention has been paid to the problem of LASER safety, but the ability of electrocautery units to ignite endotracheal tubes is also well described. Electrocautery units develop very high frequency alternating current which creates localized high temperature (>1200 °C) when it contacts tissue. This temperature is high enough to ignite the plastic in most endotracheal tubes.
The initial management of an electrocautery-induced airway fire includes immediately halting use of the electrocautery, discontinuing ventilation and removing the endotracheal tube. The field may be flooded with saline if necessary. The trachea can be reintubated, or a tracheal appliance placed in the tracheal stoma if the procedure is a tracheostomy as in this case. Bronchoscopy can be used to evaluate the airway for the extent of injury. Cases reported in the literature have ranged in severity from no patient injury to death.

The steps used to minimize the chance of airway fires are similar to those used for LASER surgery. First, the most heat resistant tube available should be used. Second, the cuff should be filled with saline rather than air, which reduces the chance of ignition at this vulnerable spot. Third, wet pledgets can placed around the tube in the surgical field. Fourth, the lowest possible concentration of oxygen compatible with adequate respiratory status should be employed. Importantly, nitrous oxide is not a suitable substitute for oxygen, as it supports combustion as well as oxygen does. A nonflammable gas such as helium or nitrogen should be used instead. Finally, the surgeon must be made aware of the possibility of ignition if a vulnerable tube is employed.

Reference:
1. Bailey MK, Bromley HR, Allison JG, et al. Electrocautery-induced airway fire during tracheostomy. Anesth Analg 1990; 71:702-4.
2. Simpson JL, Wolf GL. Endotracheal tube fire ignited by pharyngeal electrocautery. Anesthesiology 1986; 65:76-7.
3. Sosis MB, Braverman B. Prevention of cautery-induced airway fires with special endotracheal tubes. Anesth Analg 1993; 77:846-7.
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Wednesday, November 21, 2007

What are the most frequent mechanisms of cerebral injury during cardiopulmonary bypass?

The most common mechanisms of cerebral injury during CPB are microemboli and hypoperfusion, which in some extent are mutually exclusive. In clinical practice, air, atherosclerotic debris, and fat are the major types of microemboli causing brain injury and all cause neuronal ischemia/ necrosis by blocking the small cerebral vessels.

Reference:
Kincaid E, Hammon J. Cardiopulmonary bypass in Manual of cardiothoracic surgery – Yuh D., Vricella L, Baumgartner W. 2007

Neurologic complications post CPB

Tuesday, November 20, 2007
Are there any special physical examination "tricks" that should be included in the evaluation of tracheal stenosis?
Placing the patient supine can reveal whether this position can be tolerated and may suggest whether the patient's airway is more likely to be obstructed. Moreover, pulmonary function tests, evaluating forced inspiratory and expiratory maneuvers, may reveal whether a lesion is extra- or intrathoracic. High tracheal extrathoracic lesions may worsen with forced inspiration, as large negative pressure within the tracheal accentuates airway narrowing. By contrast, variable intrathoracic lesions may worsen with forced expiration due to generation of relatively positive pressures favoring closure.

Monday, November 19, 2007

How do inhalational anesthetics agents alter hepatic blood flow?

All volatile anesthetics vasodilate the hepatic artery and preportal vessels. This decreases mean hepatic artery pressure and increase venous pooling in the splanchnic vessels. Portal flow decreases as well, with the overall result of suboptimal perfusioin of the liver. In addition, the autoregulation is abolished and blood flow becomes pressure dependent. This is usually tolerated well by patients with normal hepatic function (as metabolic demand is also decreased by these drugs). Patients with hepatic disease are more susceptible to injury secondary to preexisting impaired perfusion.

Friday, November 16, 2007

Your anesthetized patient is scheduled to have an MRI exam on his spine, postoperatively. You consider that during the exam, the patient must have ECG monitoring because of cardiac rhythm instability. What precautions would you take?
The radiofrequency fields that occur during an MRI can heat ECG cables and electrodes, seriously burning skin under the electrodes. The following precautions should be undertaken:

1. Use MRI – compatible equipment; coordinate with the MRI staff technologist before the imaging study to ensure that cables and electrodes are available those have been cleared for use in the MRI environment. Make sure electrodes used aren’t past their expiration date.

2. Search the patient for any previously used electrodes and cables that may have been inadvertently left behind, either in clothing, in the sheets, or on the patient. Remove any electrodes and cables that are no longer being actively used for monitoring. This will prevent the possibility of burns from leftover electrodes.
3. Be aware that, even though you're using electrodes and cables that are approved for use during an MRI, a serious burn can still develop if the electrodes aren't in complete contact with the skin surface (that is, if excess hair hasn't been removed or there's an air gap between the electrode and the skin). When there's an air gap, the electrical pathway is broken and heat can build up at the center of the electrode as well as letting the current arc from the electrode to the skin, which can cause burns. Don't let the cables form a loop and keep them off the patient's skin by placing a blanket under them.
4. After the MRI study is complete, patients (especially those who can't verbalize), should be examined for possible burns or reddening of the skin under the electrodes.
5. If your patient has a burn, make sure she gets appropriate treatment. Document all medically relevant information in the medical record and file an incident report. Notify your facility's biomedical engineering department and quarantine the electrodes involved plus any remaining in the packet.

Reference:
LANGE S et al. Nursing 2006, Volume 36, Number 11, p. 18

Thursday, November 15, 2007

Is labor sufficient stimulus to initiate autonomic hyperreflexia in a pregnant patient with a chronic C5 –C6 spinal cord injury?
Yes. There are many case reports describing this response in laboring pregnant patients. Intraventricular hemorrhage has also been reported due to autonomic hyperreflexia in the laboring patient. In one report the authors describe a woman who presented in early labor with autonomic hyperreflexia. They note that the establishment of epidural anesthesia resolved the response, and interestingly note that the response returned with the resolution of the epidural anesthesia.
Reference:

1. McGregor JA Meeuwsen J. Autonomic hyperreflexia: a mortal danger for spinal cord-damaged women in labor. Am J Obstet Gynecol 1985 Feb 151(3):330-3.
2. Katz VL Thorp JM Jr Cefalo RC. Epidural analgesia and autonomic hyperreflexia: a case report. Am J Obstet Gynecol 1990 Feb 162(2):471-2.
3. Gimovsky ML Ojeda A Ozaki R Zerne S. Management of autonomic hyperreflexia associated with a low thoracic spinal cord lesion. Am J Obstet Gynecol 1985 Sept 153(2):223-4.
4. Stephen B. Corn, M.D. and B. Scott Segal, M.D.
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Wednesday, November 14, 2007

Prerenal oliguria can be characterized by:

a. urinary Na > 40 mEq/l and urine osmolarity < 250 mOsm/ l
b. urinary Na < 40 mEq/l and a high urinary K level
c. urinary Na > 40 mEq/l and urine osmolarity > 400 mOsm/ l
d. urinary Na < 40 mEq/l and urinary osmolarity > 400 mOsm/ l
e. none of the above

Answer: d

Monday, November 12, 2007

You evaluate preoperatively a 65 years old male (120 kg, 180 cm) scheduled for carotid endarterectomy. His past medical history is significant for coronary artery disease, CCS (Canadian Cardiovascular Society score) of 4, NYHA class IV, symptomatic sick sinus syndrome, asymptomatic critical left carotid artery stenosis, uncontrolled type II diabetes mellitus, and chronic renal insufficiency.
According with ACC/ AHA guidelines, what is his perioperative cardiac risk?

The carotid endarterectomy is a noncardiac surgical procedure that carries an intermediate cardiac risk (reported as less than 5%).
However, the patient per se has several major clinical predictors of increased perioperative cardiovascular risk according with AHA/ ACC guidelines (unstable angina and decompensated CHF, significant symptomatic arrhythmia).

Therefore, by combining the patient (major predictors) and surgical procedure (intermediate risk) cardiac risk factors, we can conclude that the patient is at high perioperative cardiac risk for MI, CHF and death.

Friday, November 9, 2007

What are the main pharmacological and pharmacokinetic characteristics of Fenoldopam relevant to clinical practice?

Fenoldopam is a benzazepine derivative that is a slightly more potent agonist than dopamine at DA1 receptors but does not act as an agonist at DA2 receptors or α- and β-adrenergic receptors. Administered parenterally, it acts predominantly as a vasodilator in peripheral arteries and as a diuretic in the kidneys. Administered directly into the central nervous system, fenoldopam stimulates adenylate cyclase activity in the caudate nucleus, and it induces contralateral rotation in rats with lesions of the caudate nucleus — an effect that is consistent with the activation of D1-like receptors. However, because it is poorly soluble in lipids, it does not penetrate the blood–brain barrier, and it has no central nervous system effects when administered intravenously.

The mean elimination half-life of intravenously infused fenoldopam after the cessation of a 2-hour infusion is 9.8 minutes. During longer infusions (up to 48 hours), the elimination half-life may be shorter. After an infusion has begun, steady-state plasma concentrations are reached within 30 to 60 minutes. In plasma, 85 to 90 percent of fenoldopam is bound to proteins, and its volume of distribution is approximately 600 ml per kilogram. There is a predictable relation between the dose and the plasma concentration of fenoldopam, and there is a linear relation between the reduction in blood pressure and the rate of infusion of fenoldopam.

Reference:

Murphy M, et al. Fenoldopam — A Selective Peripheral Dopamine-Receptor Agonist for the Treatment of Severe Hypertension. NEJM 2001, 11;21 345:1548-1557

Thursday, November 08, 2007

Methemoglobin shifts the oxyhemoglobin dissociation curve to:

a. right
b. left

Answer: b.

Wednesday, November 07, 2007

If SvO2 falls, it may be indicative of:

a. increase in VO2
b. decrease in CO
c. decrease in Hb
d. increase in SaO2
e. a, b, c
f. b, c, d

Answer: e

Tuesday, November 06, 2007

What are the beneficial effects of short time (12-18 hours) smoking cessation?

Smoking cessation 12-18 hours before surgery results in:

1. decrease carboxyhemoglobin levels

2. decrease sympathetic tone
3. normalization of the oxyhemoglobin dissociation curve

Monday, November 05, 2007

How does change CVP tracing during tachycardia and bradycardia?

Tachycardia produces fusion of individual CPV waveforms components particularly the *a* and *c* waves.
During slow heart rhythm a mid-diastolic plateau pressure wave *h* becomes evident on the CVP tracing.

Reference:

Mark J. Central venous pressure, left atrial pressure. In Atlas of Cardiovascular monitoring. 1998; 22-23.

Friday, November 02, 2007

What is platypnea-orthodeoxia syndrome (POS)?

Platypnea-orthodeoxia is a rare syndrome characterized by hypoxemia in the upright position after pneumonectomy and relieved by recumbency. Platypnea was reported by Butchell in 1949 and first described by Robin in 1976. Nevertheless the cause remains unclear, as fewer than 50 cases have been reported.
It seems that POS is associated with three groups of disorders: (1) intracardiac shunts, (2) anatomic pulmonary vascular shunts, and (3) pulmonary parenchymal shunts. In any patient presenting with dyspnea and hypoxemia after pneumonectomy, POS should be one of the differential diagnoses.

Reference:
1. E.D. Robin and R.F. McCauley, An analysis of platypnea-orthodeoxia syndrome including a “new” therapeutic approach, Chest 112 (1997), pp. 1449–1451.
2. G.P. Chen, S.L. Goldberg and E.A. Gill Jr, Patent foramen ovale and the platypnea-orthodeoxia syndrome, Cardiol Clin 23 (2005), pp. 85–89
3. M. Faller, R. Kessler, A. Chaouat, M. Ehrhart, H. Petit and E. Weitzenblum, Platypnea-orthodeoxia syndrome related to an aortic aneurysm combined with an aneurysm of the atrial septum, Chest 118 (2000), pp. 553–557

Thursday, November 1, 2007

Your patient came off CPB without any inotropic support, stable, and Protamine was administered; within 2 minutes after Protamine was given, arterial BP is 55/30 mmHg and the monitor did not display PAP and CVP. Why did the patient develop hypotension after protamine was administered?

There are several causes of hypotension following Protamine administration.
In a classic paper, Horrow proposed 3 different types of circulatory reactions to Protamine:

1. type I = systemic hypotension from rapid injection – predictable pharmacologic reaction histamine mediated
2. type II = anaphylactic reaction or anaphylactoid reaction
   anaphylactic reaction antibody IgE mediated
   immediate anaphylactoid response without antibody involvemet
   delayed anaphylactiod response – noncardiac pulmonary edema
3. type III = catastrophic pulmonary vasoconstriction with systemic hypotension

Reference:
Horrow JC. Protamine: A review of its toxicity. Anesth Analg 64: 348-361, 1985.