Treatment of Local Anesthetic Cardiac Toxicity

AAQ
Montreal
16 Avril 2013
Guy Weinberg, MD
Chicago, IL
Topics for Today

LAST

LIPID

FUTURE CHALLENGES
Classic Treatments for LAST Have Miserable Outcomes
Epidemiology

Mulroy M. Systemic Toxicity and Cardiotoxicity From Local Anesthetics: Incidence and Preventive Measures. Regional Anesthesia and Pain Medicine, 2002; 27: 556–561

INCIDENCE SYSTEMIC TOXIC REACTIONS

- ~7.5 – 20/10,000 PERIPHERAL NERVE BLOCKS
- ~1/10,000 EPIDURAL ANESTHESIA

maybe higher... under-reporting and misdiagnosis

maybe lower now ... USGRA

Cardiac arrest with PNB ~1/5000 (MH ~ 1/20,000-50,000 ~3 deaths/year).
The danger of experiential practice:
Rare, uncommon events can be viewed as irrelevant
We’re all familiar with the refrain……
“That’s never happened to me”
As though, ‘that never could happen to me’

Remember, inference, like hubris, is not our friend
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Claims with eye or peripheral nerve blocks performed perioperatively from 1980 through 2000 were analyzed. The injury patterns associated with peripheral nerve blocks and payment factors were analyzed. Local anesthetic toxicity was associated with 7 of 19 claims with death or brain damage.
Emerging Patterns of Local Anesthetic Toxicity

• Non-anesthesia providers
• Office and off-site
• Atypical presentation
• Susceptible populations:
  – Elderly, metabolic disease
  – Very low cardiac output
  – Interval events: tourniquet, acidosis

• CNS: Extremely variable presentation
• Cardiac: variable and variable timing (late LAST)
  – Any arrhythmia;
  – Progressive bradycardia and hypotension
Cardiac History in Reports of Lipid Rescue from LA Toxicity

= Susceptible Groups:

Rosenblatt: prior MI, RBBB and LAHB, angina
Litz I: LABB, Stokes attacks, MVR, TVR
Warren: S/P MI, RBBB, HTN
Foxall: stable angina, QwV1-3, PAC
Litz II: CAD, MVR
Smith: S/P CABG, 2 stents

Note the unintended benefits of LipidRescue Resuscitation:
More reports; clearer definition of clinical phenotype of LA toxicity.
More insights into underlying mechanism and difference from lab models
A Close Call in a Patient with a Metabolic Disease

20 yo patient with isovaleric acidemia: During SQ injection of bupivacaine, he develops arrhythmia with systolic BP 70, then complete heart block, then VT. Bupivacaine dose = 22mg.

Patient is severe carnitine deficiency

Carnitine

REQUIRED FOR FATTY ACID TRANSPORT INTO MITOS AND ATP SYNTHESIS FROM HEART’S PREFERRED FUEL

Biochemical Properties of Subsarcolemmal and Interfibrillar Mitochondria Isolated from Rat Cardiac Muscle*

(Received for publication, July 29, 1977)

June W. Palmer, & Bernard Tandler, and Charles L. Hoppel§

From the Veterans Administration Hospital and Departments of Pharmacology and Medicine, School of Medicine, and the Department of Oral Biology and Medicine, School of Dentistry, Case Western Reserve University, Cleveland, Ohio 44106
Substrate Utilization

Bupivacaine
Control vs. Lipid Rescue (15mg/kg)

Simplest Demonstration Possible
Translation of laboratory findings to the clinical setting

... the first successful use of Lipid

Successful Use of a 20% Lipid Emulsion to Resuscitate a Patient after a Presumed Bupivacaine-related Cardiac Arrest

Meg A. Rosenblatt, M.D.,* Mark Abel, M.D.,† Gregory W. Fischer, M.D.,† Chad J. Itzkovich, M.D.,‡ James B. Eisenkraft, M.D.§

Anesthesiology 2006; 105:217-8

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INTERSCALENE BLOCK  20ml Bupivacaine 0.5% - 20ml Mepivacaine 1.5%
Within 30 sec seizures (Propofol 50+100mg); 90 sec later CARDIAC ARREST…
CPR,O2-ventilation,epinephrine,atropine,amiodarone,vasopressin, defibrillation…
PULSELESS VT… Within 15 sec OF LIPID pulse/blood pressure detectable

YES!!
Meg Rosenblatt (RAINER) saves the day
CASE REPORT
Successful resuscitation of a patient with ropivacaine-induced asystole after axillary plexus block using lipid infusion*

R. J. Litz, M. Popp, S. N. Stehr and T. Koch

CASE REPORT
Levobupivacaine-induced seizures and cardiovascular collapse treated with Intralipid®

G. Foxall,1 R. McCahon,2 J. Lamb,3 J. G. Hardman4 and N. M. Bedforth3

Kardio- und neurotoxische Nebenwirkungen nach akzidenteller intravasaler Bupivacainapplikation
Therapie mit Lidocain, Propofol und Lipidemulsions

Lipid Reversal of Central Nervous System Symptoms of Bupivacaine Toxicity
Andrew G. Spence, F.R.C.A., King Edward VII Memorial Hospital, Hamilton, Bermuda. aspence@transact.bm
ADOPTION OF LIPIDRESCUE IN LONDON HOSPITALS

Picard et al, Anaesthesia, 2009
LipidRescue™ Resuscitation
... for drug toxicity

Welcome

LipidRescue™ resuscitation refers to the use of an intravascular infusion of a lipid emulsion to treat severe, systemic drug toxicity or poisoning. It was originally developed to treat local anesthetic toxicity, a potentially fatal complication of regional anesthesia that can occur in other situations where patients receive local anesthetic injections. More recently, LipidRescue™ has been shown to help reviewed medical literature and elsewhere to be an effective antidote for poisoning or overdose caused by a wide array of other (not local anesthetic) lipophilic agents. Initial support for this view was provided by a most remarkable case report where lipid emulsion infusions apparently saved a patient from overwhelming hypoxia and overdose. Since then, evidence from both laboratory models and case reports, indicates that LipidRescue™ can effectively in treat a wide variety of non-local anesthetic overdoses, including reversal of both cardiovascular and central nervous system (CNS) signs and symptoms of toxicity.

This site was established in 2006 to serve as a source of information on LipidRescue™ methodology and related issues. The intent was to provide a venue for the robust exchange of ideas on topics including the mechanisms, epidemiology, diagnosis, presentation, prevention and treatment of life-threatening local anesthetic overdose and other types of severe cardiac and CNS toxicity.

Content includes the aforementioned aspects of local anesthetic toxicity plus links to related educational sites, typical experiments from my laboratory and other information.
In summary, there is convincing anecdotal and experimental evidence that IFE is effective in treating toxicity caused by local anesthetics.

"..Current evidence suggests that IFE should be administered as soon as a diagnosis of local anesthetic toxicity is established, ..."
Practice Advisory on Treatment of Local Anesthetic Systemic Toxicity

For Patients Experiencing Signs or Symptoms of Local Anesthetic Systemic Toxicity (LAST)

- Get Help
- Initial Focus
  - **Airway management**: ventilate with 100% oxygen
  - **Seizure suppression**: benzodiazepines are preferred
  - **Basic and Advanced Cardiac Life Support (BLS/ACLS)** may require prolonged effort
- **Infuse 20% Lipid Emulsion** (values in parenthesis are for a 70 kg patient)
  - **Bolus 1.5 mL/kg** (lean body mass) intravenously over 1 min (~100 mL)
  - **Continuous infusion at 0.25 mL/kg/min** (~18 mL/min; adjust by roller clamp)
  - Repeat bolus once or twice for persistent cardiovascular collapse
  - Double the infusion rate to 0.5 mL/kg per minute if blood pressure remains low
  - **Continue infusion** for at least 10 mins after attaining circulatory stability
  - Recommended upper limit: approximately 10 mL/kg lipid emulsion over the first 30 mins
- **Avoid** vasopressin, calcium channel blockers, β-blockers, or local anesthetic
- **Alert** the nearest facility having cardiopulmonary bypass capability
- **Avoid propofol** in patients having signs of cardiovascular instability
- **Post LAST events** at www.lipidrescue.org and report use of lipid to www.lipidregistry.org
Part 12: Cardiac Arrest in Special Situations
2010 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care
Terry L. Vanden Hoek, Chair; Laurie J. Morrison; Michael Shuster; Michael Donnino; Elizabeth Sinz; Eric J. Lavonas; Farida M. Jeejeebhoy; Andrea Gabrielli

This section of the 2010 AHA Guidelines for CPR and ECC addresses cardiac arrest in situations that require special treatments or procedures beyond those provided during basic life support (BLS) and advanced cardiovascular life support (ACLS). We have included 15 specific cardiac arrest situations. The first several sections discuss cardiac arrest associated with internal physiological or metabolic conditions, such as asthma (12.1), anaphylaxis (12.2), pregnancy (12.3), morbid obesity (12.4), pulmonary embolism (PE) (12.5), and electrolyte imbalance (12.6).

The next several sections relate to resuscitation and treatment of cardiac arrest associated with external or environmentally related circumstances, such as ingestion of toxic substances (12.7), trauma (12.8), accidental hypothermia (12.9), avalanche (12.10), drowning (12.11), and electric shock/lightning strikes (12.12).

The last 3 sections review management of cardiac arrest that may occur during special situations affecting the heart, including percutaneous coronary intervention (PCI) (12.13), cardiac tamponade (12.14), and cardiac surgery (12.15).

Part 12.1: Cardiac Arrest Associated With Asthma
Asthma is responsible for more than 2 million visits to the Pathophysiology
The pathophysiology of asthma consists of 3 key abnormalities:
- Bronchoconstriction
- Airway inflammation
- Mucous plugging

Complications of severe asthma, such as tension pneumothorax, lobar atelectasis, pneumonia, and pulmonary edema, can contribute to fatalities. Severe asthma exacerbations are commonly associated with hypercarbia and acidemia, hypotension due to decreased venous return, and depressed mental status, but the most common cause of death is asphyxia. Cardiac causes of death are less common.4

Clinical Aspects of Severe Asthma
Wheezeing is a common physical finding, although the severity of wheezing does not correlate with the degree of airway obstruction. The absence of wheezing may indicate critical airway obstruction, whereas increased wheezing may indicate a positive response to bronchodilator therapy.

Oxygen saturation (SaO2) levels may not reflect progressive alveolar hypoventilation, particularly if oxygen is being administered. Note that SaO2 may fall initially during therapy because β2-agonists produce both bronchodilation and vasodilation and
RECOMMENDATIONS:

• Oxygenation/ Ventilation!
• CPR
• Seizure Control (BDZ preferred)
• 20% LIPID 1.5ML/KG BOLUS
• INFUSION 0.25-0.5ML/KG/MIN X 30min
• REPEAT BOLUS x 2 IF NEEDED
• NO Propofol, CC or β Blockers, LAs
• Ready Cardiopulmonary Bypass *
• Don’t give up – bupivacaine is protective
1. “Such accidents are more frequent than is commonly supposed”
2. “Unexplained differences in susceptibility exist”
3. “A proportion of cases are avoidable mistakes”
4. “In treatment, the first place must be assigned to artificial respiration, perhaps with cardiac massage”
5. The committee advises strongly against the routine use of morphin and epinephrin in the treatment of accidents”
Lipid vs Pressors

Lipid

Epi

ADH
Lipid + Epinephrine: Paradox of Initial Recovery
Epinephrine Impairs Outcome
Things you should know about ILE

• Need 20%, start with bolus then follow
• Dose to ideal body weight (esp in US)
• Infusion rate is not precise (roller clamp OK)
• Don’t forget to turn it off (12mL/kg/30min)
• Works with ALL local anesthetics
• Early is better
• Safe – scant evidence of adverse events
• Less pressor therapy (NO vasopressin)
• Severe CAD can impair efficacy of ILE
Mazoit et al. ASA ’08 in vitro lipid LA binding

SO, HOW DOES IT WORK?
Rapid binding of local anesthetics by lipid emulsions

This is *in vitro* experiment shows very rapid drop of ‘free’ (nonlipid-bound) local anesthetic concentration in 1% solutions of Intralipid or Medialipid over time. The starting concentrations of both local anesthetics is 125mg/L. Intralipid binds more anesthetic than Medialipid and each lipid binds more bupivacaine than ropivacaine.

Typical initial lipid resuscitation bolus: 100mL of 20% = 20g fat

Total fat in a single serving of chicken soup: 27.2g
But, there is more to lipid than a sink

Phosphorylation of GSK-3β Mediates Intralipid-induced Cardioprotection against Ischemia/Reperfusion Injury

Siamak Rahman, M.D.,* Jingyuan Li, M.D., Ph.D.,† Jean Chrisostome Bopassa, Ph.D.,‡ Soban Umar, M.D., Ph.D.,† Andrea Iorga, B.Sc.,‡ Parisa Partownavid, M.D.,§ Mansoureh Eghbali, Ph.D.‖
RESEARCH ARTICLE

Fatty acids antagonize bupivacaine - induced I_{Na} blockade

ALLAN R. MOTTRAM¹, CARMEN R. VALDIVIA², and JONATHAN C. MAKEILSKI²

¹University of Wisconsin, Division of Emergency Medicine, 600 Highland Ave, F2/204 CSC, MC 3280, Madison, United States
²University of Wisconsin, Department of Medicine, 600 Highland Ave, Madison, United States
“Intravenous lipid emulsion infusion reduced the context-sensitive half-life of total plasma bupivacaine from 45 min to 25 min; p = 0.01. We observed no significant adverse effects of lipid emulsion. In conclusion, lipid emulsion may slightly increase the rate of bupivacaine tissue distribution. No ‘lipid sink’ effect was observed with the non-toxic dose of bupivacaine used”.
DIRECT CARDIOVASCULAR BENEFIT
Myocardial Accumulation of Bupivacaine and Ropivacaine Is Associated with Reversible Effects on Mitochondria and Reduced Myocardial Function

Nicole Hiller, MSc,* Peter Mirtschink, MD,† Christine Merkel, MD,* Lilla Knels, MD,‡ Reinhard Oertel, PhD,§ Torsten Christ, MD,|| Andreas Deussen, MD,¶ Thea Koch, MD,* and Sebastian N. Stehr, MD, DESA*
Mechanisms of Lipid Rescue
-More than a Sink
Mechanism(s) Summary

INTRACELLULAR (METABOLIC)

INTRAVASCULAR (SINK + Redistribution)

MEMBRANE (CHANNEL OR SIGNALLING)
SO, WHAT’S NEW?

ORIGINAL ARTICLE

ASRA Checklist Improves Trainee Performance During a Simulated Episode of Local Anesthetic Systemic Toxicity

Joseph M. Neal, MD,* Robert L. Hsiung, MD,* Michael F. Mulroy, MD,* Brian B. Halpern, RN,† Alison D. Dragnich, MD,* and April E. Slee, MSc‡

Or Controversial

Pediatric Anesthesia

ORIGINAL ARTICLE

Comparison of epinephrine vs lipid rescue to treat severe local anesthetic toxicity – an experimental study in piglets

Jacqueline Mauch¹,², Olga Martin Jurado³, Nelly Spielmann¹, Regula Bettschart-Wolfensberger³ & Markus Weiss¹

1 Department of Anesthesia, University Children’s Hospital Zurich, Zurich, Switzerland
2 Department of Anesthesia and Perioperative Medicine, Kantonsspital Aarau, Aarau, Switzerland
3 Section Anesthesiology, Equine Department, Vetsuisse Faculty University of Zurich, Zurich, Switzerland
# Checklist for Treatment of Local Anesthetic Systemic Toxicity (LAST)

The Pharmacologic Treatment of Local Anesthetic Systemic Toxicity (LAST) is Different from Other Cardiac Arrest Scenarios

- Get Help
- Initial Focus
  - Airway management: ventilate with 100% oxygen
  - Seizure suppression: benzodiazepines are preferred; AVOID propofol in patients having signs of cardiovascular instability
  - Alert the nearest facility having cardiopulmonary bypass capability
- Management of Cardiac Arrhythmias
  - Basic and Advanced Cardiac Life Support (ACLS) will require adjustment of medications and perhaps prolonged effort
  - AVOID vasopressin, calcium channel blockers, beta blockers, or local anesthetic
  - REDUCE individual epinephrine doses to <1 mcg/kg
- Lipid Emulsion (20%) Therapy (values in parenthesis are for 70kg patient)
  - Bolus 1.5 mL/kg (lean body mass) intravenously over 1 minute (~100mL)
  - Continuous infusion 0.25 mL/kg/min (~18 mL/min; adjust by roller clamp)
  - Repeat bolus once or twice for persistent cardiovascular collapse
  - Double the infusion rate to 0.5 mL/kg/min if blood pressure remains low
  - Continue infusion for at least 10 minutes after attaining circulatory stability
  - Recommended upper limit: Approximately 10 mL/kg lipid emulsion over the first 30 minutes
- Post LAST events at [www.lipidrescue.org](http://www.lipidrescue.org) and report use of lipid to [www.lipidregistry.org](http://www.lipidregistry.org)
Administering epinephrine resulted in an overshoot in MAP over baseline by factor 1.5–2.5 and in heart rate by factor 1.4–1.9. With epinephrine administration, self-limiting tachy-arrhythmias were observed, sometimes transiently compromising blood pressure.

For the treatment of severe hemodynamic compromise owing to bupivacaine intoxication in piglets, first-line rescue with epinephrine was more effective than Intralipid.
Case Report
Successful resuscitation of bupivacaine-induced cardiotoxicity in a neonate

ERICA P. LIN MD AND LORI A. ARONSON MD
Department of Anesthesiology, Cincinnati Children’s Hospital Medical Center, Cincinnati, OH, USA
Archie Sirianni saved the day
>90 Minutes Total CPR

1min after 100mL lipid, ROSC, CPR stopped, QRS narrows. Patient recovers, neurologically intact.
Toxicologists Now Aware of Lipid Therapy

Use of Lipid Emulsion in the Resuscitation of a Patient With Prolonged Cardiovascular Collapse After Overdose of Bupropion and Lamotrigine

Archie J. Sirianni, MD  
Kevin C. Osterhoudt, MD  
Diane P. Calello, MD  
Allison A. Muller, PharmD  
Marie R. Waterhouse, MD  
Michael B. Goodkin, MD  
Guy L. Weinberg, MD  
Fred M. Henretig, MD

From the Department of Anesthesiology (Sirianni) and Division of Cardiology (Goodkin), Riddle Memorial Hospital, Media, PA; the Department of Pediatrics, University of Pennsylvania School of Medicine and The Children’s Hospital of Philadelphia, Philadelphia, PA (Osterhoudt, Calello, Muller, Waterhouse, Henretig); the Section of Clinical Toxicology, Division of Emergency Medicine, and the Poison Control Center, The Children’s Hospital of Philadelphia, Philadelphia, PA (Osterhoudt, Calello, Muller, Henretig); and the Department of Anesthesiology, University of Illinois College of Medicine at Chicago, and Jessie Brown VA Medical Center, Chicago, IL (Weinberg).

Animal studies show efficacy of intravenous lipid emulsion in the treatment of severe cardiotoxicity associated with local anesthetics, clomipramine, and verapamil, possibly by trapping such lipophilic drugs in an expanded plasma lipid compartment (“lipid sink”). Recent case reports describe lipid infusion for the successful treatment of refractory cardiac arrest caused by parenteral administration of local anesthetics, but clinical evidence has been lacking for lipid’s antidotal efficacy on toxicity caused by ingested medications. A 17-year-old girl developed seizure activity and cardiovascular collapse after...
Case report: successful lipid resuscitation in multi-drug overdose with predominant tricyclic antidepressant toxidrome

Martyn Harvey¹ and Grant Cave²
CONCLUSIONS: These data show that the effects of lipid infusion on LA-induced cardiac arrest are strongly dependent on the administered LAs itself. We conclude that lipophilicity of LAs has a marked impact on the efficacy of lipid infusions to treat cardiac arrest induced by these drugs.
Intractable cardiac arrest due to lidocaine toxicity successfully resuscitated with lipid emulsion

Stephanie K. Dix, MD; Gregg F. Rosner, MD; Monica Nayar, PharmD; Julian J. Harris, MD; Maya E. Guglin, MD; Jeffery R. Winterfield, MD; Zhiling Xiong, MD, PhD; Gilbert H. Mudge, Jr., MD

Use of Intravenous Lipid Emulsion to Reverse Central Nervous System Toxicity of an Iatrogenic Local Anesthetic Overdose in a Patient on Peritoneal Dialysis

D Bruce Lange, Daniel Schwartz, Gerald DaRoza, and Robert Gair

Therefore, at approximately 30 minutes from the initial signs of lidocaine toxicity, a 20% lipid emulsion (Intralipid, Fresenius Kabi) was administered as a 1.5-mL/kg intravenous bolus (100 mL) over approximately 10 minutes. Within approximately 5 minutes (ie, after 50 mL of lipid was infused), the patient became more alert and exhibited improved muscle function. In approximately 10 minutes (after 100 mL), the patient was able to speak coherently and
CASE REPORT

Treatment of cocaine overdose with lipid emulsion

R. Jakkala-Saibaba,¹ P. G. Morgan² and G. L. Morton²

¹ Specialist Registrar, 2 Consultant, John Hammond Department of Anaesthesia, East Surrey Hospital, Redhill, UK

Summary
We describe the management and recovery of a 28-year-old man following a history of overdose by nasal inhalation of cocaine. The patient was presented in a comatose state suffering from seizures and marked cardiovascularly instability. Intravenous lipid emulsion was administered following initial resuscitation and tracheal intubation, as a means of treating persistent cardiac arrhythmias and profound hypotension. Following lipid emulsion therapy, the patient's life-threatening cardiovascular parameters rapidly improved and he recovered well without any side effects, thus being discharged within 2 days.
Usefulness of Intravenous Lipid Emulsion for Cardiac Toxicity from Cocaine Overdose

Natasha Purai Arora, MD\textsuperscript{a}, William Allen Berk, MD\textsuperscript{b}, Cynthia Kurke Aaron, MD\textsuperscript{c,d},
and Kim Allan Williams, MD\textsuperscript{e,*}

Figure 1. Initial electrocardiogram showing wide-complex tachycardia (heart rate 143 beats/min, QRS duration 148 ms) with a prolonged corrected QT interval of 595 ms.
Figure 3. Electrocardiogram immediately after infusion of ILE showing regular sinus rhythm (heart rate 118 beats/min) with normal QRS (82 ms) and corrected QT (412 ms) intervals.
Figure 9. Arterial pressure. Bar, 1 min. First arrow, cocaine 5mg/kg; second arrow, lipid.

Figure 10. Continuous ECG. Each strip is 5 sec; arrow, lipid.
Intraosseous injection of lipid emulsion reverses bupivacaine-induced cardiovascular toxicity in rats: a preliminary observation.
Successful resuscitation from bupivacaine-induced cardiovascular collapse with intravenous lipid emulsion following femoral nerve block in an emergency department.

Martyn Harvey,1 Grant Cave,2 Giles Chanwai1 and Tonia Nicholson1
1Waikato Hospital, Hamilton and 2Hutt Hospital, Lower Hutt, New Zealand
Kensley Kirby, 5-Year-Old Atlanta Girl, Dies From Lethal Dose Of Local Anesthetic
After the three-hour procedure, Sobrino left the clinic, without ensuring his patient was in stable condition, investigators said. They said he left Javellana with no nurse, only a medical assistant, and without discharge instructions or a follow-up phone number.

After the procedure, Javellana vomited and was drowsy. She was told an adult should pick her up and stay with her for 24 hours, but not the reason. When no one came to pick up Javellana, Sono Bello staff put her in a cab alone, with no idea of where she was going, the state said. Javellana died in a hotel room the next day. The King County Medical Examiner ruled she died from 'acute lidocaine intoxication'. 
Investigators: Autopsy reveals overdose caused cosmetic surgery death

by LINDA BYRON / KING 5 News
Are New Laws or Regulations Required To Improve Safety in ‘Cosmetic Surgery’?

A144
October 14, 2012
10:00:00 AM - 11:30:00 AM
Room 103A

Are State Regulations for Liposuction Preventing Deaths? The Case of Los Angeles County

Selma Calmes, M.D., J. Daniel Augustine, M.D.
Los Angeles County, Los Angeles, California, United States

INTRODUCTION: Numerous deaths due to liposuction have been reported since the technique was introduced in the US in 1962. Reported causes of death included bacterial infections, bleeding, perforation of abdominal viscera, pulmonary embolus (venous or fat), lidocaine toxicity and fluid overload. As a result, 20 states established regulations for liposuction. California’s regulations began in 1990 and revised in 2003. These emphasize volume of fat removed, monitors and procedure duration. Are these regulations preventing deaths? We reviewed all deaths determined by the Los Angeles County Coroner’s office to be from liposuction.
Conclusions

• LAST occurs despite our best efforts
• Preparation makes a difference
• Lipid infusion makes a difference
• Education makes a difference
• Let our colleagues know about LAST