

ABSTRACTS

Thursday, May 15, 2003

— Opening Plenary Session —

JFK in Dallas, a Trauma Care Perspective

Adolph H. Giesecke, MD
Former Jenkins Professor of Anesthesiology and Chairman
Department of Anesthesiology and Pain Management
University of Texas Southwestern Medical Center, Dallas, Texas, USA



I was at Parkland Hospital on November 22, 1963, and assisted in the unsuccessful attempt to resuscitate the president and gave anesthesia to Governor Connally. I shall describe the events of that day, emphasizing the roles of doctors at Parkland, the conclusions of the Warren Commission, the controversy surrounding the conspiracy theories, the conclusions of the Select Committee, the movie "JFK," and the evidence that closed the case in 1992. The lecture is dedicated to Pepper Jenkins, Jim Carrico, and Paul Peters, valued colleagues who were there and who have subsequently died.

JFK came to Texas to try to heal a rift between Lyndon Johnson, his vice president, and John Connally, the Governor of Texas. While riding past the School Book Depository in his open-topped limousine, he was shot through the neck and head. Governor Connally, who was riding in front of the president, was shot through the chest, wrist, and thigh. JFK was brought to Parkland Hospital for an attempted resuscitation, which lasted 25 minutes. The doctors who were primarily involved in the resuscitation were Jim Carrico, Pepper Jenkins, Mac Perry, and Charles Baxter, assisted by Kemp Clark, Paul Peters, Bob McClelland, and myself.

Lyndon Johnson ordered the body removed to Bethesda, took the oath of office aboard Air Force One, and ordered Chief Justice of the Supreme Court Earl Warren to investigate the crime. The Warren Commission concluded that the shots were fired from one rifle held by one man, Lee Harvey Oswald, who was perched in the sixth floor window of the School Book Depository. The public was not satisfied and conspiracy theories flourished. The Congressional Select Committee repeated the investigation in 1979 and concluded that Oswald was not the only gunman, and the conspiracy included figures from organized crime. Oliver Stone's movie, "JFK" starring Kevin Costner released in 1991, was based on the discredited conspiracy theory proposed by Jim Garrison, the District Attorney of New Orleans.

Dennis Breo published the evidence, which convinced me that no conspiracy existed. He interviewed the pathologists who did the autopsy; reviewed the films, photographs, drawings, and microscope slides; and interviewed the key players in the resuscitation Carrico, Jenkins, Perry, and Baxter. Based on Breo's article, GD Lundberg, 2 editor of JAMA, concluded that we now have "unequivocal forensic evidence, without reservation that JFK was struck by two bullets fired from behind, from one high velocity rifle...one gunman." His conclusion was confirmed in a carefully researched and written book by GL Posner, called Case Closed. 3 All participants in this congress should visit the Sixth Floor Museum, dedicated to the story of the assassination and housed in the old School Book Depository.

References

1. Breo DL. JFK's death, the plain truth. *JAMA* 1992; 267:2794-802.
2. Lundberg GD. Closing the case in *JAMA* on the John F. Kennedy autopsy. *JAMA* 1992; 268:1736-8.
3. Posner GL. *Case Closed*. Random House, New York, 1993.

— Presidents' Forum —

President's Address: What's New in Trauma

Michael J.A. Parr, MB BS MRCP FRCA FANZCA FJFICM
Intensive Care Specialist, University of New South Wales, Liverpool Hospital,
Sydney, Australia

Learning Objective: To summarise some recent advances in trauma management and to identify topics for future initiatives.

The management of patients following injury continues to evolve as our understanding of the physiology of injury improves. Technology advances are providing better diagnostic options, allowing earlier diagnosis and selective intervention. Noninvasive or minimally invasive techniques are providing new methods of intervention, and therapeutic advances are providing more options for the management of difficult cases and complications.

We have a greater understanding of the economic consequences of trauma care and this is driving more selective management for specified patients. Trauma care dogma is increasingly challenged in the light of sound science, and investigation or review of unproven beliefs that govern treatment decisions should be a priority.

At the time of writing, we once again face the potential for major armed conflict and perhaps never before has the treat of nuclear, biological, and chemical weapons been so great. The challenges and concerns these raise affect us all.

We all have potential roles at local, national, and international levels to improve the management and outcome for the victims of trauma. These improvements will be driven by the enthusiasm and commitment of those involved in trauma care.

Applications of Dexmedetomidine in the Trauma Patient

Michael A.E. Ramsay, MD, FRCA
Baylor University Medical Center
Dallas, Texas, USA

Learning Objectives:

- To understand the pharmacology of α_2 -adrenoceptor agonists.
- To learn how the properties of sedation and analgesia without respiratory depression may be applied to the management of the trauma patient.
- To predict the effect on the hemodynamic profile of the patient.

The α_2 -adrenoceptors are located in the central nervous system, peripheral nervous system, vascular smooth muscle, and a variety of other organs. Presynaptic activation of the α_2 -adrenoceptor modulates the release of norepinephrine, resulting in a reduction in the stress response. In many instances, this reduction can be very beneficial and cardioprotective. In a shock situation, it could be deleterious when the production of catecholamines may be essential to supporting the circulation. The activation of the α_2 -adrenoceptor in the spinal cord and locus ceruleus in the brain produces analgesia and sedation without respiratory depression. The quality of sedation produced is different from most other sedatives that act on GABA receptors. Dexmedetomidine has a sedative profile that resembles non-REM sleep, and patients roused from sedation can be assessed neurologically without evidence of being obtunded.¹

Dexmedetomidine is the most selective α_2 -adrenoceptor agonist available and is an imidazole compound, but without the steroid suppression action seen with etomidate. It is metabolized by the liver into inactive metabolites excreted in the urine. The redistribution half-life is approximately 8 minutes, and it has a terminal half-life of 3.5 hours; therefore, it is a readily controlled sedative when administered as an infusion.

The dosing is labeled to allow up to 0.7 mcg/kg/h for 24 hours in initially intubated patients undergoing mechanical ventilation. Venn et al reported dexmedetomidine use in the medical ICU for up to 7 days without the development of tolerance or dependence and without any rebound hypertension on discontinuing the drug. They also found that doses as high as 2.5 mcg/kg/h were required to properly control sedation in this patient group.²

Dexmedetomidine's analgesic effect has been shown to reduce the need for opioids by 50% in postoperative cardiac surgery patients.³

The lack of respiratory depression has been demonstrated by Hall et al from measurements of end-tidal CO₂, by Ebert et al from arterial blood gas analysis, and from CO₂ response curves by Ramsay et al.^{4,5} This combination of sedation and analgesia with no respiratory depression lends itself to the management of the trauma patient. Controlled sedation can be maintained while facilitating weaning from mechanical ventilation. Sedation and pain management can be provided for the head injury patient without risk of CO₂ retention. The effect of dexmedetomidine on cerebral blood flow has been examined in human volunteers.⁷ It reduces cerebral blood flow, probably as a result of reduced cerebral metabolic rate. This reduction may be an advantage in the management of many head injury patients, particularly if mechanical ventilation can be avoided.

The success in using dexmedetomidine in "fast-track" cardiac surgery patients offers the potential that chest trauma patients may be managed effectively without the need for mechanical ventilation or thoracic epidural analgesia.

Dexmedetomidine allows us an opportunity to re-evaluate how we provide sedation and analgesia to the trauma patient.

References

1. Nelson LE, Lu J, Guo T, et al. The alpha2-adrenoceptor agonist dexmedetomidine converges on an endogenous sleep-promoting pathway to exert its sedative effects. *Anesthesiology* 2003; 98:428-36.
2. Venn M, Newman J, Grounds M. A phase II study to evaluate the efficacy of dexmedetomidine for sedation in the medical intensive care unit. *Intensive Care Med* 2003; 29:201-7.
3. Venn RM, Bradshaw CJ, Spencer R, et al. Preliminary UK experience of dexmedetomidine, a novel agent for postoperative sedation in the intensive care unit. *Anaesthesia* 1999; 54:1136-42.
4. Hall JE, Uhrich TD, Barney JA, et al. Sedative, amnestic, and analgesic properties of small-dose dexmedetomidine infusions. *Anesth Analg* 2000; 90:699-705.
5. Ebert TJ, Hall JE, Barney JA, et al. The effects of increasing plasma concentrations of dexmedetomidine in humans. *Anesthesiology* 2000; 93:382-94.
6. Ramsay MAE, Jones CC, Knorr HC, et al. Dexmedetomidine does not depress the CO₂ response curve in postoperative patients. *Anesthesiology* 2002; 96:A1355.
7. Prielipp RC, Wall MH, Tobin JR, et al. Dexmedetomidine-induced sedation in volunteers decreases regional and global cerebral blood flow. *Anesth Analg* 2002; 95:1052-9.

[Dr. Ramsay receives grant/research support from Abbott Laboratories (the manufacturer of Precedex®) and is a consultant and member of the speakers bureau for that company. In regard to "off-label" uses, Dr. Ramsay will present clinical trial data related to the long-term use of dexmedetomidine (Precedex®).]

Stress Management for Care Providers in the Trauma Setting

Jessie A. Leak, MD

Associate Professor of Anesthesiology, University of Texas, MD Anderson Cancer Center, Houston, Texas

What do you want to have? Where do you want to go? Who do you want to go with? How the hell do you plan to get there? Write it down. Go do it. Enjoy it. Share it. It doesn't get much simpler or better than that.
—Lee Iacocca

Stress management is a ubiquitous subject that many think about but few actively practice. Many of us want to reduce stress, particularly in the workplace, but fail to realize that stress does not occur in a vacuum. If we are feeling stress at work, chances are that we have stress in other areas of our life; these may include personal relationships, anger issues, issues involving our physical environment at home or at work, financial issues, and, most importantly, loss of opportunities to address our individuality: mind, body, and spirit. Without attention to all these areas of our life, we bring this excess baggage to work and perceive an undesirable or unpleasant work environment.

What is Stress? Webster's dictionary describes stress as "any mental or physical tension or strain." Russ Hanlin, CFO, Sunkist, says that "it's always appeared to me that stress is within the individual and not manufactured by the situation."

Hans Selye discussed the scientific theory of General Adaptation Syndrome that describes a built-in ability that our bodies have to adapt to situations to a certain extent. Beyond this point, stress occurs. However, stress can be cumulative and therefore insidiously destructive when homeostasis is no longer possible because of internal resource depletion.

Why am I Feeling Resource Depleted or Experiencing Burnout? Loss of control (particularly in the workplace), the outward manifestation of which is stress, can be intensified for anesthesiologists because of the exaggerated loss of a doctor-patient reciprocal relationship that other physician specialists enjoy with their patients. In other words, the loss of receiving positive strokes from our patients in conventional doctor-patient relationships may intensify a chronic depersonalization.

Anesthesiologists may have little or no interaction with their patients, except in the immediate perioperative period. The loss of a reciprocal relationship with our patients is intensified in an oncology or burn ward-type setting (areas with many suffering patients).

Additional factors that can contribute to loss of control (stress) may include 1) the constant need to suppress symptoms of fatigue and exhaustion; 2) economic factors that compel the physician to perform in the dual role of physician and business manager/CEO; 3) difficult relationships with colleagues; 4) increased severity of illness of patients; 5) liability concerns ("every patient is a potential law suit" mentality); 6) night call/sleep deprivation; or 7) compliance issues, e.g., HIPPA, Medicare.

You are not alone. A 1994 study in Anesthesiology reported that nearly half of the anesthesiologists surveyed felt they were under chronic pressure at work. Female physicians between the ages of 45 and 55 without a partner, with full-time hospital work and the attendant administrative responsibilities in an understaffed area are at greatest risk to experience chronic pressure or stress. This group also commits suicide at six times the rate of the general population.

Chassot described the triad of burnout as emotional exhaustion, depersonalization (loss of empathy), and a lack of personal accomplishment. If you are not sure if you are burned or burning out, see if you recognize any of the following burnout symptoms: feeling tired even with adequate sleep, work dissatisfaction, forgetfulness, sadness, irritability, increased incidence of illness, subpar job performance, substance abuse, decreased concentration, avoidance of interaction with others, increased boredom with work, decreased work accomplishment despite seeming hard work, dreading going to work, avoiding social activities, feeling like work is a dead-end ("why bother"), and the perception that what you were hired to do is not meeting with reality.

Where Do I Go From Here? It is important not to compare yourself to others. What may drain one individual may be a breeze for another. Simply honor what you know is draining you and address the issue. You have a right to be the final judge of your stress issues and to accept them as legitimate. Barbara Larivee tells us in *Moving Into Balance* that "the journey toward personal fulfillment and true transformation requires major restructuring that cannot be prescribed with an intervention formula...The pathway cannot be preplanned. Each of us has our own internal gauge for when we are ready to deal with a critical life issue."

Once you have identified that you are burned out/stressed out, it is imperative to re-establish some control in your life. Ironically, when we are burned out, the last thing that we want to do is change because change is work. What we want is less work, but avoidance of change can become a fear of change (which causes increased stress).

It is not unusual to find that you have stressors in one or more areas in your life: work, relationships (including issues with anger management or toxic relationships), dysfunction in your physical environment, financial woes, and most importantly no time or imbalance in your body, mind, and/or spiritual life. This may include medical issues or a disconnect with our spiritual life, which may or may not include our religious practices or beliefs.

It is important to take inventory as soon as possible. During this process, it is quite helpful to establish what your life purpose may be and what makes your life meaningful. Once you discover what is draining you the most, you have three options: 1) Take care of the issue by yourself and do it! 2) Delegate the task to someone else or hire someone to do it; or 3) Throw it out and let it go!

Now is the time which is the borderline between going up and going down; now is the time when by slipping into laziness even for a moment you will endure constant suffering; now is the time when by concentrating for an instant you will enjoy constant happiness. Focus your mind single-mindedly; strive to prolong the results of good karma.
—The Tibetan Book of the Dead

Bibliography

1. Odette Pollar. 365 Ways to Simplify Your Work Life: Ideas That Bring More Time, Freedom and Satisfaction to Daily Work. Chicago: Dearborn Financial Pub, 1996.
2. Chassot P. Stress in European operating room personnel. World Congress of Anesthesiologists, 2000 Proceedings 2000, 63–5.
3. Gaba DM, Howard SK, Jump B. Production pressure in the work environment. California anesthesiologists' attitudes and experiences. Anesthesiology 1994; 81:488–500.
4. Heim E. Stressors in health occupations. Do females have a greater health risk? New

Zealand Psychosom Med Psychoanal 1992; 38:207–26.

5. Leak JA. Stress management: communicating with the lion and the lamb. ASA Newsletter 2002; 66(11): 33–4.
6. Leak JA. Stress management: finding your purpose in the Ark. ASA Newsletter 2001; 65(11):27–8.
7. Leak JA. Stress management: slaying the dragon. ASA Newsletter Part I. 2000; 64(10):27–8.
8. Leak JA. Stress management: slaying the dragon. ASA Newsletter. Part II. 2000; 64(11):21–2.
9. Leak JA. Stress management: calming the lion. ASA Newsletter 1999; 63(8):21–2.

Emergency Ventilatory Management of the Trauma Patient: Elemental or Detrimental?

Paul E. Pepe, MD, MPH, FACEP, FCCP, FACP, FCCM

Professor of Surgery, Medicine, and Public Health and Riggs Family Chair in Emergency Medicine. The University of Texas Southwestern Medical Center and Parkland Health and Hospital System, Dallas

Medical Director, Dallas Metropolitan Medical Response System (MMRS) and Medical Director, Dallas Metropolitan Bio Tel (EMS) System

Learning Objectives: 1) To understand the differences in ventilatory techniques used during normal hemodynamic conditions versus those required during circulatory arrest/compromise, 2) To appreciate the potential detrimental effects of airway and ventilatory techniques commonly used by emergency care providers for cases of cardiac and trauma resuscitation, 3) To recognize the rationale and appropriate circumstances for recommended ventilatory strategies/adjuncts, both basic and advanced and 4) to learn improved strategies for delivering appropriate ventilatory techniques during cardiac and trauma resuscitations.

Emergency care providers have been trained to make airway management a priority in cardiac, respiratory, and trauma resuscitation. Nevertheless, most providers of emergency care, both in and out-of-hospital, often lack a fundamental understanding of ventilatory physiology during circulatory arrest/compromise. Furthermore, texts and guidelines for emergency respiratory care traditionally have been somewhat generic, generally emphasizing "hyperventilation," a concept that is also not well understood. The purpose of this talk is to review the physiology of ventilation in the unusual circumstances of circulatory arrest/compromise. The discussion will describe how both basic and advanced airway techniques can be life-saving if used properly, but can also be detrimental when traditional training techniques are followed too zealously. The lecturer will conclude with updated recommendations for the management of both cardiac and trauma resuscitation and will also review the value of each of the various airway adjuncts currently available to emergency care providers.

References

1. American College of Surgeons Committee on Trauma. Shock. In *Advanced Trauma Life Support Program for Physicians. Instructor Manual*. Chicago: ACS, 1993, pp 75–110.
2. Noc M, Weil MH, Tang W, et al. Mechanical ventilation may not be essential for initial cardiopulmonary resuscitation. *Chest* 1995; 108:821–7.
3. Idris AH, Staples ED, O'Brien DJ, et al. Effect of ventilation on acid-base balance and oxygenation in low blood-flow states. *Crit Care Med* 1994; 22:1827–34.
4. Pepe PE. Acute respiratory insufficiency. In Harwood-Nuss A et al, eds. *The Clinical Practice of Emergency Medicine*. Philadelphia, Lippincott, 1996, chapter 140; pp 636–40.
5. Pepe PE, Marini JJ. Occult positive end-expiratory pressure in mechanically-ventilated patients with airflow obstruction - the auto-PEEP effect. *Am Rev Resp Dis* 1982; 126:166–70.
6. Franklin C, Samuel J, Hu TC. Life-threatening hypotension associated with emergency intubation and the initiation of mechanical ventilation. *Am J Emerg Med* 1994; 12:425–8.
7. Durham LA, Richardson RJ, Wall MJ, Pepe PE, Mattox KL. Emergency center thoracotomy: impact of prehospital resuscitation. *J Trauma* 1992; 32:775–9.
8. Marion DW, Firlirk A, McLaughlin MR. Hyperventilation therapy for severe traumatic brain injury. *New Horizons* 1995; 3:439–47.
9. Pepe PE. Resuscitation of the patient with major trauma. *Curr Opin in Crit Care* 1995; 1:479–86.
10. Pepe PE. Preoperative fluid resuscitation for post-traumatic hemorrhage: elemental or detrimental? In Lawin P, Peter K, Prien T, eds. *Intensivmedizin*. New York, Stuttgart, Georg Thieme Verlag, 1995, pp 72–7.
11. Pepe PE. Controversies in resuscitation: to infuse or not to infuse (2). *Resuscitation* 1996; 31:7–10.
12. Bickell WH, Wall MJ, Pepe PE, et al. Immediate versus delayed fluid resuscitation for hypotensive patients with penetrating torso injury. *N Engl J Med* Oct. 27, 1994; 331:1105–9.
13. Pepe PE, Mattox KL, Fischer RP, Matsumoto CM. Geographical patterns of urban trauma according to mechanism and severity of injury. *J Trauma* 1990; 30:1125–32.
14. Cobb LA, Alvarez H, Copass MK. A rapid response system for out-of-hospital cardiac emergencies. *Med Clin North Am* 1976; 60:283–90.
15. McManus WF, Tresch DD, Darin JC. An effective prehospital emergency system. *J Trauma* 1977; 17:304–10.
16. Curka PA, Pepe PE, Ginger VF, et al. Emergency medical services priority dispatch. *Ann Emerg Med* 1993; 22:1688–95.
17. Applebaum D. Patient selection for advanced prehospital care in a two-level emergency medical system. *Prehosp Disast Med* 1989; 4:36.
18. Sanders AB, Kern KB, Berg RA, et al. Survival and neurologic outcome after cardiopulmonary resuscitation with four different chest compression-ventilation ratios. *Ann Emerg Med* 2002; 40:553–62.
19. Pepe PE, Raedler C, Lurie K, Wigginton J. Emergency ventilatory management in hemorrhagic states: elemental or detrimental? *J Trauma* 2003 (in press).
20. Pepe PE, Mosesso VN, Falk JL. Prehospital fluid resuscitation of the patient with major trauma. *Prehosp Emerg Care* 2002; 6(1):81–91.

Fluid Management in Trauma

Richard P. Dutton, MD

R Adams Cowley Shock Trauma Center, University of Maryland, Baltimore, Maryland, USA

Learning Objective: To better understand the anesthesiologist's role in controlling life-threatening hemorrhage.

Fluid resuscitation is a rapidly evolving area of trauma practice, particularly in early hemorrhagic shock (while the patient is still actively bleeding). The surgeons have brought new diagnostic and therapeutic options to the table, including FAST, angiography, and damage control techniques. What has anesthesia contributed?

I will briefly review the history and recent academic literature on early resuscitation, including the arguments for and against deliberate hypotensive management, early use of blood products, hypothermia, hypertonic resuscitation fluids, and pro-coagulants. The anesthesiologist plays a critical role in the application of each of these therapies and should be familiar with the evidence that supports their use.

I will conclude with a discussion of over-the-horizon approaches to hemorrhagic shock, including new diagnostic modalities, new treatment options, and new approaches to long-term resuscitation and prevention of organ system failure.

[Editors' note re "off-label" use: Dr. Dutton will discuss investigational and anecdotal use of dry fibrin sealant bandages, hemoglobin-based oxygen carriers, and recombinant FVIIa.]

Thursday, May 15, 2003

Simultaneous Afternoon Sessions

— Session A —

New Dimensions in Trauma and Critical Care

Co-Chair: James G. Cain, MD

Co-Chair: Christopher M. Grande, MD, MPH

Sedation for the Critically Injured Trauma Patient: Precedex®, a Novel Alternative

James Gordon Cain, MD

Allegheny General Hospital, Pittsburgh, Pennsylvania, and West Virginia University, Morgantown, West Virginia, USA

Learning Objective: To provide an introduction to dexmedetomidine, a newly introduced alpha 2 blocking sedative-analgesic, and its use in critically ill trauma patients.

Dexmedetomidine (Precedex®), a lipophilic imidazole derivative, selective alpha2 agonist (1300:1, a2:a1), is approved for use in initially intubated critically ill patients. Precedex® offers analgesia, sedation, inhibition of shivering, decreased sympathetic outflow, and decreased catecholamines without significantly decreasing respiratory drive. With this decrease in sympathetic outflow, mild to moderate hypotension and bradycardia may occur. Precedex® does not have a direct myocardial effect. A novel aspect of Precedex® is that, compared with an equal level of baseline sedation with standard agents, it allows easy arousability. This would be particularly advantageous in facilitating reproducible, serial neurologic examinations at will in patients with TBI while avoiding the drastic swings in sedation with volatile hemodynamics associated with the current propofol-based technique. Dexmedetomidine reduced propofol and morphine requirements and improved hemodynamic stability during bispectral (BIS) index-guided intensive care unit sedation. Additional benefits of Precedex® may be a modest decrease in cerebral blood flow, along with additional neuroprotective properties in its own right.

Bibliography

- Hall JE, Uhrich TD, Barney JA, et al. Sedative, amnestic, and analgesic properties of small-dose dexmedetomidine infusions. *Anesth Analg* 2000; 90:699-705.
- Housmans PR. Effect of dexmedetomidine on contractility, relaxation, and intracellular calcium transients of isolated ventricular myocardium. *Anesthesiology* 1990; 73:919-22.
- Jolkkonen J, Puurunen K, Koistinaho J, et al. Neuroprotection by the alpha2-adrenoreceptor agonist, dexmedetomidine, in rat focal cerebral ischemia. *Eur J Pharmacol* 1999; 372:31-6.
- Kuhmonen J, Pokorny J, Miettinen R, et al. Neuroprotective effects of dexmedetomidine in the gerbil hippocampus after transient global ischemia. *Anesthesiology* 1997; 87:371-7.
- Prielipp RC, Wall MH, Tobin JR, et al. Dexmedetomidine-induced sedation in volunteers decreases regional and global blood flow. *Anesth Analg* 2002; 95:1052-9.
- Takroui MS, Seraj MA, Channa AB, et al. Dexmedetomidine in the intensive care unit: a study of hemodynamic changes. *Middle East J Anesthesiol* 2002; 16:587-95.
- Talke P, Li J, Jain U, et al. Effects of perioperative dexmedetomidine infusion in patients undergoing vascular surgery. The Study of Perioperative Ischemia Research Group. *Anesthesiology* 1995; 82:620-33.
- Tiitisch AE, Welte M, von Homeyer P, et al. Bispectral index-guided sedation with dexmedetomidine in intensive care: a prospective randomized, double blind, placebo-controlled phase II study. *Crit Care Med* 2002; 30:1007-14.
- Venn RM, Bradshaw CJ, Spencer R, et al. Preliminary UK experience of dexmedetomidine, a novel agent for postoperative sedation in the intensive care unit. *Anaesthesia* 1999; 54:1136-42.
- Venn RM, Bryant A, Hall GM, Grounds RM. Effects of dexmedetomidine on adrenocortical function, and the cardiovascular, endocrine and inflammatory responses in post-operative patients needing sedation in the intensive care unit. *Br J Anaesth* 2001; 86:650-6.
- Venn RM, Hell J, Grounds RM. Respiratory effects of dexmedetomidine in the surgical patient requiring intensive care. *Crit Care* 2000; 4:302-8.

The Hazards of Nutraceuticals in the Management of the Trauma Patient

Jessie A. Leake, MD

Associate Professor of Anesthesiology

University of Texas, MD Anderson Cancer Center, Houston, Texas, USA

Learning Objectives: 1) To outline the herbs and dietary supplements (nutraceuticals) that are most commonly used in the preoperative phase; 2) to discuss the various preparations in which these products are available; 3) to broadly cover the state of gov-

ernment regulation of these products; and 4) most importantly, to target some of the more important potential drug-nutraceutical interactions and hazards, particularly as they may relate to the care of the patient receiving anesthesia.

Because of the paucity of government regulation regarding purity, contents, manufacturing, and health claims as well as pharmacologic and physiologic predictability, it is difficult to fund and perform double-blind, placebo-controlled studies on these products. For this reason, much of the material concerning potential hazards and adverse drug interactions is based on extrapolation, anecdote, and/or uncontrolled case studies on active ingredients that are not consistently available in the products that we purchase in our local pharmacies, grocery stores, and gyms.

SUMMARY OF POTENTIAL INTRAOPERATIVE COMPLICATIONS

Untoward cardiovascular effects	Ephedra Ginseng Licorice St. John's Wort (potential indirect effect) Vitamin E Triiodothyroacetic acid GBL, BD, GHB
Enhanced bleeding potential	Ginseng Ginkgo Ginger Garlic Feverfew Vitamin E
Potential for prolongation of anesthesia	Valerian Kava-kava St. John's Wort (anecdotal only)
Possible renal insufficiency or hepatotoxicity	Licorice (renal) Creatine (renal) Echinacea (hepatic) Kava-kava (hepatic)
Possible abnormal thyroid functions	Triiodothyroacetic acid Vitamin E
Potential for electrolyte disturbances	Goldenseal Licorice
Risk of decreased effectiveness of HIV protease inhibitors	St. John's Wort

SUMMARY OF INDIVIDUAL HERBS AND SUPPLEMENTS

Name	Common Uses	Potential Side Effects
Echinacea	Common cold, bronchitis	Possibly hepatotoxic. May decrease effectiveness of corticosteroids
Ephedra	Diet aid; antitussive	Death. Cardiovascular instability. Multiple drug interactions
Feverfew	Migraine prophylactic	May inhibit platelet activity and increase bleeding. Avoid use in pts on anticoagulants.
Garlic	Lipid & BP lowering; Antiplatelet, antioxidant, antithrombotic qualities	May potentiate warfarin and increase bleeding; affects platelet aggregation.
Ginger	Antinauseant	May be a potent inhibitor of thromboxane synthetase and may increase bleeding time. Use with caution with warfarin.
Ginkgo	Circulatory stimulant	May enhance bleeding in pts on anticoagulant or antithrombotic therapy.
Ginseng	"Adaptogenic" Energy level enhancer Antioxidant	Avoid use with other stimulants—may experience tachycardia or hypertension. May increase bleeding, especially in patients on anticoagulant or antithrombotic therapies. Known to have anti-platelet properties.
Goldenseal	Diuretic; anti-inflammatory	Functions as aquaretic, not as diuretic; see no sodium excretion, just free water excretion. May worsen edema or hypertension.
Kava-kava	Anxiolytic	May potentiate barbiturates and prolong anesthesia.
Licorice	Gastric and duodenal ulcers	Glycyrrhizic acid in licorice may cause high blood pressure, low potassium, and edema. Contraindicated in renal insufficiency, liver conditions, hypertonia.
St. John's Wort	Mild to moderate depression Anxiety	May prolong effects of anesthesia (anecdotal only). May decrease effectiveness of all HIV protease inhibitors as well as all nonnucleoside reverse transcriptase inhibitors. May decrease blood levels of digoxin.
Valerian	Sedative	May potentiate barbiturates.