Cardiac Arrest during Total Hip Arthroplasty in a Patient on an Angiotensin Receptor Antagonist

Susan M. Goodman, MD • Daniel Krauser, MD • C. Ronald Mackenzie, MD • Stavros Memtsoudis, MD, PhD

Received: 22 February 2011/Accepted: 13 October 2011/Published online: 11 May 2012
© Hospital for Special Surgery 2012

Statement of Need

The Renin Activating System, or RAS, plays a critical role in the regulation and maintenance of hemodynamic stability, a function which increases in importance under stresses such as anesthesia and surgery. Factors such as dehydration, common in the fasting state prior to surgery, as well as anemia produced by anticipated operative blood loss magnify this demand. Common antihypertensive agents, Angiotensin converting enzyme inhibitors (ACEI) and Angiotensin receptor antagonists (ARA) block this important compensatory capability. Profound hypotension, increased mortality, and impairment of renal function have been described as a consequence of RAS blockade when ACEI/ARAs are taken immediately prior to general and vascular surgery. This has not been studied in orthopedic surgery performed under regional anesthesia. This case report and discussion will fill this knowledge gap so that a potential modifiable cause of profound hypotension and cardiac arrest can be considered.

Target Audience

This activity is targeted at primary care physicians, specialty physicians, orthopaedic surgeons, general medicine, residents and fellows.

Objectives

Hospital for Special Surgery professional education activities are intended to improve knowledge, competence and performance of our learners and to lead to better patient

care. At the conclusion of the activity, the participant should be able to:

- Discuss and understand chemical messenger systems, such as the Renin Activating System, in maintenance of hemodynamic stability in the setting of regional anesthesia and orthopaedic surgery.
- Describe antihypertensive agents, such as Angiotensin converting enzyme inhibitors (ACEI) and Angiotensin receptor antagonists (ARA), and their interactions with the Renin Activating System blockade, and how they are less more likely to produce profound hypotension for patients in the orthopaedic setting.
- Discuss factors, such as dehydration and anemia, which may magnify the potential for hypotension in orthopaedic patients undergoing regional anesthesia.
- Describe the mechanism by which an increase in intra-abdominal and intra-thoracic pressure can diminish venous return and further increase the importance of Renin Activating System mediated compensation.

Accreditation

Hospital for Special Surgery is accredited by the Accreditation Council for Continuing Medical Education to provide continuing medical education for physicians.

Credit Designation

Hospital for Special Surgery designates this Journal-based CME activity for a maximum of **1.0 AMA PRA Category 1 Credit(s)**TM. Physicians should claim only the credit commensurate with the extent of their participation in the activity.

Faculty Disclosure

In accordance with the Accreditation Council for Continuing Medical Education's Standards for Commercial Support, all CME providers are required to disclose to the activity audience the relevant financial relationships of the planners, teachers, and authors involved in the development of CME content. An individual has a relevant financial relationship if he or she has a financial relationship in any amount occurring in the last 12 months with a commercial interest whose products or services are discussed in the CME activity content over which the individual has control. It is the policy of Hospital for Special Surgery to disclose **all** financial relationships that planners, teachers, and authors have with commercial interests. Relationship information appears below.

Activity Directors/Authors/Planning Committee

Susan Goodman, MD has disclosed no financial relationships.

Daniel Krauser, MD has disclosed no financial relationships.

C. Ronald Mackenzie, MD has disclosed no financial relationships.

Stavros Memsoudis, MD has disclosed no financial relationships.

Planning Committee/CME Committee/Hospital for Special Surgery Staff

Antonia Augurt, LCSW has disclosed no financial relationships.

Charles Cornell, MD has disclosed the following financial relationships:

Exactech	Salary, Royalty or Honoraria
Clinical Orthopedics and Related Research	Senior Associate Editor

Charles Cornell, MD will not be discussing products/services of the commercial interests with which a relationship exists.

Natanya Gayle, MPH has disclosed no financial relationships.

Allison Goldberg, MPA has disclosed no financial relationships.

Other than those noted above, Hospital for Special Surgery Office of CME staff and CME Committee members have no relevant financial relationships to disclose regarding this activity.

Cardiac Arrest during Total Hip Arthroplasty in a Patient on an Angiotensin Receptor Antagonist

Susan M. Goodman, MD · Daniel Krauser, MD · C. Ronald Mackenzie, MD · Stavros Memtsoudis, MD, PhD

Received: 22 February 2011/Accepted: 13 October 2011 /Published online: 11 May 2012
© Hospital for Special Surgery 2012

Abstract Angiotensin-converting enzyme inhibitors (ACEI) and angiotensin receptor antagonists (ARA) are effective and well-tolerated first-line drugs in the therapy of hypertension and, therefore, are frequently encountered in the perioperative setting. Hemodynamic compensation for volume depletion seen in the perioperative period is normally mediated by the renin–angiotensin system, which is blocked by ACEI/ARA. These drugs may contribute to severe hypotension during anesthesia induction and may have contributed to the cardiac arrest seen in this patient. Additional factors such as increased intra-abdominal pressures and respiratory obstructive episodes leading to diminished venous return, as well diuretic use and the fasting state, common in the perioperative orthopedic patient, are likely to have contributed as well. Medication use may be an easily modifiable risk factor for severe hypotension and possible cardiac arrest in the perioperative setting.

Keywords cardiac arrest · perioperative antihypertensive therapy · Angiotensin converting enzyme inhibitors · Bezold–Jarisch reflex · intra-operative hypotension · renin angiotensin system (RAS)

Introduction

Due to the prevalence of hypertension in the general population [15,16], patients undergoing total joint arthroplasty are frequently taking one or more anti-hypertensive medica-

tions, including agents acting on the angiotensin system. While the latter drug category has shown beneficial effects in the treatment of hypertension in general, their continued use in the perioperative period has been questioned as it has been linked to increased risk of hemodynamic instability [3,4], especially when used with diuretics [13].

In this article, we discuss the case of a hypertensive patient on an angiotensin receptor antagonist (ARA) and diuretic, who suffered severe hypotension and cardiac arrest after induction of anesthesia. While the reasons for this event are likely multifactorial, we take the opportunity to highlight the debate about continuation of ARA and other drugs affecting the angiotensin system in the perioperative period.

Case Report

A 57-year-old male was admitted for elective hip arthroplasty for avascular necrosis of the femoral head. His prior history was remarkable for rheumatoid arthritis for which he received infliximab and obesity (body mass index (BMI) of 43.2). Additional history was notable for hypertension, well controlled on olmesartan 20 mg combined with hydrochlorothiazide 12.5 mg, obstructive sleep apnea for which he used continuous positive airway pressure at night, and hepatitis at age 25 for which he had been given glucocorticoids for several months. His pre-operative evaluation was unremarkable and included a normal electrocardiogram (EKG) and chemistries. His creatinine was 1.1 mg/dL, and his hemoglobin of 10.7 g/dl revealed moderate anemia. Cardiac evaluation included a normal stress test and angiogram, which had been performed 1 year prior to surgery.

His vital signs at the time of admission revealed his blood pressure of 140/76, his heart rate of 70, and his temperature of 36.7. He was obese, with limited painful hip motion, but his physical exam was otherwise unremarkable. He took his olmesartan/hydrochlorothiazide on the morning of surgery as instructed.

S. M. Goodman, MD (✉) · D. Krauser, MD · C. R. Mackenzie, MD · S. Memtsoudis, MD, PhD
Hospital for Special Surgery,
535 East 70th Street, New York, NY 10021, USA
e-mail: goodmans@hss.edu

S. M. Goodman, MD · D. Krauser, MD · C. R. Mackenzie, MD · S. Memtsoudis, MD, PhD
Weill Cornell Medical College, New York, NY, USA

Preparation for surgery included the application of nasal oxygen and standard American Society of Anesthesiologists monitoring and placement of an arterial line. He was given midazolam in divided doses prior to placement of a combined spinal/epidural anesthetic with 2.2cc of 0.5% bupivacaine. Shortly thereafter, the patient became profoundly hypotensive and bradycardic and progressed to asystole. With only escape beats present, advanced cardiac life support protocol was instituted immediately. The trachea was intubated, cardiac compressions ensued and atropine 2 mg IV was given followed by epinephrine 5 mg IV and subsequently vasopressin 40 U with restoration of pulse and blood pressure after 9 min. A bedside echocardiogram revealed normal left and right ventricular function. The patient was extubated later that day, and no neurologic deficits were present.

He was discharged the following day and readmitted 1 month later for surgery. In response to the events described, the olmesartan/hydrochlorothiazide was discontinued and substituted by metoprolol prior to hospital discharge. The metoprolol was taken on the subsequent morning of surgery. He underwent an uncomplicated hip arthroplasty under general anesthesia and was discharged on the fifth post-operative day.

Discussion

Angiotensin converting enzyme inhibitors (ACEI) and angiotensin receptor antagonists (ARA) are first-line drugs for the treatment of hypertension and have additional benefits of renal function preservation in patients with diabetic and non-diabetic nephropathy. They are long-acting, effective agents, which are increasingly prescribed and therefore frequently encountered in patients presenting for orthopedic surgery. As perioperative hypertension increases, the risk of surgical complications such as increased operative blood loss, cardiac arrhythmias, and stroke, patients are usually instructed to continue their anti-hypertensive medications on the morning of surgery. In fact, drugs such as beta blockers (BB) and calcium channel blockers (CB) are known to have additional beneficial effects in the perioperative period, protecting against myocardial ischemia and arrhythmia [5,19]. Hypotension, however, may also contribute to perioperative morbidity and mortality and may ensue when potent long-acting anti-hypertensive agents are taken on the operative day. Hypotension has been reported in vascular surgery and general surgery patients chronically treated with ACEI/ARA agents undergoing general anesthesia but has not been studied extensively in patients undergoing orthopedic surgery under regional anesthesia [3,18].

Although both ARA and ACEI exert their effects by interfering with the renin angiotensin system (RAS), ARA differ from ACEI. ACEI inhibit the synthesis of angiotensin II (AII), resulting in high levels of vasodilatory bradykinin. ARA selectively block AII receptor binding, leading to increased AII levels with normal bradykinin levels, and

have gained popularity in response to bradykinin-related side effects such as cough commonly seen with ACEI [14,17]. AII vasoconstrictive effects are prevented by both ACEI/ARB, although more selective and more complete renin–angiotensin blockade may be seen with ARA use.

Hemodynamic stability is maintained in periods of physiologic stress by the RAS. AII, a potent vasoconstrictor, additionally potentiates vasopressors such as norepinephrine. AII has an important role in compensating for arterial hypotension brought on by anesthesia, as factors such as hypovolemia, hyponatremia, and anemia activate the RAS. Once activated, RAS controls blood pressure by both AII-driven vasoconstriction as well as AII effects on sodium and water reabsorption [7]. Early studies were performed in which a commercially available preparation of AII was used to treat hypotension in the setting of anesthesia induction in patients on chronic ARA treatment undergoing elective vascular surgery. Although this product is no longer available, hemodynamic observations made by transesophageal echocardiography and radial artery monitoring revealed a prompt reversal of hypotension without a concomitant increase in pulse rate in hypotensive patients given AII bolus injections [10]. In extensions of this work, the hemodynamics at the time of anesthesia induction in vascular surgery patients treated with CB and BB were compared to patients treated with ACEI and ARA. ARA-treated patients were more likely to develop significant hypotension at the time of anesthesia induction than CB-, BB-, or ACEI-treated patients [3]. Moreover, ARA-treated patients required more ephedrine to maintain blood pressure and were more likely to be refractory to ephedrine or phenylephrine, requiring a vasopressin receptor agonist to stabilize blood pressure. There were no cardiac events in the ARA-treated group, as documented by ST-segment changes, post-operative EKG, and troponin levels. Three patients in the BB/CB and ACEI group, all of whom had significant intraoperative bleeding, had EKG changes and troponin elevations [3]. In additional studies, when 30-day mortality was used as an endpoint to compare patients undergoing elective abdominal aortic aneurysm repair who were chronically treated with ARA/ACEI, a crude mortality rate of 5.8% in the ACEI/ARB-treated patients versus 1.9% in the patients undergoing surgery without RAS blockade was reported. Re-analysis using a propensity score for RAS blockade, which was calculated using factors such as congestive heart failure, diabetes, and serum creatinine, had little effect on the observed difference in mortality [18].

The incidence of hypotension after anesthesia induction for general surgical procedures was studied retrospectively in 267 patients chronically treated with ACEI/ARA therapy. Fifty-four percent of patients treated with ACEI/ARA therapy within 24 h of surgery were hypotensive after induction of anesthesia. Additionally, patients who continued ACEI/ARA therapy and took a dose within 10 h of surgery were more likely to have moderate hypotension than patients who discontinued therapy more than 10 h pre-operatively. There were, however, no significant differences in adverse events between the groups [8].

Acute kidney injury, which has recently been described in an ACEI-treated patient undergoing knee arthroplasty, is a well-recognized complication of cardiac surgery, with a reported incidence as high as 30–40%. In a retrospective cohort study of 1,358 patients undergoing cardiac surgery, the relationship of AKI to ACEI/ARA therapy was analyzed. Most factors associated with AKI such as age, race, combined valve surgery and coronary artery surgery, diabetes, and neurologic disease, cannot be modified. ACEI/ARA therapy, which could be discontinued prior to surgery, was associated with a 27.6% increased risk of AKI [1]. Similar conclusions were reached in a study of acute kidney injury after elective aortic surgery, where ACEI/ARA therapy was the only statistically significant factor associated with AKI identified [6]. Additionally, acute kidney injury has recently been reported in an ACEI-treated patient undergoing knee arthroplasty [18].

Our patient, who had taken olmesartan/hydrochlorothiazide, a potent combination of an ARA plus a diuretic, as instructed on the morning of surgery, had severe hypotension progressing to cardiac arrest after anesthesia induction.

The causes leading to hypotension and asystole in the presented case are likely multifactorial. Neuraxial anesthesia is known to cause decreases in preload and afterload via vasodilatation induced by chemical sympathectomy, resulting in pooling of intravascular volume in the lower extremities [11]. The effect of chemical sympathectomy on the peripheral vasculature in the setting of decreased compensatory abilities due to RAS blockade, hypovolemia, and anemia are likely all contributory. Diuretic use on the morning of surgery was also likely to have contributed to dehydration and hypovolemia. While compensatory mechanisms are usually sufficient to counteract these events in the vast majority of patients, in this case, the result was a decrease in right heart filling pressures and bradycardia/asystole, consistent with the Bezold–Jarisch reflex. This reflex, mediated by cardiopulmonary vagal afferent nerve fibers, is accentuated in the clinical setting of hypovolemia and anemia, producing paradoxical bradycardia and precipitous hypotension as well as hypopnea. This has been described when prolonged standing with pooling of blood

in the lower extremities leads to diminished intracardiac volume, as well as in the setting of anesthesia and surgery [12].

The role of volume depletion in this patient, frequently present prior to surgery due to use of diuretics and the fasting state, cannot be overlooked. Additionally, increased intra-abdominal pressures secondary to the patient's body habitus with his markedly elevated BMI as well as the potential adverse effects of respiratory obstructive episodes on venous return may have played a role in the events observed [11]. Adrenal insufficiency due to steroid use is unlikely to have played a role given the decades intervening between steroid therapy and the surgery described here. Supporting the significant contribution of ARA use to his profound hypotension after anesthesia induction includes his uncomplicated surgery off olmesartan/hctz 1 month after the event described above. Adverse events are frequently multifactorial. Although the Bezold–Jarisch reflex was the likely final event in the cascade of events that occurred, the severity of the reaction was logically accentuated by the patients' volume status, body habitus, and inhibition of compensatory peripheral vasoconstriction seen in ARA-treated patients. A similar case has been reported in which a patient treated with enalapril had severe hypotension after regional anesthesia induction prior to hip arthroplasty, with a subsequent uncomplicated arthroplasty performed under regional anesthesia after discontinuation of enalapril [9].

There are seven ARAs currently available for use in the USA. The potent direct renin inhibitor, aliskiren (Tekturna), which interferes with the conversion of angiotensin to angiotensin 1, should probably be considered similarly, although there are no reports of hypotension in the perioperative setting associated with its use. While the indications for use in congestive heart failure, nephropathy, and proteinuria differ slightly, all seven, as well as aliskiren, are approved for use in hypertension. The differences in inhibition of A11 at trough level, specific affinity to the Angiotensin 1 (AT1) receptor, and biologic half-life effect potency and duration of action. Below is a chart describing the pharmacokinetics and efficacies of the available ARAs:

Drug	Trade name	Half-life (h)	Inhibition at trough (%)	AT1 affinity	Mean BP drop (SBP/DBP) ^a	Dose (mg)
Losartan	Cozaar	2	30	1,000-fold	8/5	50–100
Candesartan	Atacand	9		10,000-fold	10/6	4–32
Valsartan	Diovan	6	30	20,000-fold	8/4	80–320
Irbesartan	Avapro	13	50	8,500-fold	10/7	150–300
Telmisartan	Micardis	24	40	3,000-fold	10/6	40–80
Olmesartan	Benicar	15	70	12,500-fold	14/12	5–40

^aAt moderate dose range [2]

Further studies are needed to clarify the effect of ACEI/ARA therapy on AKI and hypotension in patients undergoing orthopedic surgery under regional anesthesia. ACEI/ARA,

diuretic therapy, and logically the direct renin inhibitor, aliskiren, should be discontinued 24 to 48 h prior to surgery. Antihypertensive medications such as calcium channel blockers

or beta blockers should be substituted only if needed for blood pressure control. This pre-operative alteration in medication could eliminate an important risk factor for intraoperative hypotension, acute kidney injury, and possibly cardiac arrest.

Disclosures Each author certifies that he or she has no commercial associations (e.g., consultancies, stock ownership, equity interest, patent/licensing arrangements, etc.) that might pose a conflict of interest in connection with the submitted article.

Each author certifies that his or her institution has approved the reporting of this case, that all investigations were conducted in conformity with ethical principles of research.

References

1. Arora P, Rajagopalam S, Ranjan R, Kolli H, Singh M, Venuto R, Lohr J. Preoperative use of angiotensin-converting enzyme inhibitors/angiotensin receptor blockers is associated with increased risk for acute kidney injury after cardiovascular surgery. *Clin J Am Soc Nephrol*. 2008;5:1266–1273.
2. Barreras A, Gurk-Turner C. Angiotensin II receptor blockers. *Proc (Bayl Univ Med Cent)*. 2003;1:123–126.
3. Brabant SM, Bertrand M, Eyraud D, Darmon PL, Coriat P. The hemodynamic effects of anesthetic induction in vascular surgical patients chronically treated with angiotensin II receptor antagonists. *Anesth Analg*. 1999;6:1388–1392.
4. Brabant SM, Eyraud D, Bertrand M, Coriat P. Refractory hypotension after induction of anesthesia in a patient chronically treated with angiotensin receptor antagonists. *Anesth Analg*. 1999;4:887–888.
5. Butterworth J, Furberg CD. Improving cardiac outcomes after noncardiac surgery. *Anesth Analg*. 2003;3:613–615.
6. Cittanova ML, Zubicki A, Savu C, Montalvan C, Nefaa N, Zaier K, Riou B, Coriat P. The chronic inhibition of angiotensin-converting enzyme impairs postoperative renal function. *Anesth Analg*. 2001;5:1111–1115.
7. Colson P, Ryckwaert F, Coriat P. Renin angiotensin system antagonists and anesthesia. *Anesth Analg*. 1999;5:1143–1155.
8. Comfere T, Sprung J, Kumar MM, Draper M, Wilson DP, Williams BA, Danielson DR, Liedl L, Warner DO. Angiotensin system inhibitors in a general surgical population. *Anesth Analg*. 2005;3:636–44, table of contents.
9. Cozanitis DA. The importance of interrupting angiotensin converting enzyme inhibitor treatment before spinal anaesthesia—a controlled case report. *Anaesthesiol Reanim*. 2004;1:16–18.
10. Eyraud D, Mouren S, Teugels K, Bertrand M, Coriat P. Treating anesthesia-induced hypotension by angiotensin II in patients chronically treated with angiotensin-converting enzyme inhibitors. *Anesth Analg*. 1998;2:259–263.
11. Hartmann B, Junger A, Klasen J, Benson M, Jost A, Banzhaf A, Hempelmann G. The incidence and risk factors for hypotension after spinal anesthesia induction: An analysis with automated data collection. *Anesth Analg*. 2002;6:1521–9, table of contents.
12. Kashiwara K. Roles of arterial baroreceptor reflex during bezold-jarisch reflex. *Curr Cardiol Rev*. 2009;4:263–267.
13. Kheterpal S, Khodaparast O, Shanks A, O'Reilly M, Tremper KK. Chronic angiotensin-converting enzyme inhibitor or angiotensin receptor blocker therapy combined with diuretic therapy is associated with increased episodes of hypotension in noncardiac surgery. *J Cardiothorac Vasc Anesth*. 2008;2:180–186.
14. Larochelle P, Flack JM, Marbury TC, Sareli P, Krieger EM, Reeves RA. Effects and tolerability of irbesartan versus enalapril in patients with severe hypertension. irbesartan multicenter investigators. *Am J Cardiol*. 1997;12:1613–1615.
15. Liu SS, Della Valle AG, Besculides MC, Gaber LK, Memtsoudis SG. Trends in mortality, complications, and demographics for primary hip arthroplasty in the united states. *Int Orthop*. 2009;3:643–651.
16. Memtsoudis SG, Della Valle AG, Besculides MC, Gaber L, Laskin R. Trends in demographics, comorbidity profiles, in-hospital complications and mortality associated with primary knee arthroplasty. *J Arthroplasty*. 2009;4:518–527.
17. Moser M. Angiotensin-converting enzyme inhibitors, angiotensin II receptor antagonists and calcium channel blocking agents: A review of potential benefits and possible adverse reactions. *J Am Coll Cardiol*. 1997;7:1414–1421.
18. Raiton CJ, Wolpin J, Lam-McCulloch J, Belo SE. Renin-angiotensin blockade is associated with increased mortality after vascular surgery. *Can J Anaesth*. 2010;8:736–744.
19. Wijeyesundera DN, Beattie WS. Calcium channel blockers for reducing cardiac morbidity after noncardiac surgery: A meta-analysis. *Anesth Analg*. 2003;3:634–641.

Cardiac Arrest during Total Hip Arthroplasty in a Patient on an Angiotensin Receptor Antagonist

Susan M. Goodman, MD • Daniel Krauser, MD • C. Ronald Mackenzie, MD • Stavros Memtsoudis, MD, PhD

Received: 22 February 2011/Accepted: 13 October 2011 /Published online: 11 May 2012
© Hospital for Special Surgery 2012

Instructions for Post-Test and CME Credit

In order to earn CME credit for this journal activity, you must read the article and successfully pass the post test. A passing grade of 70% is required to earn credit. For questions related to the post-test, contact Allison Goldberg, MPA, Director of Professional Education at 212-606-1834.

Option 1: Take the post-test on-line

1. Go to the HSS Journal homepage at www.springer.com/hss.
2. Click on CME articles.
3. Click on “Cardiac Arrest during Total Hip Arthroplasty in a Patient on an Angiotensin Receptor Antagonist” to view the full-text pdf article.
4. After you have reviewed the article click on ‘Complete the Current CME Test Online’ to register and complete the test.

Option 2: Take the post-test via hard copy printed in the Journal

You may complete the hard copy of the post test. Please mail the test to:

Journal CME Post-Test
Office of Continuing Medical Education
Education Division
535 East 70th Street
New York, NY 10021

Cme Questions for “Cardiac Arrest during Total Hip Arthroplasty in a Patient on an Angiotensin Receptor Antagonist”

1. What factors may complicate stopping ACE inhibitors perioperatively:
 - a. Intra and postoperative hypertension.
 - b. Postoperative hypotension
 - c. Difficulty in converting to other antihypertensive agents effectively and timely.
 - d. a & c
 - e. all of the above
2. Which factor can contribute to postoperative hypotension in patients on ACE inhibitors:
 - a. Postoperative pain
 - b. Intra and postoperative anemia and blood loss
 - c. perioperative anxiety
 - d. Prolonged NPO status
 - e. b & d
 - f. all of the above
3. Intra and postoperative hypertension increases the risk of:
 - a. surgical blood loss
 - b. cardiac arrhythmias
 - c. stroke
 - d. all of the above
4. ACEI inhibit the synthesis of angiotensin II. The effect of AII includes:
 - a. vasodilation
 - b. vasoconstriction
 - c. vasopressor potentiation
 - d. a & c
 - e. b & c

5. Significant hypotension at the time of anesthesia induction is most common in patients treated with:
 - a. calcium channel blocking agents
 - b. beta blocking agents
 - c. ACEI
 - d. Angiotensin receptor antagonists
6. Acute kidney injury is a common complication of cardiac surgery. Risk factors include:
 - a. increased age
 - b. Diabetes
 - c. neurologic disease
 - d. ACEI/ARA therapy
 - e. b & c
 - f. all of the above
7. Medications which should be continued and taken on the morning of surgery in a patient with hypertension, angina, and normal cardiac function:
 - a. metoprolol
 - b. furosemide
 - c. lisinopril
 - d. Plavix
8. The Bezold –Jarisch reflex, mediated by cardiopulmonary vagal fibers, results in paradoxical bradycardia and precipitous hypotension. This is accentuated with:
 - a. prolonged standing
 - b. hypovolemia
 - c. anemia
 - d. all of the above
9. Beta blockers have a protective effect on patients in the intra and postoperative period, decreasing the incidence of which of the following:
 - a. myocardial infarction
 - b. stroke
 - c. hypotension
 - d. wound dehiscence

CME REGISTRATION FORM

The ACCME requires that each accredited CME provider evaluate and assess the effectiveness of all CME accredited activities. This activity evaluation will help us document our efforts to address physician practice gaps and educational needs.

Should you have any questions, please contact the Office of Continuing Medical Education. Thank you.

Expires April 30, 2013

Please complete the following contact information:

First Name:

Last Name:

Title (ie; MD, PhD, NP)

Email Address

Specialty (ie Orthopaedics, Rheumatology, Pediatrics)

Please indicate your profession:

☐ Primary Care Physicia	☐ General Medicine
☐ Orthopaedics	☐ Resident/Fellow
☐ Rheumatology	☐ Other
☐ Anesthesiology	

EVALUATION (must be completed for you to receive credit)

How likely are you to implement the following strategies in your practice:

(Scale: 1= Not At All Certain; 7= Very Certain)

Discuss and be able to use appropriate chemical messenger systems, such as the Renin Activating System, in maintenance of hemodynamic stability in the setting of regional anesthesia and orthopaedic surgery.	1 2 3 4 5 6 7
	□ □ □ □ □ □ □

Describe antihypertensive agents, such as Angiotensin converting enzyme inhibitors (ACEI) and Angiotensin receptor antagonists (ARA), and their interactions with the Renin Activating System blockade, and how they are less likely to produce profound hypotension for patients in the orthopaedic setting.	1 2 3 4 5 6 7
	□ □ □ □ □ □ □

Discuss factors, such as dehydration and anemia, which may magnify the potential for hypotension in orthopaedic patients undergoing regional anesthesia.	1 2 3 4 5 6 7
	□ □ □ □ □ □ □

Describe the mechanism by which an increase in intra-abdominal and intra-thoracic pressure can diminish venous return and further increase the importance of Renin Activating System mediated compensation.	1 2 3 4 5 6 7
	□ □ □ □ □ □ □

As a result of what I have learned, I will make the following change(s) in my practice:

1. _____
2. _____
3. _____

Overall, the content covered in this journal article to date has been useful and relevant to my scope of practice.

☐ Strongly Disagree	☐ Disagree	☐ Agree	☐ Strongly Agree

If you disagree please explain:

Do you feel a commercial product, device, or service was inappropriately promoted in the educational content?

- ☐ Yes ☐ No

If yes, please comment

Please provide at least one topic to be covered in a future article that would help you in your practice and/or improve the health of your patients:

Please provide at least one suggestion for improving this article so that it will be more effective: