John G. Brock-Utne

Clinical Anesthesia

Near Misses and Lessons Learned Second Edition



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Foreword

Training of anesthesiologists has changed dramatically during my 20+ years in academics. More and different clinical rotations have become required for residents, often at the expense of time spent in the relatively "mundane" environment of the operating room. But the operating room and similar sites are where most anesthesiologists will practice their specialty. With less time for training, how can anyone possibly learn how to respond to every unusual, rare, and occasionally lifethreatening anesthetic emergency that can arise during routine clinical care? Simulation labs have been a wonderful addition, but often it is little tricks combined with outside of the box thinking, that is, the fine polish needed to become a true consultant in anesthesiology.

In the newest edition of *Clinical Anesthesia: Near Misses and Lessons Learned*, Dr. John Brock-Utne presents multiple examples of real, unique, and unanticipated clinical scenarios, where his quick thinking and years of experience prevented near catastrophe. Each chapter is a single case, with a description of the background and the problem encountered. More importantly, Dr. Brock-Utne presents the solution to the problem (often quite simple and elegant), a summary of the lessons learned, and references. Like telling stories around a campfire, Stanford attendings and residents alike have always enjoyed listening to Dr. Brock-Utne tell his "war stories." While reading this book, I could almost hear his voice. Now, a new and larger audience can profit from his knowledge and expertise, like many of us have benefited over the years at Stanford University. The only question I have is how does he always seem to be in the middle of such craziness? Better him than me, because he always knows how to get out of a jam and I have learned much from him over the years. Enjoy.

Department of Anesthesiology, Perioperative and Pain Medicine, Stanford University, Stanford, CA, USA Timothy Angelotti

Preface to the Second Edition

Dear reader,

Some of you may have read the first edition of *Clinical Anesthesia: Near Misses and Lessons Learned* published by Springer in 2008. Unfortunately the layout of the first edition had the clinical problem and the solution on the *same* page. This was obviously not ideal.

Hence, I was delighted when Rebekah Collins, senior medical editor at Springer, suggested a new edition in which they would correct the layout of the book, i.e., have the problem on one page and the solution with a discussion of the problem on a separate page. This layout had been successfully used in the following three books that I have written, namely, the first edition (1999) and second edition (2013) of *Near Misses in Pediatric Anesthesia* and first edition (2011) of *Case Studies in Near Misses in Clinical Anesthesia*.

The first edition of *Clinical Anesthesia: Near Misses and Lessons Learned* had 62 cases. In this second edition, those 62 cases have been modernized with updated references. Forty-one new cases have been added totaling 103 cases. I sincerely hope you will enjoy mulling over these clinical anesthesia problems as much as I have enjoyed writing them.

At times the solutions would seem simplistic to some readers. This will of course depend on their level of training or experience. For this I do apologize. However, the suggested management of the cases may be controversial, for this I do not apologize. My sincere hope is that these cases may form the bases for a teaching discussion between faculty member and medical students and resident/fellows in training in anesthesiology and critical care.

To paraphrase Hippocrates:

The art is long, Life is short; Experiment perilous, Decisions difficult.

After 47 years in clinical anesthesia, I honestly can say that Hippocrates was right.

Stanford, CA, USA

John G. Brock-Utne

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Last, but not least, my wife, Sue, our three boys, their wives, and our five grandsons and one granddaughter.

Contents

Case 1: No Fiber-Optic Intubation System: A Potential Problem	1
Case 2: Is the Patient Extubated?	3
Case 3: A Strange Computerized ECG Interpretation	7
Case 4: An Elderly Lady with a Fractured Neck of the Femur	11
Case 5: A Spinal Anesthetic that Wears Off Before Surgery Ends – What to Do?	15
Case 6: Just a Simple Monitored Anesthesia Care (MAC) Case	19
Case 7: Smell of Burning in the Operating Room	23
Case 8: A Diabetic Patient for Inguinal Hernia Repair	27
Case 9: The Case of the "Hidden" IV	31
Case 10: Postoperative Painful Eye	33
Case 11: Awake Craniotomy	35
Case 12: Gum Elastic Bougie	37
Case 13: You Smell Anesthesia Vapor – Where Is It Coming From?	41
Case 14: Manual Ventilation of a Patient Turned 180 Degrees Away from the Anesthesia Machine by a Single	
Operator – Is it Possible?	43
Case 15: Life-Threatening Arrhythmia in a 5-Month-Old	47
Case 16: Tongue Ring	53
Case 17: Hasty C-Arm Positioning – A Recipe for Disaster	57
Case 18: Inability to Remove a Nasogastric Tube	61
Case 19: An Unusual Cause of Difficult Tracheal Intubation	63

Case 20: Pulmonary Edema Following Abdominal Laparoscopy	67
Case 21: A Possible Solution to a Difficult Laryngeal Mask Airway Placement	71
Case 22: Postoperative Airway Complication Following Sinus Surgery	75
Case 23: An Unusual Capnograph Tracing	79
Case 24: A Respiratory Dilemma During a Transjugular Intrahepatic Porto-Systemic Shunt (TIPSS) Procedure	83
Case 25: A Tracheotomy Is Urgently Needed and You Have Never Done One	87
Case 26: General Anesthesia for a Patient with a Difficult Airway and Full Stomach	91
Case 27: A Jehovah's Witness Patient and a Potentially Bloody Operation	95
Case 28: Laparoscopic Achalasia Surgery	99
Case 29: Sudden Intraoperative Hypotension	101
Case 30: Blood Pressure Difference Between a Noninvasive and an Invasive Blood Pressure Measurement	105
Case 31: Severe Decrease in Lung Compliance During a Code Blue.	109
Case 32: Shortening Postanesthesia Recovery Time After an Epidural. Is It Possible?	113
Case 33: At Times You Need to Be a MacGyver.	117
Case 34: Delayed Cutaneous Fluid Leak from a Puncture Hole After Removal of an Epidural Catheter	121
Case 35: Traumatic Hemothorax and Same-side Central Venous Access	125
Case 36: A Single Abdominal Knife Wound. Easy Case?	129
Case 37: A -Over Vaporizer with a Non-rebreathing Circuit	133
Case 38: Unexpected Intraoperative "Oozing"	137
Case 39: Central Venous Access and the Obese Patient	141
Case 40: Check Your Facts	145
Case 41: Intraoperative Epidural Catheter Malfunction	147

Case 42: Breathing Difficulties After an ECT	151
Case 43: White "Clumps" in the Blood Sample from an Arterial Line	155
Case 44: Anesthesia for a Surgeon Who Has Previously Lost His Privileges	159
Case 45: Airway Obstruction in an Anesthetized Prone Patient	163
Case 46: A Question You Should Always Ask	165
Case 47: Postoperative Vocal Cord Paralysis	167
Case 48: This Is a Serious Problem	171
Case 49: A Leaking Endotracheal Tube in a Prone Patient	175
Case 50: An Impossible Situation?	177
Case 51: An "Old Trick" but a Potential Serious Problem	181
Case 52: A Loud "Pop" Intraoperatively and Now You Cannot Ventilate	185
Case 53: Postoperative Median Nerve Injury	187
Case 54: A Patient in a Halo.	189
Case 55: It Is Now or Never	193
Case 56: General Anesthesia in a Patient with Daily Use of Prescribed Amphetamine	195
Case 57: What Is Wrong with This Picture?	199
Case 58: The One-Eyed Patient	203
Case 59: A Near Tragedy	205
Case 60: Robot-Assisted Surgery: A Word of Caution	207
Case 61: An Airway Emergency in an Out-of-Hospital Surgical Office	211
Case 62: A Case of Recent Hip Replacement Coming for a Cystoscopy	213
Case 63: A High Glucose Concentration in an Epidural Catheter Aspirate: Should One Be Concerned?	215
Case 64: A General Anesthesia in a Patient Who Has Had a Recent Eye Operation	217
Case 65: Another Awake Craniotomy	219

Case 66: Spinal Fracture and Flail-Segment Rib Fractures Following a Motor Vehicle Accident	223
Case 67: Angioedema in the Emergency Department	225
Case 68: Cranioplasty: Should You Be Concerned?	229
Case 69: More Haste Less Speed	231
Case 70: A Pregnant Patient for a Carpal Tunnel Operation	235
Case 71: A Request to Provide Isoflurane Anesthesia for Treatment of Status Epilepticus	237
Case 72: No Methylene Blue in the Urine: What Would You Do?	239
Case 73: A Right Upper Lobe Tumor and Concurrent Tracheal Polyp: What Lung Isolation Technique Would You Use?	243
Case 74: Complete Heart Block During Central Line Placement	243 245
Case 75: Cervical Hematoma Following Neck Surgery.	243
	247
Case 76: Transient Language Disturbance Following General Anesthesia	251
Case 77: A Flexible Suction Catheter Complication	255
Case 78: A Neurosurgical Case with a Sudden Disappearance of the Arterial Line Waveform	257
Case 79: Not Another Corneal Abrasion	261
Case 80: A Maxillofacial Operation	265
Case 81: A Patient with a Transplanted Heart for Cholecystectomy	269
Case 82: A High Total Spinal in an Obstetric Patient	271
Case 83: Peroral Endoscopic Myotomy (POEM)	273
Case 84: A Neonatal Emergency	277
Case 85: This Could Be Serious	279
Case 86: A Case of Acoustic Neuroma	281
Case 87: Is the IV Infiltrated?	283
Case 88: Communication Is Essential	287
Case 89: Watch Out	289
Case 90: A Simple Case but It Goes On and On	291
Case 91: Endotracheal Intubation in the ICU: Watch Out.	295

Contents

Case 92: A Straightforward Case, or Is It?	297
Case 93: Postoperative Red Urine	299
Case 94: Patient's Toes Suddenly Become White During a Lower Limb Operation	303
Case 95: A Percutaneous Tracheostomy	307
Case 96: A Patient in the Prone Position – Watch Out	311
Case 97: A Patient with Obstructive Sleep Apnea	315
Case 98: A Case of Wegener Granulomatosis	319
Case 99: What Can Possibly Go Wrong?	323
Case 100: Severe Case of Hyperkalemia During Rapid Blood Transfusion	325
Case 101: A Monitor Is Just a Machine	329
Case 102: A Case of Preoperative Sinus Tachycardia	331
Case 103: Bonus Question	333
Index	335

Case 1: No Fiber-Optic Intubation System: A Potential Problem



You are to anesthetize a 19-year-old Indian woman (42 Kg) who is otherwise healthy but is coming for a removal of a large keloid scar $(7 \text{ cm} \times 8 \text{ cm})$ on the front of her neck. This was caused 2 years before by hydrochloric acid (HCl) when she tried to drink it in an attempt to commit suicide. Someone prevented her from doing so, but during the tussle that ensued, the large cup of HCl spilt down the front of her neck causing a severe third-degree burn. She survived but is now left with a large keloid scar that has pulled her chin to nearly touch the sternum, and she can only open her mouth slightly (0.5 cm between the top and bottom teeth). You see this young woman in the preoperative area and decide that an awake nasal or oral fiber-optic intubation is needed. Unfortunately, there is no scope available, and the surgeon tells you that if we do not do it today, the young woman will not come back. You decide to proceed and take her back to the operating room after an IV is started and 1 mg of midazolam. After routine monitors are placed, you attempt an inhalation induction with sevoflurane to be followed by a blind oral or nasal endotracheal intubation. Unfortunately, you lose her airway during the induction and she stops breathing. The saturation falls to 82%. You turn the sevoflurane off and attempt to ventilate with 100% oxygen but with great difficulty. With the sevoflurane off, she slowly commences to breathe again and her saturation improves. Your attempt at an awake nasal intubation also fails. There is no other airway equipment available, for example, a Trachlight (Laerdal Medical A/S, Stavanger, Norway). You suggest to the surgeon that he does a tracheostomy under local anesthesia. The surgeon says that will be impossible as there are no landmarks, and it is very difficult to anesthetize the keloid scar with local anesthesia. But more important is the fact that the tracheostomy will be in the surgical site, and therefore it is not an option. You attempt to place the smallest pediatric LMA that you can find. Unfortunately, even that LMA is too big. In desperation you now try to pass a pediatric gum elastic bougie blindly into her trachea both through the mouth and nose. This also fails. Understandably she is now getting very upset and agitated. The surgeon looks at you and wonders if there is anything else that can be done to secure the airway without doing a tracheostomy. What will you suggest?

Many years ago (1973) in King Edward 8 Hospital, Durban, South Africa, Dr. Derek Ardendorf a plastic surgeon and I were confronted with this problem. An inhalational induction failed, and Dr. Ardendorf chose not to do an elective tracheostomy for the reasons mentioned. So what did we do? I gave the patient the following drugs intravenously: diazepam 5 mg followed by atropine 0.6 mg and ketamine 2 mg/Kg. With the patient asleep but breathing, the surgeon cut away at the keloid making it possible for me to extend her neck and open her mouth. When I could get the laryngoscope in the mouth and saw the epiglottis, I gave succinylcholine 40 mg and secured the airway. Hemostasis was then achieved and the surgery was completed successfully. I kept an eye on future development and she did very well. The last time I heard from her, she had gotten married.

Discussion

To put this problem more in perspective: in those days this case was done without oximetry, capnography, and automated noninvasive blood pressure machine. However, we had ECG machines in most rooms. Furthermore, there were no pediatric gum elastic bougies or LMAs. The LMA was introduced into the United States in about 1990.

Lesson

In difficult cases like these, it is imperative that you and the surgeon agree on a plan and preferably have plans B and C. Furthermore, you must have confidence in your colleague's ability as well as your own.

Case 2: Is the Patient Extubated?



An otherwise healthy 48-year-old man is being ventilated in the ICU following major abdominal surgery. You are called urgently because the ICU nurse informs you that she can hear air escaping from the patient's mouth. She is concerned that the patient may have become extubated. His vital signs are HR 90, BP 140/90, and oxygen saturation 96% on FiO₂ of 100%. You arrive and find him somewhat sedated but agitated. You talk to him but he does not answer back despite trying to do so. The nurse tells you that the patient was previously saturating at 92–94% on 40% FiO₂. The ventilator is alarming. The endotracheal tube (ETT) (#8) is taped at 22 cm. A universal bite block (B&B Medical Technologies, Vista, CA 92083) is seen in his mouth (Fig. 1). The bite block consists of a 5-cm-long hollow plastic tube that has a 0.5-cm-long longitudinal opening. This opening stretches from top to bottom going through the whole length of the bite block. An anchoring device (a plastic strap) is available on the bite block so as to attach it to the ETT. An audible leak is heard. You detach him from the ventilator, and with an Ambu bag, you confirm that he has got bilateral air entry although they are distant. Air/bubbles can be heard/seen coming from his mouth. You decide to blow up the ETT cuff, as there must be a leak due to lack of air in the ETT cuff. However, the cuff on the pilot tubing is already blown up and feels very tight. You push some more air into the pilot tubing. No improvement is seen and you can still hear a leak at the mouth. The ventilator continues to alarm.

What will you do and what is the cause of your dilemma?



Fig. 1 Universal bite block

Since you believe there must be something wrong with the cuff and/or pilot tubing, you exchange the existing ETT with a new ETT using a gum elastic bougie [1]. The cuff on the new ETT is blown up and no more leaks are heard. The patient is sedated and the ventilator now works without alarming. You look at the ETT and the bite block that have been removed in one piece (i.e., they are both anchored together with the plastic strap). You see the cause of the problem. The bite block had migrated down the ETT and clamped off the pilot tubing completely (Fig. 2). If one had discovered the problem, one could have moved the bite block up the ETT, thereby releasing the obstruction in the pilot tubing (Fig. 3).

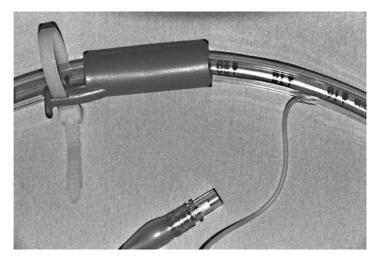


Fig. 2 Bite block seen on ETT

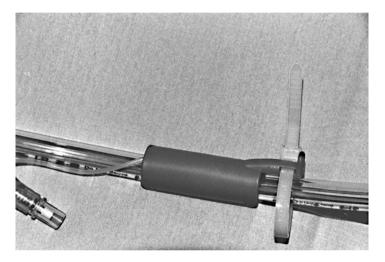


Fig. 3 Bite block shown to clamp off pilot tubing

Discussion

This case is similar to a previously described case [2]. When faced with obstructed pilot tubing, that causes a leak due to the inability to fill the ETT cuff with air, the correct thing to do is to relieve the obstruction if possible. If that cannot be done, then one is forced to replace the ETT. Replacing the ETT may also be considered the safest thing to do, as the original pilot tubing may generate a leak anytime after the obstruction is relieved.

Lesson

Beware of bite blocks as they can cause problems.

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Case 3: A Strange Computerized ECG Interpretation



A 38-year-old man is scheduled for vasectomy reversal. He is healthy except for a history of several anxiety attacks, after a death of his youngest child, 6 months before this procedure. His symptoms included shortness of breath, palpitations, and dizziness. He denies any history of cardiac or neurological disease, syncope, exercise-induced chest symptoms, and any family risk factors for coronary artery disease. He states he is fit and his wife concurred that he exercises regularly. Since the patient was deemed healthy and not scheduled for major surgery, he was not evaluated in the Anesthesia Preoperative Assessment Clinic prior to surgery.

On the day of surgery, a full history and examination is done. The history was as above with one general anesthetic for sterilization which had been uneventful. On examination nothing abnormal was detected. His initial blood pressure was 133/73 mm Hg, and the heart rate was 73 sinus rhythms by palpation. Since the patient is concerned that his anxiety symptoms might actually represent a cardiac disorder and no previous ECG had been done, an ECG is ordered. With the patient lying flat, a 20 G intravenous catheter is inserted in the back of his right hand. The ECG is done at the same time. The ECG is shown below (Fig. 1). The patient is awake and cooperative but feels a bit anxious. His blood pressure is 80/50 mm Hg and heart rate deemed to be regular at 36 beats per min.

With the above information and now the result of the computerized ECG, what would you do? Will you proceed or will you cancel the case for further workup by cardiologist? What will you tell the patient about his ECG?

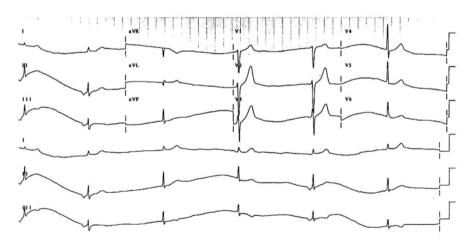


Fig. 1 Abnormal ECG prior to general anesthesia (see text) atrial fibrillation, ventricular rate 35, borderline ST elevation

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A diagnosis of vaso-vagal reaction was made and the patient was treated with atropine 0.5 mg IV. An ECG repeated 5 min later shows sinus rhythm at a rate of 51 bpm (Fig. 2). The patient was evaluated as stable, and he was reassured that this second ECG was normal. He was told that the reason for the repeat ECG was that he had had a fainting episode while the IV was being placed. The faint had caused his heart to slow down and give an abnormal ECG tracing. He underwent an uneventful general anesthetic, and two subsequent ECGs done in the postanesthesia care unit at 2 and 4 h after the initial abnormal tracing showed sinus rhythm with a normal heart rate and a borderline left axis deviation. His PACU and post-discharge course was uneventful.

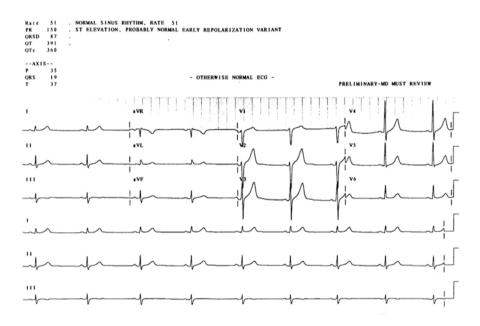


Fig. 2 Normal ECG after vaso-vagal reaction and treatment with atropine 0.5 mg IV

Discussion

A similar case has been reported previously [1]. Computerized ECG programs have been used in clinical practice for nearly 20 years. Surprisingly, there is little published date analyzing their accuracy and effect on clinical practice. A study by Jakobsson et al. [2] showed that 82% of computer interpretations were judged to be adequate, versus 64% of the physician's interpretations. Spodnick [3] found that computer interpretations of ECG's recorded 1 min apart were significantly and grossly different in 36 of 92 (39%) unselected pairs of tracings. Of interest here is that atrial fibrillation

was the computer interpretation in 11 of the 36 pairs for one tracing, but a totally different rhythm was diagnosed in the second identical tracing. Our case presents several limitations of computer ECG interpretation. Firstly, the machine incorrectly diagnosed atrial fibrillation, while in fact Fig. 1 shows two abnormal rhythms present. Broad inverted P waves are seen in two complexes in the rhythm strip from leads II and III (at the bottom of Fig. 1), suggesting an ectopic atrial focus. P waves generated by the sinus node should be upright in all leads except V 1, where they may be biphasic [4]. Figure 2 shows the return of normal P-wave morphology, in which the P waves are upright in all leads. In the remainder of the complexes seen in the rhythm strips from leads II and III (Fig. 1), no P waves are present, which is consistent with junctional rhythm. In essence the computer program misinterpreted the variable rate (due to the alternating presence of two "escape" rhythms) and the absence of P waves in a majority of the complexes as atrial fibrillation.

Should this patient have had an ECG preoperatively a few days prior to surgery? Roizen recommends not obtaining ECG's in asymptomatic men under the age of 40 years [5]. This is based on the very low incidence of significant abnormalities detected on the preoperative ECG in men under the age of 40 who are asymptomatic and who have had a thorough preoperative evaluation [6]. At Stanford, healthy asymptomatic men under the age of 50 do not undergo routine preoperative ECG testing in the Anesthesia Preoperative Assessment Clinic [7]. In retrospect, if a preoperative NORMAL ECG had been found, one would not have ordered a repeat preoperative ECG on the day of surgery. As it was, the abnormal ECG, obtained on the day of surgery, did little to reassure him that he was not suffering from some potentially significant cardiac disease. In this case, the computer interpretation was significantly in error and, if not reviewed, could have led to an unnecessary surgical cancellation with possible hospitalization and added stress to the patient. It is important to realize that ECG programs do not consider other pertinent clinical data, like with this relatively fit, young, anxious man with no history of heart disease and who is having an IV started while the ECG tracing was obtained. Atrial fibrillation, with a very slow ventricular response rate, might be expected in a patient with chronic atrial fibrillation and a very high serum digoxin level or with serious underlying conduction disease.

Lesson

A computerized ECG result must always be reviewed by the physician and interpreted in light of clinical data.

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Case 4: An Elderly Lady with a Fractured Neck of the Femur



An 83-year-old lady (70 kg, 5'5" tall) is admitted to the emergency room after a fall in her nursing home. She has a fractured neck of the femur, but otherwise no trauma. In addition, she has many medical problems, including coronary artery disease, hypertension, and chronic obstructive lung disease. On examination she is cooperative, orientated for time and place. She has mild to moderate bilateral ankle and sacral edema, HR 100 atrial fibrillation, and BP 170/100. The EKG shows an old MI with left axis deviation. Room oxygen saturation is 91%. Her chest is clear except for crepitations at the bases and increased respiratory wheeze. She is orientated for time and place and requests a spinal anesthetic as she is worried about going to sleep. You are happy to oblige and explain that she must either sit up or lie on her side for you to do the spinal. She absolutely refuses to sit up or lie on her side claiming this will be very painful. She has received 10 mg of morphine in the emergency room. You attempt to sit her up but she complains of severe pain. You give her midazolam 0.5 mg and fentanyl 50 micrograms slowly. A little later she says she is feeling better. However, her oxygen saturation has now fallen to 87 % on room air. Supplemental oxygen improves to 93%. When you attempt to sit her up again, she refuses point blank. You consider giving her a small dose usage of ketamine so that you can place the spinal in a lateral position. However, ketamine can produce unacceptable increase in BP, and the prior use of atropine to prevent excessive salivation can produce an unwanted increase in heart rate.

What else could you do to make her pain-free, so that you can perform the spinal block?

A femoral nerve block.

Discussion

This is a relative easy block to perform, if you do it frequently. I normally do this block as soon as the patient has agreed to a spinal anesthetic for a fractured neck of the femur, preferably in the preoperative holding area. By the time the patient arrives in the OR, the block is working. As to the anatomy: the vein, artery, and nerve lie from medial to lateral. The femoral nerves lie behind and lateral to the vascular sheath and, unlike the vessels, are not within it. All three are deep to the fascia lata, but unfortunately the exact position of the femoral nerve in relation to the artery is inconstant. It may be close to the sheath or several centimeters lateral to it. Remember also that it is usually more deeply placed. These factors often make the blocking of the nerve more difficult than anticipated. When the sciatic and femoral nerve blocks are combined, the femoral nerve block is the one that most often fails [1]. However, the advent of the nerve stimulator does make this block easier. When successful, it provides analgesia for the upper part of the femoral shaft, including the neck. The technique consists of drawing a line between the anterior superior iliac spine and the public tubercle [2–4]. This line marks the inguinal ligament. The needle should be inserted just below the ligament and 1cm lateral to the artery. You may feel a "click" as the needle passes through the fascia lata. If this occurs, you should be ascertaining paresthesia or a nerve twitch with the stimulator. If you are 3-4 cm deep in an average-sized person, you are too deep. Start again either laterally or medially. I deposit 15–20 ml of lidocaine 1–1.5% and find this in most cases to be very satisfactorily. If you do not find the nerve, then inject it in a fanwise fashion, from the artery to a point 3 cm laterally to it.

Lesson

In these cases, a successful femoral nerve block can be invaluable, and your patients will thank you.

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Case 5: A Spinal Anesthetic that Wears Off Before Surgery Ends – What to Do?



A 68-year-old woman, previously healthy, is scheduled for a large inguinal hernia repair. She has a class 3 Mallampati score and weighs 230 lbs and is 5 feet 5 inches. She tells you that a previous general anesthetic, not long ago, was complicated by great difficulty in securing the airway with an endotracheal tube. She was advised to tell any future anesthesiologist about this potential problem. She is nervous about a general anesthetic and requests a spinal anesthetic. You place an uneventful spinal at L3-4 with 1.4 ml bupivacaine 0.5%. The spinal works well and the legs are placed in lithotomy position and the surgery begins. Operative problems, mainly with lack of proper equipment, are encountered, and the procedure that should have taken 30 min. is now 2 h and still ongoing. The patient begins to complain of pain. You give some sedation with midazolam up to 2 mg and fentanyl up to 75 mg, while the surgeon injects into the surgical site and around with 50 ml of lidocaine 1%. Neither has much effect, as the patient still complains of pain and looks irritated by the whole proceedings. You consider an awake fiber-optic intubation, followed by a general anesthetic. Unfortunately, you are now told that all fiber-optic intubation equipments are being serviced and would not be back before tomorrow. You dismiss the idea of more sedation and inducing general anesthesia via a face mask as being potentially too dangerous. The surgeon, who is a friend of yours and usually very reliable with his estimated surgical time, tells you that he will be only 10–15 min. You believe him, but what are you to do?

You ask the patient to take 30 ml of oral Bicitra (an antacid) which she does. Metoclopramide 10 mg IV is also given prior to giving atropine 05 mg [1] and IV ketamine 1–2 mg/kg. In these cases ketamine can be a real winner, as respiration is not depressed except in large doses [2]. An adequate surgical anesthesia is established and the surgeon completes his case. Both he and the patient are very grateful.

Discussion

The prevention of aspiration cannot be guaranteed with the use of ketamine, without safeguarding the airway. Where there is a risk of aspiration, an oral antacid and IV metoclopramide should be considered. Both drugs also increase the lower esophageal tone, and the oral antacid acts via increasing the pH of the stomach [3, 4]. A related compound to ketamine is phencyclidine, which was used in anesthesia but withdrawn because of the high incidence of hallucination [5]. It is however still used in animals. An intravenous dose of 2 mg/kg of ketamine will produce anesthesia in 30 s and last for 5–10 min. An intramuscular dose of 10 mg/kg results in anesthesia in 3–4 min, lasting 10–20 min. Remember that you must always use a drug like benzodiazepines and/or droperidol prior to ketamine, in order to reduce the incidence of emergence sequelae [6]. These include postoperative disorientation, confusion, and irrational behavior. Auditory and visual hallucinations are common. Visual hallucinations with terrifying dreams are, in the author's opinion, much more common than auditory problems. When ketamine was first introduced in 1965 by Domino et al. [7], this was a real problem until a remedy suggested by Dundee et al. [8] using an opiate-hyoscine premedication decreased the incidence. However, small amounts of benzodiazepines are more effective. Excess salivation with the use of ketamine can also be a problem, and glycopyrrolate or atropine may be required.

Lesson

Remember ketamine [9] in these cases, as it can be a real "lifesaver." As an aside the drug is also now being used to treat depression [10].

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Case 6: Just a Simple Monitored Anesthesia Care (MAC) Case



It is at the end of a long day in the OR. You are scheduled to do an emergency BROVIAC® catheter placement under MAC. The scheduler promises you that this is your last case of the day. The patient is an 83-year-old female, who has been in the hospital for 3 days for a work-up of her severe aortic stenosis for a possible aortic valve replacement. During the work-up, she has developed acute renal failure, and hence the BROVIAC® catheter placement. The patient is admitted to the OR from the ICU and her consent is signed by her son. Neither he nor any other relative is available prior to surgery. You meet the patient outside your operating room and the surgeon is anxious to get going. You say that you would like to speak to the patient and examine her. The patient is partially orientated for time and place. She says she understands that she needs the BROVIAC® catheter for a "problem with her kidneys." She is a small lady, weighing 59 kg and 5'6" tall. She has edema of her legs and sacrum. Her vital signs are HR 110 regular with a BP of 130/90. Room air oxygen saturation is 91%. The patient is receiving 10 l/min oxygen via a face mask. Chest auscultation reveals decreased air entry at both bases with crepitations and rales all over her chest. She has shallow breathing at a rate of 34. Her neck veins are distended. You diagnose congestive cardiac failure, and the surgeon concurs with your assessment but wishes to proceed. You elect not to give her any sedation or narcotics except for more furosemide 40 mg IV. The patient is placed on the operating table, and you reassure her and place noninvasive monitors. An oxygen mask is placed with a strap on her head and 8 l of oxygen is provided. The surgeon injects 20 ml of lidocaine 1% into the surgical site. The junior intern has several attempts at finding the left subclavian vein. Suddenly there is a major drop in the end-tidal CO₂ from 38 mm Hg to 15 mm Hg, and oxygen saturation falls to 83%. You diagnose a pneumothorax and provide mask ventilation with 100% oxygen. The patient's saturation goes up to 94%, the highest it has been since you took over her care. You are about to intubate the patient's trachea when the circulating nurse shouts: "The patient is a DNR/DNI (do not resuscitate/do not intubate)." You elect not to intubate the patient but to treat her pneumothorax with an emergency chest drain. With the help of the nurse, you assemble the drain in less than a minute [1]. With a functioning chest drain and more furosemide, the patient almost returns to her baseline over a period of 10-20 min. The surgeon aborts the catheter placement, and the patient is taken back to the ICU. Unfortunately, she dies within 2 h of arriving in the ICU without any heroic attempts to save her life as per her DNR/DNI.

Shortly after her demise, you manage to meet the patient's three surviving children, a son who is a physician and two daughters who are malpractice lawyers. You explain what happened and they listen politely. At the end you ask them if they have any questions, and the one daughter says: "Thank you for taking the time to explain all this to us. We are also grateful that she seemed not to have suffered too greatly. However, I have only one question for you."

What do you think the question was?

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The question was:

"Did you or did you not at any stage during your care put a tube in our mother's windpipe?"

I told her: "No."

She said: "Thank you, doctor that will be all...."

Discussion

Do not resuscitate (DNR) and do not intubate (DNI) are orders established by competent patients or an elected healthcare agent to provide a mechanism for withholding specific resuscitative therapies in the event of a cardiopulmonary arrest. Most surgeries performed in patients with DNR/DNI orders are palliative and designed to improve patient comfort or simplify care. Hence many individual practitioners and hospitals have made an exception to this covenant and made it routine to suspend these orders the moment the patient enters the OR [2]. The fact that the majority of these patients are willing to temporarily forego their DNR status in exchange for an anticipated benefit of surgery has been challenged by both bioethicists and patient rights advocates [3–6].

My suggestion to reduce conflict and potential liability is the following:

- 1. Each department of anesthesiology should have a policy regarding DNR orders in the perioperative period [7].
- 2. Do not routinely assume that the DNR/DNI orders are abandoned for surgery without discussing it with the patient or the patient's healthcare agent. For instance, a cancer patient who is a DNR may want the DNR lifted for some sort of palliative surgery, but a patient with severe coronary artery disease (CAD) may want to keep the DNR order in place for some last ditch, risky cardiac surgery. In either case it is imperative to inform everyone what may happen with or without the DNR. The patient or the surrogate must make a decision on how they want the procedure to be done.
- 3. You may express concern about where to draw the line between anesthesia care and resuscitation that may conflict with the DNR order. It is not for you to make that decision. It is the duty of the patient or the healthcare agent to tell you. You must attempt at all costs to reach a consensus on how to handle the surgery. Do not proceed before it is reached.
- 4. It is important to write in the chart, prior to the surgery, something like this: "Discussed the risks and benefits of surgery with and without DNR, including the risk that resuscitation may inadvertently occur. Patient understands there is no guarantee about outcome. Patient/healthcare agent wishes to proceed with DNR IN PLACE or DNR NOT IN PLACE."

References

The reader is referred to the ASA publication entitled: Ethical Guidelines for the Anesthesia care of patients and do not resuscitate orders or other directives that limit treatment [8, 9].

Lesson

In our specialty the patient's autonomy is honored through the process of informed consent, and it is our duty to adhere to what has been mutually agreed upon. Be it the DNR in place or not, while the patient is in the OR.

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Case 7: Smell of Burning in the Operating Room



A 65-year-old man (ASA 2) is undergoing a transurethral resection of the prostate under spinal anesthesia. Adequate regional anesthesia is established and no sedation is given at the patient's request. Nasal oxygen 2 L/min is provided throughout the procedure. The patient's legs, abdomen, and thorax are covered with sterile paper drapes. The surgeon encounters difficulty in passing the urethral scope. He disconnects the fiber-optic illumination system (FIS) and leaves it unprotected over the level of the pubic symphysis on the drapes. The surgeon cannot pass the scope and asks the scrub nurse for another. A few minutes later, the patient tells you, "I seem to smell burning." You cannot smell anything, but after putting your nose near the patient's head, you also smell smoke.

What will you do? What can the problem be?

Tell the surgeon to remove the FIS cable and drapes quickly. Examine the patient for any evidence of burn on his skin.

Discussion

The case is similar to one previously reported [1]. In that case, after it was ascertained that there was smoke present in the room, the FIS was turned off. Smoldering drapes over the pubes symphysis were readily seen where the FIS cable was lying on the drapes. The surgeon removed the FIS cable and the drapes were seen to stop smoking spontaneously. The patient was examined under the drapes and luckily no evidence of burns was found. He had an uneventful recovery.

In another case report, the disconnected fiber-optic cable ignited disposable, non-woven paper surgical drapes that were trapped in an enriched pocket of oxygen. Oxygen had accumulated under the drapes from a faulty inflation connector of a pneumatic tourniquet. The resulting flash fire severely burned the patient's leg [2]. Based on these reports, it is obvious that FIS illumination end should never be disconnected from its scope. This will ensure that the extensive heat generated by the light does not cause fire. Combustion takes place when there is combustible material (in our case, paper drapes), supporting atmosphere (oxygen), and a source of ignition (in our case the FIS).

In a laboratory setting, the FIS can produce a smoldering fire on surgical drapes in as little as 7 s [3]. It is interesting to note that despite the manufacturers' of fiberoptic illumination systems (FIS) warning, a disconnected and therefore an unprotected FIS cable can ignite drapes and other operating room materials.

In another laboratory study, the presence of a Bair Hugger under a surgical drape, when exposed to an unprotected FIS, significantly accelerated the time to first smoke. Paradoxically, the presence of the Bair Hugger prevented damage to the patient's gown. The forced air prevented the inferior layer from coming into contact with the fiber-optic light source, thus protecting the underlying patient gown. In an actual surgical setting, it is likely that the Bair Hugger would offer some protection to the patient's skin, directly below the surgical drape [3].

The possibility of burns is ever present in the operation room and constitutes almost as much a risk to patients as to staff. Burns are essentially dependent on the energy delivered to a certain volume of tissue and the rate at which that energy can be dissipated. Burns are more likely when the blood supply to a body part is poor [4]. Although operating room burns do occur, very few cases have been reported [4, 5].

You are recommended to read a chapter on how to avoid operating room fires in the book by Larson CP Jr. and Jaffe RA entitled *Practical Anesthetic Management* [6].

Lesson

Be aware that an unprotected FIS cable can ignite drapes and other operating room materials. Few surgeons seem to be aware of this complication, and it is therefore up to you to be observant when FIS is being used.

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Case 8: A Diabetic Patient for Inguinal Hernia Repair



A 54-year-old man is admitted for an inguinal hernia repair. He has been a type 1 diabetic for 18 years. Two years ago, he developed end-stage renal disease (ESRD), secondary to his diabetic kidney. He now required daily peritoneal dialysis. The peritoneal fluid used consisted of a glucose-containing dialysis fluid in the daytime and an overnight dialysis fluid using one exchange of 2 l peritoneal fluid containing Icodextrin (7.5% w/v) (Extraneal, Baxter Healthcare, Castlebar, Ireland). Icodextrin is a cornstarch-derived glucose polymer. His insulin regimen consisted of Human Insulatard (Novo Nordisk) before bed and Humalog (lispro) before his main meals.

On the morning (7:00 am) of his surgery, in the preoperative holding area, his capillary blood sugar value was 480 mg/dl. He had not taken any insulin for 12 h and had been NPO since midnight. The blood sugar test was done with an Accu-Chek® active (Roche diagnostic, Mannheim, Germany). There were no ketones in the urine. The patient claimed his blood sugar had, in the past 2 years, been running higher. You speak to the surgeon and decide on monitored anesthesia care, which includes local anesthesia and sedation. You order 12 units of fast-acting insulin to be given IV stat (7:00 am). There is a delay in getting the patient to the operating room, but at 7:40 am you are given the go-ahead to take the patient to the operating room. Much to your surprise, you find the patient somewhat incoherent and sweating. You take blood for blood glucose estimation while you call for 50% glucose. When the 50% glucose ampoule arrives, you do not wait for the blood sugar result but give the patient 50 ml of 50% glucose IV as your clinical diagnosis is clear. The IV glucose has a good effect on the patient as he no longer is incoherent and is feeling well.

You are wondering how only 12 units of the above insulin could have caused the patient to become so severely clinically hypoglycemia. Adding to this is the fact that the nurse now informs you that the Accu-Chek® shows a blood glucose of 320 mg/ dl. This is all very puzzling. You realize something is definitely not right when the venous blood glucose results come back as 2 mmol/l (normal fasting levels, 3.33–6.60 mmol/l). You ask the lab to run the venous blood glucose estimation again. But you get the same result.

Why is there such a discrepancy between the two blood sugar estimations?

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Several reports have alerted physicians to the potential interference of dialysis fluid containing 7.5% Icodextrin, with some glucose reagent systems using a glucose dehydrogenase enzyme with coenzyme pyrroloquinoline (GDH-PQQ) [1–3]. The glucose reagent systems in question are, besides the Accu-Chek®, the ExacTech (MediSense), Advantage (Roche), and Glucotrend (Roche). The test strip devices can overestimate the true capillary blood glucose readings, leading to erroneous diagnosis of hyperglycemia. If the patient is then treated with insulin, severe and potentially life-threatening hypoglycemia can ensue.

In the above case, had there been no delay going to the operating room and the patient been given a general anesthetic (GA) instead of a MAC, there would have been no clinical warning of hypoglycemia during the GA. Hypoglycemia left undiagnosed can lead to serious clinical consequences.

Discussion

It is interesting to note that a full year before the FDA-approved Extraneal in the USA, this problem was highlighted in England [4]. The reason for this interference is the fact that up to 40% of the indwelling Icodextrin is systemically absorbed and then metabolized by alpha-amylase into several oligosaccharides, including maltose, maltotriose, and maltotetraose. The metabolite concentration peaks at 12 h after infusion into the peritoneum but remains in the circulation for 7 days.

Manufacturers of glucose test strips have also reported interference with their test results when the patient has low hematocrit and/or high uric acid levels. These manufacturers include BM Diagnostics, MediSense, Bayer Diagnostics, Roche Diagnostics, LifeScan, and Hypoguard.

Lesson

Before you rely on handheld glucose monitors in a case like this, you must be sure that:

- 1. The specific monitor is compatible with Icodextrin-based peritoneal dialysis fluid [3].
- 2. Glucose test strips can also give abnormal results in the presence of a low hematocrit and or high uric acid levels.

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Case 9: The Case of the "Hidden" IV



A 55-year-old man (70 kg, 5'11", ASA 2E) is admitted for emergency laparotomy following a stab to his abdomen. He is hemodynamically stable. He has no past history of note except a previous stab, again to his abdomen. His previous anesthesia and surgery have been uneventful. He arrives in the operating room with a 20G IV in his hand which is seen to work well. He does not like needles, and since he is considered stable, routine rapid sequence induction is initiated without any problems using the 20G. After induction, a 14G peripheral IV is placed in the right hand and attached to a Hotline® Fluid Warmer (SIMS level 1 INC., Rockland, MA 02370) for possible rapid IV fluids and blood transfusion. A central vascular line is inserted into the right subclavian vein with one stick, and no problems are encountered. The CVP is running well, and you aspirate back the blood easily from all lumens. At the surgeon's request, the left arm with the 20G IV is tucked alongside the patient's body. The right hand with the 14G IV is placed "out" at 90° to his body. The surgery commences and repairs of both small and large intestines are needed. Four hours into the operation, the urine output decreases. The surgeon tells you that he cannot feel very little urine in the bladder. The CVP, as measured with your triple lumen IV set, is within normal limits. Through the 20G IV (the IV is now seen to be working "great"), furosemide 10 mg is given and a dopamine drip is commenced. Thirty to forty minutes later, no improvement in urine output is seen. The patient is still cardiovascular stable with a normal CVP, but the urine output is only 3 ml in the last hour. You give more furosemide and increase your dose of dopamine again via your 20G IV. However, 30 min later, there is still no improvement in urine output.

Before contemplating other drugs/doses/fluids, what should you do?

Check your 20G IV line. In a previously reported case, the 20G IV line was found to be cut 30 cm from where 20G needles were placed in the vein [1]. Interestingly, the distal end, attached to the 20G catheter, had a tight knot, while the proximal end was completely open. It now became obvious why the IV was working "great." We could not ascertain how this happened, as, in our case, all concerned denied any involvement. Somehow the IV was cut and a knot was made either before or after the IV cut. It is of course possible that the tucking of the right arm with a sheet could have made a knot in the IV tubing, and the surgeon later inadvertently cut the IV proximately to the knot.

Discussion

In a previous publication, the problem with the "hidden" IV was highlighted [1]. The term "hidden" IV is used when the surgeon requests one or both arms to be tucked beside the anesthetized patient's body during surgery. If the arm or arms have an IV in them, then the IV is hidden from view.

Serious complications can arise when the IV insertion site is hidden. One of the more serious complications is large infiltration of fluids into the subcutaneous tissues of hand/arm/feet. I have seen a case where a right arm had to be amputated above the elbow following a 5-h cardiac bypass procedure, where liters of fluids and blood were pumped into the subcutaneous tissue of the right forearm.

Lesson

The messages of this case are:

- 1. If you have an IV that you can see is working (in this case the subclavian vein), use it rather than an IV that is hidden.
- 2. Always worry about a "hidden" IV that suddenly starts to work "great" or suddenly does not work at all. Even if you are at risk of annoying the surgeon while checking your IV site, remember you are looking after the patient. The fact that you may delay the surgeon for a minute or two is of no consequence.

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Case 10: Postoperative Painful Eye



A 50-year-old female (ASA 1) is scheduled for laparoscopic cholecystectomy. Her past history is noncontributory. She takes no medication and has no allergies. The patient is otherwise healthy and has no complaints. She has removed her glasses and states that she does not use any contact lenses. You are joined by a medical student who put all the monitors on the patient including the oxygen saturation monitor on her right index finger. The patient is induced and the medical student ventilates the patient by mask. He places an endotracheal tube in the trachea successfully. The anesthetic proceeds uneventfully, and she wakes up pain-free and is taken to the recovery room in a stable condition.

About 1 h later, the patient is still in the recovery room. You are called by the recovery room nurse to tell you that the patient is complaining of a painful eye.

What will you do and what could the cause be?

You call for an ophthalmology consult. Corneal abrasion is suspected and the diagnosis confirmed by fluorescein staining. The treatment consists of eye ointment and taping the eye closed until resolution of the injury. The patient made an uneventful recovery.

Discussion

There are many causes of postoperative corneal abrasions. These have been included in anesthesia face masks, anesthesiologist hands, watch strap, name badge and laryngoscopy during endotracheal intubation, surgical drapes and instruments, skin preparation solutions, or the direct irritant effect of inhalation anesthetic agents [1–3]. In the postoperative period, the eye may be injured by face masks, the patient's finger, or blankets or linen [1–3]. The latter is especially true if the patient is in a lateral position [1].

Instead of taping the lids, Orr and Park have suggested another eye protection option [4]. After the patient is asleep, a surgical mask is used. Instead of placing the mask and its malleable metal nasal contour strip over the nose, the mask is inverted. The contour strip becomes the inferior border allowing coverage of the both eyes.

In the case presented here, the most likely cause of the corneal abrasion was the disposable pulse oximeter probe getting into contact with the patient's cornea. This is because all other causes could be excluded [5] and the recovery nurse saw the patient rub her eye with the right index finger. This was the finger the oximeter probe was attached to.

Lesson

It may be that the ring finger is a more appropriate site for the pulse oximeter rather than the index finger. This avoids possible corneal abrasion, when the patient rubs his/her eye in the immediate postoperative period [5, 6].

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Case 11: Awake Craniotomy



A 43-year-old (ASA 2), 120 kg man is being given local anesthesia with monitored anesthesia care (MAC) for an awake craniotomy. His past history included previous surgery under general anesthesia for resection of an astrocytoma. He presented with severe seizure disorder, mainly due to further growth of the cerebral tumor. He is very nervous about having this procedure awake. Initially, remifentanil and propofol infusion with local anesthesia provided adequate operating conditions. All sedation is turned off during the language mapping, since the patient needs to be fully alert and cooperative. The patient is now only minimally sedated. Since his face is covered with drapes, he complains of severe claustrophobia and serious lack of air. His SpO₂ is 100%. Despite increasing the nasal oxygen to 10 l min and providing 15 L min of oxygen around his face from the absorber circuit of the anesthesia machine, no symptomatic relief is seen. Further sedation is not indicated since he needs to follow commands during the language mapping. His complaints are now becoming so serious that he wants the mapping and surgery stopped.

Besides inducing general anesthesia, is there anything else that you could recommend?

In a previous case [1], we blew forced cold air (13.5C), from a Polar Air Model 600 (Augustine Medical Corporation, Eden Prairie, MN 55344), over the patient's face. This gave immediate relief from the feelings of lack of air and claustrophobia. The language mapping was completed successfully and he made an uneventful recovery.

Discussion

Awake craniotomy without language mapping is relatively easy [2]. With language mapping it can be very challenging at best. Communication with the patient is of utmost importance during the procedure, so that the integrity of the speech center is maintained. The patient must understand prior to surgery that he/she will have to lie still for hours in one position. Propofol and remifentanil infusion has made this procedure well tolerated by most patients, but when it is turned off, problems can arise [3].

Lack of air underneath the drapes can be very disturbing to the patient. We have several times used the Polar Air technique with good effect when the patient complains of claustrophobia and lack of air.

Lesson

In awake craniotomy, blowing cold air over the patient's face can often help to bring about a successful language mapping procedure.

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- 2. Guenbaum SE, Meng L, Bilotta F. Recent trends in the anesthetic management of craniotomy for supratentorial tumor resection. Curr Opin Anaesthesiol. 2016;29(5):552–7.
- McDougall RJ, Rosenfeld JV, Wrennall JA, Harvey AS. Awake craniotomy in an adolescent. Anaesth Intensive Care. 2001;29:423–5.

Case 12: Gum Elastic Bougie



Today you are working with a medical student in what would seem to be a straightforward surgical list. The first patient is a 40-year-old female for a laparoscopic cholecystectomy. On examination, she has a class 2 airway, weighs 80 kg, and is 5'6". She has no other medical or surgical problems. The medical student places the IV and you induce anesthesia. The medical student is masking the patient with ease. You tell the medical student to get the laryngoscope out. He places the scope correctly but tells you that he cannot see anything. You look and have to agree with him. You decide the patient is a class 3. You take out your gum elastic bougie (GEB) and place it blindly in what you think is the trachea. You pass a #7 endotracheal tube (ETT) over the GEB but have difficultly advancing the ETT. You turn the ETT 90–160 degrees to the right, and the ETT glides into what you presume is the trachea.

As you know, it is impossible to verify correct positioning without removing the GEB from the ETT. The medical student asks you, as you are about to remove the GEM: "Is there any way you could ascertain that you are in the trachea without removing the GEM from the ETT?" You remove the GEM and proceed to verify that the ETT is in the right place. You ponder the question and wonder if there is a way to do it.

Is there?

The bronchoscopic swivel adaptor (PriMedico, Largo, FL) can be used to help confirm the correct placement of ETT without prior removal of the GEM (Fig. 1). This can be done by sliding the bronchoscopic swivel adaptor over the GEM via the bronchoscopic port. The bronchoscopic swivel adaptor is attached to the ETT, and correct placement can be confirmed by auscultation and capnography. The GEM can then be removed [1].

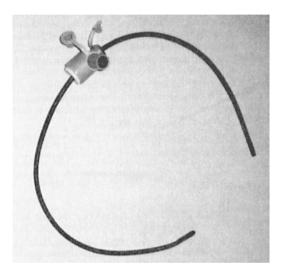


Fig. 1 The bronchoscopic swivel adaptor attached to the GEB (From: Torralva et al. [1]. Reprinted with permission from Wolters Kluwer Health, Inc.)

Discussion

This technique can prove useful when using the GEM as a tube changer, allowing ventilation during the changeover, thereby minimizing the risk of hypoxia during this procedure [2].

The GEM was first described by Robert Macintosh in 1949 [3]. Since then it has proved its worth as a reliable and easy way to gain access to the difficult airway. However, like so many things in life and anesthesia, there is a little trick, when using the GEM, which one must know. Firstly, under no circumstance should one force the GEM into the trachea or any other structures. Secondly, if you encounter problems with passing the ETT over the GEM, then turn the ETT either left or right 90–180 degrees before attempting to advance it. Never force the advancement of the ETT. If you feel you are in the trachea and despite the above maneuver you still do not succeed in advancing the ETT, then you should try a #6 ETT. This is the smallest ETT that an adult GEM will take. Obviously it is better to have a small tube than no tube at all.

Lesson

The bronchoscopic swivel adaptor can be used to confirm the correct placement of the ETT without removing the GEM.

- 1. Torralva PR, Macario A, Brock-Utne JG. Another use of a Bronchoscopic Swivel Adapter. Anesth Analg. 1999;88:1187–8.
- 2. Robles B, Hester J, Brock-Utne JG. Remember the gum-elastic bougie at extubation. J Clin Anesth. 1993;5:329–31.
- 3. Macintosh RR. An aid to oral intubation. Br Med J. 1949;1:28.

Case 13: You Smell Anesthesia Vapor – Where Is It Coming From?



A 45-year-old patient, American Society of Anesthesiologist (ASA) physical status 2, is to undergo a removal of a cerebral tumor under general anesthesia. An anesthesia machine (Drager Fabius GS, Telford, PA 18969) and breathing system check are performed before the patient's arrival. The Drager Vapor 2000 (Drager Medical AG, Lubeck, Germany) sevoflurane vaporizer is seen to be full. Noninvasive monitors are placed, and after preoxygenation the patient is anesthetized in a routine manner. Invasive monitors (arterial line and central venous line) are placed, and the operating table is turned 180° from the anesthesia machine. The operation proceeds uneventfully. Two hours into the case, you suddenly smell anesthesia vapor around the anesthesia machine. You think you can smell more vapor around the vaporizer, but you are not sure. All vital signs are within normal limits. There are no warnings to indicate low minute volumes, apnea, or no ventilation of the patient. The rotameters show adequate flow and the pipeline pressure is 50 psi. The expired tidal volume is 600 ml, peak pressure is 25 cm, and the respiratory rate is 8. End-tidal CO2 and sevoflurane concentrations are within normal limits. (Datex Capnomac Ultima, Helsinki, Finland).

What will you do? Will you ignore this finding or get another machine? Is there any way of identifying more precisely where the leak originates?

The best thing to do is to disconnect the end-tidal CO_2 anesthetic agent sampling tube from the Datex Capnomac machine and use the sampling tube to "sniff" around the vaporizer and anesthesia machine. In this case, the cause of the smell was a leaking vaporizer.

Discussion

Previously we have reported four different vaporizers of the Drager Vapor 2000 variety leaking vapor when in use [1]. We believe that the problem with the above vaporizers is that they have been damaged during refilling. When filling these vaporizers, the lever does not need to be flush with the front of the vaporizer. The lever should only be pushed to achieve slight resistance. Closing the lever all the way damages the filling port and thereby causes a leak.

The "sniff" method was successful in identifying the leaks in all the above vaporizers. We did not change the vaporizer until the end of the case. We were advised by our bioengineering technicians that this could not be done during the surgical operation. Since the amount of vapor that was leaking was minimal, we were not concerned. A bigger leak would have necessitated a machine change or the use of another vaporizer on the existing machine.

The "sniff method" may also be used to identify small leaks in the anesthetic circuit, for example, a pinhole in the tubing from the ventilator to the soda lime absorber.

Lesson

When you smell vapor around your anesthesia workstation or if you have a small leak in your circuit, do not forget the "sniff method."

Reference

1. Bolton P, Brock-Utne JG, Zumaran AA, Cummings J, Armstrong D. A simple method to identify an external vaporizer leak (The "Sniff" method). Anesth Analg. 2005;101:606–7.

Case 14: Manual Ventilation of a Patient Turned 180 Degrees Away from the Anesthesia Machine by a Single Operator – Is it Possible?



You are anesthetizing a healthy ASA 1 young man for nasal reconstruction. He is taken to the operating room and routine monitors are placed. He is anesthetized and the airway secured uneventfully. He is disconnected from the ventilator and the table turned 180°. The medical student who is working with you asks: "Why don't you turn the patient 180 degrees and then put the patient asleep?" You explain that your arms are not long enough to hold both the patient's face mask and the reservoir bag on the anesthetic machine. He sees your point. However, you wonder if there is a way to do this relatively safely with the existing equipment that you have available to you in the operating room.

Do you know a way?

Omar slave [1] (Fig. 1) makes it possible for one operator to stand at the head of the table, 180° away from the anesthesia machine, and control both the airway and manually ventilate the lungs. The modification consists of using a Portex straight gas sampling connection with a Luer port and cap (Sims Portex, Fort Meyers. FI.33905) and place it where the reservoir bag is situated on the anesthesia machine (North American Drager, Telford, PN 18969). This sampling connection is then connected to 8–12 feet (22 mm diameter) anesthetic tubing. The other end of the anesthetic tubing is extended to the head of the operating table and fitted with a valve with a bleed vent from a Baby Safe Resuscitator (Vital Signs, Tolowa, NJ 07512) (Figs. 1 and 2).



Fig. 1 With the patient turned 180° from the anesthesia machine, the anesthesiologist can ventilate the patient's lungs



Fig. 2 The one end of the anesthetic tubing (not shown) is connected to the anesthesia machine where the reservoir bag is situated. The other end of the anesthetic tubing (shown in this figure) is connected to a Portex straight gas sampling connection. This again is connected to the valve assembly from Baby Safe Resuscitator including the reservoir bag (Vital Signs, Totowa, NJ 07512). The whole length of the anesthetic tubing is 8–12 feet long

Discussion

We undertook a study [1] which showed that Omar slave could function as intended. This technique is to be used only by experienced anesthesiologists who have a surgeon and a nurse in the room. This in case anything untoward should happen. Furthermore, Omar slave must be checked for leaks, and the patients must have a class 1 or 2 airway and be of normal weight and height. I have personally used this in healthy patients on numerous occasions without any problems. It is called Omar's slave. I call it that since Dr. Omar (from Cairo, Egypt) was one of the best clinical anesthesiologist I have ever had the pleasure to work with. He taught me this technique while I was under his guidance in Oslo, Norway in 1970.

Lesson

The Omar slave can be used by a single operator for manual ventilation of a patient turned 180 degrees from the anesthetic machine while maintaining minute ventilation and normal ETCO2.

Reference

 Chu LF, Harrison K, Brock-Utne JG. Manual ventilation of a patient turned 180 degrees away from the anesthesia machine by a single operator. International Research Society Annual meeting. New Orleans, LA. March 2003:21–23 (abstract).

Case 15: Life-Threatening Arrhythmia in a 5-Month-Old



A 5-month-old child was scheduled for elective correction of transposition of the great arteries (Senning procedure). She was diagnosed with the transposition immediately after birth, and a balloon septostomy was performed with good result. Prior to this proposed operation, the child was in good physical condition and weighed 5.9Kg. She tolerated the operation well and came off cardiopulmonary bypass uneventfully. She was paralyzed, ventilated, and sedated postoperatively. Her cardiovascular system was supported with small continuous doses of dopamine, phentolamine, and epinephrine with good effect. However, a persistent tachycardia ranging from 140 to 230 beats per minute led to a decrease in mean arterial pressure and contributed to renal insufficiency with a rising creatinine. Peritoneal dialysis was initiated successfully. However, the tachyarrhythmia did not improve (Fig. 1). Digoxin (Lanoxin) 0.16, 0.08, and 0.08 mg was given according to recommendation for children aged 2 weeks to 2 years. (0.04-0.06 mg/kg was given IV during the next hours). Unfortunately, no improvement was seen, and episodes of tachycardia up to 360 beats/min were observed even after the child was fully digitalized. Verapamil, propranolol, lidocaine, and phenytoin were given with no effect on this life-threatening arrhythmia. The patient was now completely anuric. An arterial blood gas showed the pH 7.33, pCO₂ 33 mmHg, BE - 7.7 mmol/l, and pO₂ is 92 mmHg on FiO₂ 0.4. The child had been in the ICU for over 24 h, and of great concern now was the fact that the serum potassium had risen to 5.9 mmol/l. The standard recommended treatment of high potassium, including normalizing the pH and giving glucose-insulin infusion, was started. The peritoneal dialysis fluid with potassium-free solutions was now used. Despite all this, a repeat potassium showed a further increase to 7.8 mmol/l (Fig. 2). Serum calcium and magnesium were within normal limits. The child was now critical; as besides the increased serum potassium and anuria, her cardiovascular system showed a heart rate of 300 beats per min and a mean arterial pressure of 48 mm Hg.

Please study Figs. 1 and 2 below. What was going wrong? Was there anything that could be done? If so, what?

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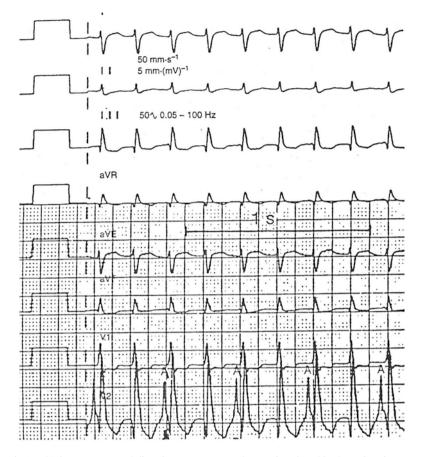


Fig. 1 Lead V2 was connected directly to a temporary heart wire placed in the atrium intraoperatively and showed an arterial rate of 150 beats/min while the ventricular rate was 300 beats/min. The narrow QRS complexes indicate that this was an automatic accelerated AV junctional tachycardia (From Husby et al. [1]. Reprinted with permission from John Wiley and Sons)

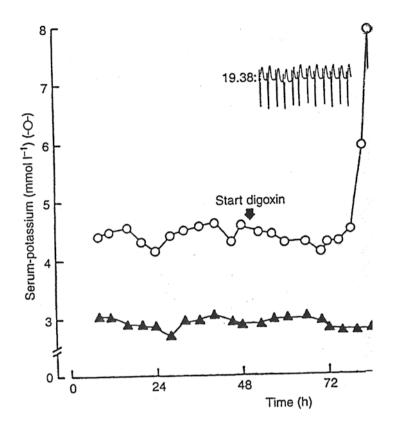


Fig. 2 Serum potassium levels (upper trace) and arterial pH (lower trace) in the recovery period over time. Note the rapid increase in serum potassium (From Husby et al. [1]. Reprinted with permission from John Wiley and Sons)

This case is akin to a previously described case [1]. The cause of the problem was digoxin intoxication, as the digoxin values came back at 8.1 nmol/l (therapeutic range 1.2–2.5 nmol/l). When this information became available, Fab (Digibind) was given IV in a recommended dose of 14.2 mg over a 60 s period. Within a minute after the administration of Fab, the tachyarrhythmia converted to a regular sinus rhythm at a rate of 140 beats/min (Fig. 3). The mean arterial pressure increased from 48 to 65 mmHg. The need for inotropic as well as ventilatory support steadily decreased over a 24-h period. It is interesting to note that serum potassium normalized within 20 min after Fab was given (Fig. 3). No adverse reactions were seen during and after the administration of Fab. The child was discharged from the ICU 3 days later and was doing very well.

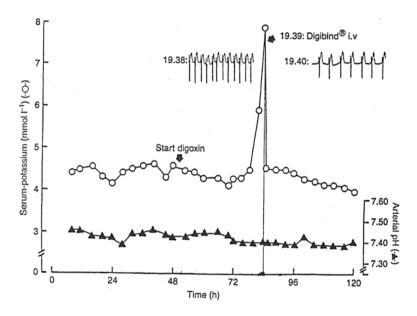


Fig. 3 Serum potassium and arterial pH in the recovery period. Note the increase in serum potassium during digoxin toxicity and the very rapid conversion of the cardiac arrhythmia (ECG strips inserted) as well as the rapid normalization of serum potassium (within 20 min) following the administration of Fab. Within 1 min, after the Fab, the patient's heart had converted to sinus rhythm (From Husby et al. [1]. Reprinted with permission from John Wiley and Sons)

Discussion

Recognition and treatment of digitalis toxicity is at times difficult. Mortality from digitalis overdoses varies, depending on numerous patient-specific factors as well as the extent of toxicity [2]. The cause of death in digitalis intoxication is mainly due to fatal arrhythmias [3].

The patient described in [1] showed an automatic accelerated AV junctional tachycardia, which is typically seen in acute digoxin overdose. Digoxin can produce many types of arrhythmias. They are mainly due to depression or blockade of conduction and/or enhanced impulse formation. Enhanced impulse formation may appear in the form of atrial, junctional, or ventricular arrhythmias. Disturbances of conduction can occur in the sinus and AV nodes. Sinus bradycardia, sinus arrest, or sinoatrial block is common and is due to the interference of conduction in the sinus node. Depression of the AV node can present as second- or third-degree AV block. Early toxicity may present with prolongation of the PR interval. In a healthy heart, an acute overdose of digitalis leads to AV conduction disturbance with very few incidences of ventricular arrhythmias or ectopy [4, 5]. In contrast, the diseased heart frequently responds to an overdose with a lot of ectopic impulses from most commonly a ventricular focus or foci [5].

Hyperkalemia and refractory hypotension are common symptoms of digitalis toxicity but are stated to be uncommon in children [2]. In the case described, hypotension proved very difficult to treat with conventional inotropic support.

Hypoxemia, acidosis, hypokalemia, hypercalcemia, and hypomagnesemia may increase the irritability of the heart, resulting in arrhythmias. In our case, neither hypoxia, severe acidosis, hypokalemia, hypercalcemia, nor hypomagnesemia was present during and after the administration of digoxin. Renal insufficiency can lead to hyperkalemia, however. In our case, the patient was receiving continuous peritoneal dialysis and laboratory values demonstrated stable values until the actual incident.

In the case described, there was a rapid increase in serum potassium approximately 28 h after full digitalization. This has been previously described [2, 6]. An increase in serum potassium in digitalis overdose has been suggested to be a prognostic sign to a poor outcome [7]. In that report [7], the authors reported a mortality rate of 35% in patients who were treated conventionally for serum potassium greater than 5 mmol/l. Fab has been shown to resolve ventricular and supraventricular ectopy within 24 h and often within 4 h in pediatric patients with digitalis intoxication [2]. In our case, the rapid reversal of both cardiac and extra-cardiac manifestations of digoxin toxicity was unexpected, but nevertheless lifesaving.

Although digitalis toxicity is commonly seen, it can easily be mistaken because of its many different manifestations. It produces toxidrome characterized by gastrointestinal, neurologic, electrolyte, and nonspecific cardiac changes. The drug has been around for more than 200 years but still to this day can cause confusion as to the diagnosis which again leads to delay in appropriate treatment [8, 9].

Lesson

Digoxin overdose can occur in infants, especially when renal impairment is present. This is despite reports that children seem to be less sensitive to the digoxin [3, 10]. Specific treatment with Fab should be considered, certainly when a refractory hyperkalemia occurs.

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Case 16: Tongue Ring



A 22-year-old female (ASA 1) is scheduled for an elective diagnostic laparoscopy under general anesthesia. Her past history is unremarkable. She takes no medication and she has no history of allergy. On the day of surgery, the patient's tongue is found to be pierced with a silver dumbbell-shaped piece of jewelry. This was not found by the preoperative clinic staff nor was it mentioned in the surgeon's note. The jewelry is seen in the anterior one third of her tongue and is sticking out of the tongue by 1 cm. You request that she remove the tongue ring, but she is reluctant to do so as it has been put in only 3 weeks ago. You warn her of the potential dangers, but she refuses to remove the tongue ring. You attempt to find a colleague that would be willing to do the case with the ring in situ. However, all your colleagues are either busy or reluctant to do the case. This leaves you with two options:

- A. Tell her that you would be happy to do the anesthetic if she removes the ring prior to surgery. If she refuses to remove the ring, you will cancel the case.
- B. Do the case with the ring in situ.

What will you do, A or B?

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A: Just say no [1].
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Refuse to do the case with the ring in situ [2–4]. Despite reports that patients have been anesthetized successfully with jewelry in situ, I would recommend that she take the ring out.

Discussion

There are two major concerns with jewelry worn by the patient in the operating room. One is burns: rings or needles [5]; metals [6] can be a source of "alternate-site burn." This is especially true for the older models of ground-referenced generators. Newer electrosurgical generators are designed to avoid alternate burn sites. However, in the instruction for use of these newer generators, it states clearly, "Patient safety is the highest concern, and one is not well served when jewelry is present in the patient undergoing surgery when electrosurgical generators are used." The insert goes on to say that if the jewelry is not removed, the RISK ASSOCIATED WITH THE PRESENCE OF JEWELRY MUST BE ASSUMED BY THE PATIENT AND THE HOSPITAL. As Rosenberg et al. [1] state: "If the companies that produce the equipment are against wearing jewelry and are willing to place the responsibility on us, why should we condone wearing rings in the operating room?" I agree.

The other concern: if a tongue ring is left in situ for the operation, the ring may cause pressure necrosis to the tongue. The ring may also become dislodged and fall into the trachea.

Mandabach et al. [2] have argued that one should evaluate the decision to cancel or not to cancel these cases on a case-by-case basis. He suggests the following precautions. First, jewelry is removed if possible. Second, the dispersive plate is placed as far away as possible from the surgical field. Third, electrocautery is not used if the jewelry is close to the site of surgery. Fourth, the surgeon should use a bipolar electrosurgical unit. This utilizes less power, since the current passes only between the tips of the unit unlike the monopolar unit, where the current passes from the tip throughout the body on its way to the dispersive pad [7, 8]. Fifth, use the newer electrosurgical units with isolated generators that limit the risks of alternate burning sites. The word here is LIMIT. I leave it to the reader to decide but I would just say NO.

Lesson

When faced with this sort of problem, I believe that you must be firm and say no for all the reasons mentioned and not least for the potential medical legal ramifications. To leave the ring in could cause harm and you do not want to do that.

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Case 17: Hasty C-Arm Positioning – A Recipe for Disaster



You have anesthetized an otherwise healthy man for a neurosurgical procedure. He is 54 years old, weighs 110 kg, and is 6 feet in height. He is placed in a left lateral position. The surgeon is in a bit of a rush, and before the patient is appropriately secured, the surgeon moves the C-arm into position over the operating room (OR) table (Skyton 3100, John Cudia & Ass. Morgan Hill, CA 95037). Suddenly the table is now going into a full left lateral tilt without you or anybody touching the table control situated at the head of the table. You grab the patient's head and the endotracheal tube and call for help. The surgeon and nurses, with outstretched hands, prevent the patient from hitting the floor. They call for a gurney as the table is still moving toward a full left lateral tilt.

What will you do?

This case is akin to one previously described [1]. In that case we kept holding the patient as we described on the previous page. What saved the day was the unplugging of the OR table from its electrical outlet. The table stops abruptly with a tilt of $30-40^{\circ}$ to the floor. His gurney was quickly brought back into the room, and the anesthetized patient was safely transferred to the gurney. With the patient safe, we searched for a possible cause and found that the bottom end of the C-arm (i.e., the part of the C-arm that is closest to the floor) was wedged onto the floor control of the Skyton OR table. The C-arm was removed and OR table was again plugged into the electrical outlet. The OR table control was now able to function correctly, and the patient was placed back on the operating table. The surgery was completed uneventfully.

Discussion

It is important to remember that the patient must be secured as soon as the positioning on the operating table is complete. Regarding the patient described above, he would most likely have fallen on the floor despite having been secured. This is because there was and is usually only one strap (over the thighs) that keeps the patient on the OR table. In all cases where strange and bizarre things happen in the OR, the patient's safety and welfare is of paramount importance. I remember having a 140 kg, 5 feet 4 inches' lady anesthetized in lithotomy position for a gynecological surgical procedure. When the surgeon ordered more Trendelenburg, the patient started to slide head first of the table. There was no time to get the OR table control to reverse the Trendelenburg, as she was gathering speed. I tried to hold her but was unsuccessful. She kept gliding off the table, head first. The surgeons did not notice because at the time they were looking at the equipment tray with their backs toward the operating field. The circulating nurse had noticed my dilemma and asked if she could help. I told her to get another anesthesiologist but the patient kept sliding. I then grabbed the head with one arm and the endotracheal tube with the other hand. The patient kept coming, and to prevent her head from hitting the floor, I sat down on the floor and her head landed in my lap. It was at that point when the surgeons returned their eyes to the operating field, and one could clearly hear one saying: "Where did the patient go?" Eventually help arrive, the Trendelenburg was reversed, the patient repositioned on the OR table, and the surgery completed. The cause in this latter mishap was that the patient's small fat legs were inadequately secured into the stirrups. Everything went well, except that unbeknown to everyone a lap was left behind in her peritoneal cavity. Another operation had to be performed to remove it. The patient was reportedly happy as she thought she had malignant tumor in her abdomen after the first operation and was ecstatic when she was told it was only a lap that had been left behind from the previous surgery. It is important to realize that in cases where the legs cannot be secured adequately in the stirrups, Trendelenburg must be limited.

In another case, an anesthesiologist was busy securing the airway in an anesthetized and paralyzed patient, when the C-arm over his head was suddenly and unintentionally activated. The C-arm forced the anesthesiologist head downward toward the patient's head. Luckily someone unplugged the electrical outlet to the C-arm, and the anesthesiologist managed to place the endotracheal tube safely in the trachea [2].

Accidental activation of control knobs on an x-ray table has also been reported [3]. In this case an anesthesiologist was trapped between the wall and the x-ray table, thereby being unable to move and physically being near the anesthetized patient under his care.

Lesson

- 1. Beware of hasty surgeons working a C-arm before the patient is secured onto the OR Table.
- 2. Make sure the OR table floor control is far away from the bottom part of the C- arm.

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Case 18: Inability to Remove a Nasogastric Tube



A 4-week-old, ASA 1, boy is scheduled for a pyloromyotomy. Prior to anesthesia his nasogastric tube (NGT) is removed. The anesthesia induction and maintenance is uneventful. A new NGT is easily inserted during surgery (Argyle feeding tube, Sherwood Medical, size Ch 8, external diameter 2.7 mm × 107 cm). Its correct position is verified by air insufflations through the NTG and observing a slight dilation of the stomach [1]. The patient is taken to the pediatric ICU for recovery. Several hours later, the nurse attempts to manipulate the NGT since it seems to be occluded. While she is attempting to move the NGT, she is surprised to see a loop of the NGT suddenly appear in the mouth. She pushes the NGT in again but after that she cannot move the NGT up or down. You are called and confirm that the NGT is stuck. Using your index finger you examine the baby's mouth but have difficulty ascertaining what is going on.

What will you do?

You call for fluoroscopy and find that the NGT has formed a knot with a large loop at the level of the oropharynx. You cut the NGT as it emerges from the nostril. Under brief sedation with propofol, you retrieve the NGT orally with a Magill forceps using your laryngoscope under direct visual control. You then reinsert a new NGT under fluoroscopic control.

Discussion

This case is similar to one already reported [2]. In that case it was hypothesized that the NGT was initially inserted too far. This allowed) the NGT to form a loop in the oropharynx. It is possible that with the nurse manipulating the NGT, a knot with a large loop was formed. This loop prevented the NGT from being removed via the nose. If blind traction had been used to remove the NGT, the baby would have suffered severe damage to its palate etc. [3–10].

Lesson

If a NGT cannot be removed easily, an x-ray should be done. Always check the markings on the inserted NGT (if present) to make sure it is not inserted too far.

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Case 19: An Unusual Cause of Difficult Tracheal Intubation



A 45-year-old, 80 kg Sikh man from India is admitted for repair of scapholunate dislocation. His past medical history and physical exam is unremarkable. He is classified as an ASA 1. He has got a full beard and speaks English very well. He requests a regional block, but unfortunately it proves to be inadequate for the surgery. General anesthesia is decided upon. Since it is not possible to accurate define the size of his thyroid-mental distance, a rapid sequence induction and intubation is decided upon. After preoxygenation, general anesthesia is induced with intravenous propofol 200 mg followed by succinylcholine 100 mg. Ventilation is easily accomplished by mask. At laryngoscopy the patient's jaw is found not to be relaxed. Trismus is considered. The nerve stimulator shows loss of twitch. A Macintosh #3 laryngoscope blade is passed into the pharynx with great difficulty due to the very restricted mouth opening. Only the epiglottis is seen, but you manage to successfully place a #7 endotracheal tube in the trachea.

He does not have a wired jaw nor does he have bilateral temporal-mandibular joint disease. Why can he not open his mouth?

Further examination of his airway reveals the cause of the problem. His religious belief prevents him from cutting his hair. The excess hair has been put up in a bun on top of his head. From this bun, a long strand of hair is woven tightly together into a cord (0.5–1 cm wide) that goes under his jaw. This cord is severely restricting the mouth opening (Fig. 1).



Fig. 1 A bearded Sikh with a cord that severely restricts the mouth opening (From Brodsky et al. [1]. Reprinted with permission from Wolters Kluwer Health, Inc.)

Discussion

The above case is similar to two previous ones which describe this potential problem [1-3]. In our case [1] (published in 1991) our cursory preoperative examination did not reveal any airway abnormalities caused by disease or trauma to the neck and/ or the jaw. Had we asked him to open his mouth, we would have discovered the patient's inability to do so. In another communication we recommend that all bearded Sikhs should be examined preoperatively for beard restrainers [4]. We also recommend that the patient be informed that the cord may have to be loosened or even cut [4]. However, Bhogal [5] disagrees strongly with cutting the cord as this is considered a serious religious sin. The interesting thing about this cord is that it can be made of any material. It can be elastic and therefore may have some give in it. The position of the bun can be placed anywhere on the top of the head. It can be nearer the forehead or nearer the occiput. The cord, which is attached to bun, can therefore be anywhere. If you know a Sikh is using the cord to restrain his beard, then you can ask him to manipulate the bun to make the cord looser or better still not to use the cord on the day of surgery. There is no religious objection to the latter.

Lesson

A bearded Sikh will not object to not wearing the cord on the day of surgery. If he can't take it off, then consider moving the bun forward.

All efforts must be made to control the airway without cutting the cord. The latter is considered a serious sin.

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Case 20: Pulmonary Edema Following Abdominal Laparoscopy



A 69-year-old, 50 kg woman with pelvic pain is admitted for laparoscopic carbon dioxide laser lysis of pelvic adhesions. Her past history is significant for hypertension and hyperlipidemia. The clinical exam is unremarkable, and although there are no cardiac symptoms, a preoperative ECG shows a left anterior fascicular block. Her medication includes hydrochlorothiazide, triamterene, and gemfibrozil. She is not allergic to any medication. She is sedated with midazolam 2 mg IV and taken to the operating room. Thereafter monitors are placed, and she has a successful induction of general endotracheal anesthesia. She is maintained with desflurane/fentanyl/oxygen/air mixture. Vecuronium is used for muscle relaxation. During the operation the patient receives a total of 2100 ml of crystalloid fluid IV over the 185 min procedure (1000 mL Lactate Ringer's solution before the procedure started, 1000 mL 0.9% saline during the first 90 min and 100 ml 0.9% saline during the final 95 min). The surgeon infuses a lactated Ringer's solution through the laparoscope to wash away blood and debris and thereby improve visualization.

At the end of the procedure, the nurse tells you that a total amount of 4400 ml was instilled, and a similar amount was recovered by the surgeon while sucking out the peritoneal cavity. Her urinary output is 300 ml. The neuromuscular blockade is reversed with glycopyrrolate and neostigmine. Spontaneous breathing ensues. The patient responds to verbal command and maintains a 10 s head lift. The endotracheal tube is removed, but, while still in the operating room, she becomes less alert and seems to have difficulty with breathing. You give her 100% oxygen per mask; the saturation improves to 93% from 84%. Your nerve stimulator shows four strong twitches. Pupils are small to midsize but you elect to give naloxone IV up to 0.4 mg. As expected, there is no clinical improvement. You examine the patient's chest and bilateral rales are heard over the lower half of her lung fields. You take her to the postoperative recovery ward with 100% oxygen via the face mask. A chest x-ray confirms the diagnosis of pulmonary edema.

Why is the patient in pulmonary edema? How will you treat this?

In this case after asking the nurse again, the anesthesia team discovered that the amount of recovered fluid from the peritoneal cavity was only 1950 ml compared to the 4400 ml injected. It is the anesthesiologist's responsibility to ascertain what these numbers are at the end of the case. Do not rely on others for that information, but check it yourself. Furosemide, 20 mg IV improved the patient over a 20–30 min period after a massive diuresis. It is advisable to do electrolyte estimations and an ECG. If the ECG shows new evidence of ischemia, the patient should be admitted for observation, and serial cardiac enzymes should be done. In the previous described case above, the patient was discharged from the hospital the following day and had an otherwise uneventful recovery [1].

Discussion

This patient was fine during general anesthesia, since positive ventilation of the lung prevented any signs of excessive systemic fluid absorption. This case is akin to a previously reported case [1] and other reports [2, 3]. It illustrates the fact that endoscopic procedures are not without significant anesthetic problems. One such complication is an excessive increase in intravascular volume resulting from absorption of irrigating fluid. This occurs in 0.14–0.34% of patients undergoing endoscopic uterine surgery [4]. Excessive intravascular volume manifesting as hemolysis, hyponatremia [5], and mild disseminated intravascular coagulation and/or pulmonary edema has been reported with glycine [6], dextrose [7], sterile water [8], or dextran 70 [9] when these agents are used as the irrigating solutions. The factors that influence the degree of fluid absorption include injection pressure, extent of tissue trauma, and amount of fluid and duration of infusion.

This case shows that the above complications can also occur during non-uterine endoscopic surgery when crystalloid solution is used as irrigating fluid. It is important to realize that these patients undergo extensive preoperative bowel preparation. Therefore, their intravascular volume is depleted, and often aggressive preoperative and/or intraoperative fluid resuscitation must be done. In this case the fluid management was made even more problematic as besides having a preoperative bowel preparation, and she was on preoperative diuretic therapy for chronic hypertension.

Recently the use of perioperative lung ultrasonography was able to diagnose one subclinical pulmonary edema [10]. Although lung ultrasonography is reported as feasible [10, 11], it will most likely not become routine application during these surgeries.

In these cases, the amount instilled into the peritoneal cavity and the amount removed should be recorded every 15 min [1]. Serum sodium levels should be checked when fluid absorption exceeding 1500 ml [3]. Had the patient not responded so quickly to the therapy, consideration as to placing a central line could be made. The same argument could be made for high risk patients undergoing this procedure [1].

Lesson

It is the anesthesiologist's responsibility to ascertain that the amount of fluid injected and the amount of fluid recovered from the peritoneal cavity, ideally, should be equal.

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Case 21: A Possible Solution to a Difficult Laryngeal Mask Airway Placement



A 40-year-old ASA 1 female with a Mallampati class 2 airway is scheduled for cystoscopy and biopsy of the bladder under general anesthesia. She weighs 80 kg and is 165 cm in height. Her only complaint is hematuria. This is her first hospital admission and first general anesthetic. She refuses a spinal or epidural. You induce general anesthesia with propofol 150 mg. The placement of soft reusable laryngeal mask airway (LMA) with the Brain technique proves impossible as the LMA will not pass the junction with the posterior pharynx. You attempt to insert the LMA with other different techniques, including rotating the LMA 180 degrees, to no avail. Unfortunately, no other LMA's are available.

Do you know of a trick to make a soft reusable LMA easier to place?

Yodfat [1] was the first to describe a technique for LMA placement using a stylet. His method which we have modified slightly is described below [2].

A conventional endotracheal tube style (Slick Stylette (Polamedco, Marina del Rey, CA) is folded in half (Fig. 1a). This reduces its length to approximately 22 cm. The stylet is lubricated and inserted into the LMA to be used (Fig. 1b). Care must be taken to ensure that the tips of the stylet do not protrude beyond the LMA aperture bars. With the stylet in place in the LMA, the LMA is bent to 90 degrees close to the junction of the airway tube and mask (Fig. 1c). The LMA is then lubricated in the usual manner. The LMA cuff is partially inflated and the tip is curled anteriorly. The patient's mouth is opened by grasping the mandible with the non-dominant hand while the LMA is inserted in a manner that effectively mimics the force vectors used in the Brain technique (Fig. 2). The tip of the LMA is placed against the hard palate and advanced, using continuous pressure, rotating the LMA so that the mask follows the curvature of the airway into its final resting position in the

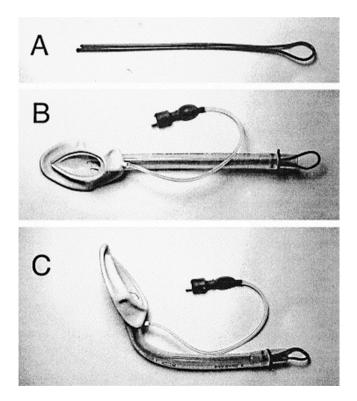


Fig. 1 (a) Photograph of the stylet bent in half. (b) View of stylet having been introduced into the LMA. Note that the tips of the stylet do not extend beyond the aperture bars. (c) The LMA with stylet, after having been bent 90 degrees and now ready for insertion (see text) (From Jaffe and Brock-Utne [2]. Reprinted with permission from Elsevier)

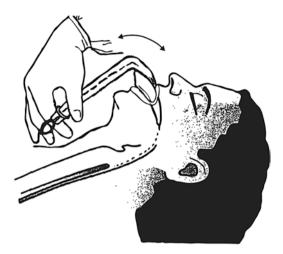


Fig. 2 The LMA with stylet in place is inserted into the airway in manner that mimics the original Brain technique. The figure has been simplified by omitting the non-dominant hand, which would be used to grasp and lift the mandible (From Jaffe and Brock-Utne [2]. Reprinted with permission from Elsevier)

pharynx. The strongly curved stylet serves as a functional replacement for the operator's finger. The advantage being that it can be kept safely outside the patient's mouth. The stylet is then removed by withdrawing it from the LMA tube. Finally, the mask is inflated in the usual fashion.

Discussion

There is no doubt the Brain technique has been associated with less than satisfactory seating of the LMA [3]. Brain insists that this is because many anesthesiologists do not use his technique correctly [4]. Whatever the reason for the less than satisfactory result in placing the LMA, many use the half-twist or full-twist 180 degrees' insertion technique. The original Brain technique describes the LMA as being held as a pen, with the index finger positioned against the proximal cuff. Then, with continuous pressure against the hard palate, the LMA is advanced into the pharynx. The big disadvantage of this technique is that the anesthesiologist's finger and knuckles may scrape against the patient's teeth. Patients with small mouth openings or difficult oropharyngeal passageways may require considerable effort, with multiple attempts to achieve proper LMA placement. Even in patients with normal airways, an anesthesiologist who is not blessed with slim fingers may get scraped by the patient's teeth.

Many of the LMA's now available have a fixed curve like the modification described here. So if you have a possible concern with placing an ordinary LMA,

then placing a stylet in the LMA may be solution, or use one of the curved LMAs if they are available. Be aware that in many parts of the world, the reusable LMA is the only LMA available [5]. A recent LMA review is worth reading [6].

Lesson

It is worth practicing this modification of the Yodfat LMA insertion technique until it is done easily. When faced with a difficult LMA placement, this technique can prove to be of real winner especially if you do not have the curved variety.

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Case 22: Postoperative Airway Complication Following Sinus Surgery



A 28-year-old man (ASA 1) with chronic sinusitis is scheduled for functional endoscopic sinus surgery (FESS) since he has failed medical management. He is 84 kg and 5 feet 10 inches. He has had one previous general anesthetic for an acute appendix at age 10. Otherwise his medical history and physical exam is unremarkable. He is at present not taking any medication and has no known allergies to medicines. He has a normal white cell count and his Hb is 14 mg%. After sedation with midazolam 2 mg IV, he is taken to the operating room where a routine general anesthetic is induced uneventfully. Tracheal intubation (grade 1 view) is done atraumatically on the first attempt after the patient is fully checked to be fully paralyzed with a nerve stimulator (vecuronium 7 mg.). The pharynx is deemed normal both preoperatively and during endotracheal intubation. The endotracheal tube (ETT) is secured, and bilateral air entry is recorded with presence of CO₂ on the capnograph. General anesthesia is maintained with oxygen, nitrous oxide, isoflurane, morphine, and fentanyl. The operation lasts 90 min, and the vital signs throughout the surgery are within normal limits. The estimated blood loss is 900 ml. No airways or oral packs are used during the surgery, except a nasal posterior pack that was placed before the FESS commenced and removed after surgery. The inferior nasal vault was not packed, but small hemostatic sponges were placed in the ethmoid cavities. Before the patient was fully awake, the pharynx was gently suctioned using Yankauer suction, an oral gastric tube placed on its first attempt, suction applied, and the tube withdrawn completely. 50 ml of gastric juice was aspirated. With the patient fully awake and able to follow commands, the ETT is removed. In the recovery room, the patient is comfortable with stable vital signs. However, 10 min later he complains of coughing and difficulty in breathing. Despite supplemental oxygen (6 l/min) via an oxygen mask, his oxygen saturation decreased to 86%. You are called back and examine the patient's chest and can hear only minimal scattered expiratory noises (stridors). Racemic epinephrine is given with minimal improvement. You put another saturation monitor on his finger but the saturation is still 86%.

What will you now do?

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Always remember in these cases to examine the throat too. In this case a large swollen and elongated uvula was seen. The tip of the uvula, deemed to be 13–14 cm long, could not be seen. The elongated uvula triggered from time to time a gag reflex with coughing and difficulty in breathing. In a previously reported similar case, dexamethasone 8 mg was administered and the nebulized racemic epinephrine continued [1]. The head of the bed was elevated to 75 degrees. After 2 h the patient was discharged from the recovery room to the ward. Dexamethasone (8 mg every 8 h) was continued. He was discharged home the next day with marked reduction in uvular swelling. There was no evidence of edema on postoperative day 3.

Discussion

There are many causes of uvulitis. The main causes include mechanical and thermal trauma, infection, chemical and allergic reactions, and non-allergic complementmediated disorders [2–7]. From these references, the disconcerting fact is that uvular edema can develop any time from 45 min to 24 h after the event that caused it. Dexamethasone is considered the treatment of choice for uvular edema. This is due to the drug's anti-inflammatory potency, which is 25 times greater than that of hydrocortisone, and a long half-life of 36-72 h. Steroids exhibit their effects by decreasing capillary endotracheal permeability, leading to a decrease in mucosal edema and decreasing the inflammatory reaction, by stabilizing lysosomal membranes. In posttraumatic uvulitis, steroids have been shown to bring about dramatic relief [8]. If it is believed that the uvular edema is an allergic reaction, then diphenhydramine is recommended [2, 6].

If you question a patient following this type of surgery, who complains of difficulty in breathing, it is most likely that he/she will report that there is something logged at the back of the throat. Besides the uvular edema, remember to look for other problems in the back of the pharynx, like dislodged packs or sponges, bone fragments, blood clots, or foreign bodies.

Lesson

- 1. Always examine your patient's throat prior to anesthesia.
- 2. Should postoperative respiratory obstruction occur following FESS, always remember to examine the throat, not just the chest.
- 3. The onset of uvula edema can be delayed for hours. It can also get worse before it gets better.
- 4. Treatment must be aggressive and the patient admitted to ICU for observation and treatment.

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Case 23: An Unusual Capnograph Tracing



You have anesthetized a healthy 24-year-old man (ASA 1E) for an emergency laparoscopic appendectomy. He has no history of previous anesthetic nor does he take medication regularly and has no history of allergy to medication. There is no family history of any problems with anesthesia. He is 75 kg and 6 feet. After rapid sequence induction, you secure the airway and see a CO_2 waveform on the capnograph (which incorporates a sidestream CO₂ monitor [Datex Capnomac Ultima, Datex, Helsinki, Finland]), confirming bilateral air entry. The endotracheal tube is taped at 23 cm. The vital signs are normal. However, on closer inspection of the capnograph waveform, you now notice that the capnograph is very different to what you normally see (Fig. 1). The tracing starts from a zero, and a normal plateau is reached, but just before the trace should normally go to zero, there is a marked peak in the tracing before rapidly returning to zero. Tidal volumes, respiratory rates, and minute volume are within normal limits. The peak pressure is 24 cm. The patient's vital signs remain normal. You have not seen anything like this before, and since you have checked the Narcomed 2B, North America Dräger anesthesia machine prior to the anesthetic, you are now wondering what this trace could mean.

Should you be concerned? What do you think the problem is? What, if anything, can you do?

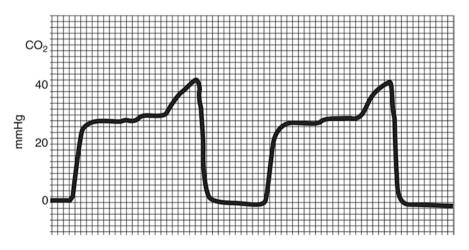


Fig. 1 Capnograph waveform (From Sims [1]. Reproduced from Anaesthesia and Intensive Care with the kind permission of the Australian Society of Anaesthetists)

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The cause of the problem is that the capnograph tubing from the patients breathing circuit at the mouth end is not screwed in properly to the Datex capnograph aspirating port [1]. As the Datex does not discriminate as to what gas it absorbed into the machine, room air is entrained and mixed with the patient's respiratory gases. Initially this mixing gives a lower than normal plateau. However, the reason for the peak is that it corresponds to the inspiratory cycle of the ventilator. Mixed expired gases, during the initiation of inspiration, are forced up the capnograph tube preventing less or no room air entrainment. These lead to the peak which is seen on the capnograph tracing.

Discussion

This unusual capnogram was a concern until the cause was found and easily rectified. The same size leak at the patient end of the sampling tube does not have as marked as an effect [2]. The described pattern is not seen when the patient is breathing spontaneously, but a falsely low end tidal CO_2 measurement is seen with no terminal hump [2]. During calibration, a falsely low end tidal CO_2 can be seen when an entrainment is present [2]. This problem with the sidestream carbon dioxide analysis cannot be seen with the mainstream carbon dioxide analyzer.

Capnography is the graphic display of CO_2 partial pressure in a "waveform" format (Fig. 2). Capnography is superior to capnometry, which is the measurement of numerical display of CO_2 concentration on partial pressure in the patient's airway during the respiratory cycle. Capnography assesses not only alveolar ventilation but

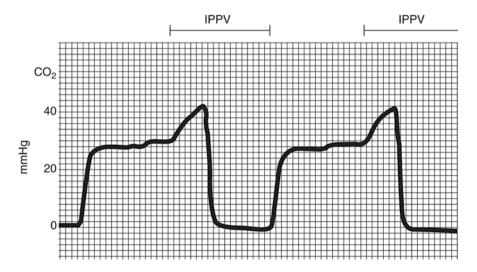


Fig. 2 Capnograph waveform (for explanation see text) (From Sims [1]. Reproduced from Anaesthesia and Intensive Care with the kind permission of the Australian Society of Anaesthetists)

the integrity of the airway, proper functioning of anesthesia delivery system, ventilator function, cardiopulmonary function, and potential rebreathing. Capnography has been used among other things to diagnose incompetent and/or expiratory values [3], spontaneous respiratory effect, lung disease, and CO_2 absorbent exhaustion [4]. An abnormal capnograph trace can also be seen when there is a longer than normal sample line combined with a cracked water trap [5]. In the latter case, the trace shows a mid-plateau hump (the dromedary sign) [5].

Lesson

Capnography serves as an anesthesia disaster early warning system. Changes from a normal waveform must always be investigated and corrected if possible. In this case, the unusual waveform was due to a simple human equipment failure.

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Case 24: A Respiratory Dilemma During a Transjugular Intrahepatic Porto-Systemic Shunt (TIPSS) Procedure



A 54-year-old man (55 kg) with a history of alcohol abuse and hepatitis is scheduled for a transjugular intrahepatic porto-systemic shunt (TIPSS) in the x-ray suite. He has received sclerotherapy for variceal bleeding but recurrent bleeding has ensued. He has no other major complaints. His INR is 1.1. A radial arterial line is placed preoperatively together with a 16 G. IV in his hand. Noninvasive monitors are placed, and general anesthesia is induced with etomidate, fentanyl, and vecuronium. After the endotracheal tube (ETT) is placed in the trachea, anesthesia is maintained with 50% nitrous oxide in oxygen with isoflurane 0.8%. Bilateral air entry is confirmed and the end tidal CO_2 is 32 mmHG. The end tidal CO_2 trace looks normal. The ETT is secured. The room is darkened as is routine in these cases. The operator (radiologist) is in close proximity to the patient's head, where he is placing a central line in the internal jugular vein so that he can place the shunt in the portal venous system. With the x-ray machine on the other side of the head, you have no access to the airway or feel for pulses in the head or arms (the latter being tucked along the side of the patient). Thirty minutes into the case, there is a sudden marked increase in the peak inspiratory pressure from 28 to 42 cm H₂O. All other parameters are within normal limits. You suspect right endobronchial intubation, but you are prevented from listening to the right side of the chest because it is made sterile and covered by sterile drapes. You are reluctant to pull the ETT back since you really do not know if there is right endobronchial intubation.

Is there anything you can do to confirm your suspicions without making the surgical field on the right side of the chest unsterile?

Ask the radiologist to do a fluoroscopy of the neck and chest. That is easy for him to do and will not disturb the sterile field. If you are right, then you will see that the ETT has passed the carina into the right main bronchus. When you pull the ETT back into the trachea, you should see that the peak inspiratory pressure returns to basal levels. Furthermore, you will see that during insufflation of the lungs, the liver does not descend to the same extent with the two lungs ventilated as compared to only right lung ventilation [1].

Discussion

TIPSS is a complex angiographic procedure performed in patients with end-stage liver disease. With the exception of hepatic encephalopathy, severe procedure and shunt-related complications are rare, and early mortality is low [2].

During TIPSS, endotracheal intubation is essential as access to the patient is severely limited. The room is dark. It is important to make sure that you have enough light so you can see your workstation and the monitors. This is one case in which monitors are essential, as close clinical observation of your patient is impossible. It has been recommended [1] that during such procedures, regular observation of the monitor can be beneficial to patient care. Indeed, it may be useful to tell the radiologist that he or she should also be aware of this unusual sign of one lung ventilation.

The radiologist usually selects these patients very carefully for his procedure. Many of these patients are critically ill with sepsis and DIC. You must always give prophylaxis for sepsis. Packed red cells and fresh frozen plasma should be readily available, as serious bleeding can occur from lacerations of hepatic arteries, etc. There is a procedure mortality rate of TIPSS up to 3% [3–5].

Many years ago I did a TIPSS procedure, where a sudden airway pressure increase was observed. In addition, there was also a dramatic decrease in blood pressure. In that case, we diagnosed bilateral hemothorax with the help of the fluoroscopy. Bilateral chest drains helped get the patient off the operating table and to the ICU. Unfortunately, she later died of DIC and sepsis.

Lesson

When faced with an increase in airway pressure during a TIPSS procedure, do not forget to request the use of fluoroscopy of the neck and chest.

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Case 25: A Tracheotomy Is Urgently Needed and You Have Never Done One



You are called stat to the Cath lab for a patient who has developed sudden difficulty in breathing. You find a 73-year-old woman with neck and face severely swollen. She is unresponsive with shallow, rapid, and labored breathing. She is receiving nasal oxygen at 4 l/min and the oxygen saturation is 86%. She has a diagnosis of a superior mediastinal syndrome. In an attempt to investigate this, the radiology staff has sedated the patient with IV fentanyl 100 microgram and IV 4 mg of midazolam. With the patient adequately sedated, a Shiley catheter has been placed in the right subclavian vein. They inform you that the subclavian artery has also been hit several times and there is now a large hematoma compressing her trachea. You listen to the chest but can hear little air entry. You assist ventilation with 100% oxygen via an Ambu bag, but with no improvement. You attempt to intubate the trachea with a Mac 3 blade but see nothing. You feel that succinylcholine will not be helpful as the patient is now relaxed and completely unresponsive. A two-person mask ventilation with a large oral airway is unsuccessful. The LMA that you have called for has not arrived. The neck is so swollen that you dismiss the use of a cricothyrotomy. A tracheotomy set is produced, and an ENT specialist is called for, but you are informed that he can only be there in 10 min. The oxygen saturation is now 76%, and there is a dramatic decrease in her pulse rate and blood pressure. Everyone is looking at you as the senior anesthesiologist. You know that you have never done a tracheotomy before by yourself and certainly never under these circumstances. However, since you have seen many tracheostomies, you feel this is the only option for this woman. You grab the scalpel and feel a great sense of insecurity and dread, but you have committed yourself as you make the incision.

How will you quickly secure a surgical airway?

This case happened to me and a great colleague of mine. What we did is described as such:

With the thumb and middle finger of the left hand, I attempted to displace the sternomastoid muscles on either side. However, in this case they were impossible to feel. The main reason for placing your thumb and middle finger in this manner is to protect the great vessels. With the scalpel in my right hand, I made a 5 cm incision in the midline from the cricoid cartilage (which in this case I could not feel). The incision should traverse skin and subcutaneous tissue only. My colleague started blunt dissection of the neck with his fingers. With his fingers he opened a tract so I could just barely see the trachea. With the scalpel, I cut through one of the membranes and inserted a cuffed endotracheal tube #5. With the airway secured, her oxygen saturation improved to 100%, and her heart rate and blood pressure normalized. Only when satisfactory airway control was achieved did we address the bleeding which incidentally was profuse. She must have lost approximately 200 ml of blood. To stop the bleeding, we packed the wound with 2 inch vaginal pack and kept pressure. The ENT surgeon appeared and congratulated us on a job well done. The patient survived the incident and went home after a short hospital stay.

Discussion

Moments of sheer terror do occur in anesthesia when one is unable to ventilate and unable to intubate, and one is left with very few options except a surgical airway. The ASA has guidelines for the management of the difficult airway [1]. Although useful as an educational tool, it is just that. The introduction in 1992 into the USA of the LMA has proved to be a major advance in the difficult airway management [2]. However, I do not think we would have managed to place an LMA successfully in the above case, due to all the swelling above and below the glottis.

The finger dissection of the neck by hand was the reason this woman survived. I initially tried to get access to the trachea with forceps, etc., but had to give up due to too much blood and swelling. From starting the finger dissection to the insertion of the ETT, it took less than a minute.

I personally do not like percutaneous cricothyrotomy or percutaneous dilatational tracheotomy. Mediastinal emphysema is a serious complication of these techniques as you are seeking for the trachea blindly [3, 4]. I have seen both complications in similar cases, and all have ended with a tracheostomy that saved the patient.

Recent current knowledge [5, 6] indicates that surgical cricothyrotomy is more reliable than percutaneous cricothyrotomy as a rescue method in "cannot intubate, cannot oxygenate" situations.

Patients, who have an upper airway obstruction that cannot be relieved by positive pressure mask ventilation or bypassed by oral or nasal tracheal intubation or LMA placement, must have an immediate surgical airway. This could be a surgical cricothyrotomy but preferably, in my opinion, a surgical tracheotomy.

Lesson

The take-home message in emergency tracheostomy performed by a non-ENT surgeon is to get access to and control the airway as rapidly as possible using a scalpel followed by finger dissection. Forget about the bleeding initially. The latter can be controlled by packing and pressure while you await an ENT surgeon.

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Case 26: General Anesthesia for a Patient with a Difficult Airway and Full Stomach



A 26-year-old female (ASA 2E), weight 260 lbs., height 5'6", and BMI 43, is scheduled for an emergency appendectomy. She has a class 3 airway but tells you that she has a previous history of a traumatic and difficult endotracheal intubation. She is otherwise healthy and takes no medication. She refuses a regional block, as she has a lot of back problems. You decide on a fiber-optic intubation, but you are fully aware that topical anesthesia and sedation must be kept to a minimum in an attempt to maintain the patient's protective laryngeal reflexes and decrease the like-lihood of gastric aspiration.

What fiber-optic intubation approach would you adopt for this case?

- 1. The patient should be placed in a fully upright sitting position, but with hip flexion minimized. This upright posture may also increase the pharyngeal space by gravity.
- 2. Instructing the patient to protrude the tongue and phonate can improve the diameter of the airway and therefore make visualization easier.
- 3. Ask an assistant, standing behind the patient, to gently push the jaw forward to help with visualization.
- 4. The one that does the fiber-optic intubation should be standing facing the patient.
- 5. I use the oral route. Some recommend using the nasal rather than the oral route in these cases, as the former provides a straighter passage of the scope. Unfortunately, epistaxis in this group of patients is a common occurrence.
- 6. It has been recommended that the pharyngeal airway may be made larger with the application of nasal CPAP on the contralateral nostril [1].

Discussion

Anesthesiologists often place their patients in the supine position out of habit. For this patient it is a good reason to break this habit.

Intragastric pressures in normal fasting adults may seldom reach above 30 cm H_2O [2, 3]. In most adults, the distance between the lower esophageal sphincter (LES) and the upper esophageal sphincter is 25–30 cm H_2O . Thus, even if the LES provides limited resistance to efflux of gastric content, it is highly unlikely that the normal intragastric pressure responsible for passive regurgitation would be sufficient to propel the gastric contents into the oropharynx in a patient sitting fully upright or even standing. The sitting position is more natural for the patient and allows face to face communication with the anesthesiologist. This makes for a more relaxed patient with less need for sedation and thus a more truly "awake" intubation.

Besides having the patient sitting up, appropriate antacid [4] and drug pretreatment to decrease gastric volume and acidity while increasing LES tone are considered standard of care [5–7].

Cricoid pressure has been described as making fiber-optic intubation more difficult [8]. There are other reasons why I would not recommend the use of cricoid pressure in these cases as highlighted in my editorial [9].

Adequate anesthesia of the airway should be achieved with a combination of topical and regional techniques. If regional techniques are used (I do not personally use them), like blocking the superior laryngeal nerves, then one must wait at least 8–10 min before instrumentation is done.

My personal recommendation for topical anesthesia in these situations is 8 ml of lidocaine 4% with 4 ml of cocaine 4%. I usually use only 6–8 ml of this solution, administered with an atomizer (MAD, mucosal atomizer device, Wolfe Tory Medical, Salt Lake City, UT 89808) or either the MicroMist nebulizer, Hudson RCI,

Temecula CA 92589, USA, asking the patient to pant like a dog. No matter what I use, I take my time and find that I can manage with very little sedation. For sedation I use meperidine up to 1–1.5 mg/kg, as it does not depress the ventilation like fentanyl, plus meperidine has an atropine-like effect [10]. Glycopyrrolate is also a good drug to use. I use 2 l of oxygen flowing continuously through the suction port in the fiber-optic scope. The oxygen airflow helps to push away airway secretions, giving a clearer view and also provides improved oxygenation.

A recent report [11] describes the awake insertion of a Proseal LMA in 980 lb. patient. Interestingly the Proseal LMA was used to deliver positive pressure ventilation throughout the intubation with no significant periods of hypoxia.

Lesson

Although, there are no clinical trials to support the contention that these patients should be in a full sitting position, the above theoretical considerations and years of experience indicate that this technique maximizes patient safety. In addition, it provides minimum stress and discomfort to the both patient and anesthesiologist [12].

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Case 27: A Jehovah's Witness Patient and a Potentially Bloody Operation



A 54-year-old male (ASA 1) weight 79 kg and height 5'11" is scheduled for a prostatectomy. While checking your anesthesia machine in the OR, you hear from the circulating nurse that the patient is a Jehovah's Witness. The scrub nurse mentions that she has had a patient like this die under a similar operation. In the hope that the patient may take albumin, you get two bottles of 4% albumin from the pharmacy and place it on your anesthetic table behind the anesthesia machine. You meet him for the first time in the preoperative holding area. He is surrounded by 15 of his family and friends. They inform you of the patient's unbending refusal to accept blood transfusions and all related blood products. You inform the patient that you will abide by his wishes, but tell him, in no uncertain terms that he may die from hypovolemia. His response is to say he'd rather die than get blood products. You write down on paper or enter in your electronic medical record that you have explained the serious consequences of his refusal. If your hospital is still on "paper," then get him to sign that he does not want any blood products and that he is prepared to die. You get this witnessed by as many of his family and friends that want to sign. You make a copy of this statement for your own record and place the original in the patient's chart.

His past medical history is noncontributory. He has had no previous surgeries. On examination nothing abnormal is detected and he has a class 1 airway. He is not interested in an epidural and prefers to be asleep.

You place an IV and give him midazolam 2 mg IV and take him back to the OR. In the OR there is the same scrub nurse and circulator nurse that you have previously met that morning. However, now you see that there is one additional circulating nurse. You introduce yourself to the new nurse whom you have not seen before. She tells you her name and says pleased to meet you. You presume, since she is new in the OR, that she is being in-serviced as a circulating nurse. The patient is placed on the OR table, and you proceed to put on the noninvasive monitors. You record the baseline vital signs and start to preoxygenate the patient. The new nurse seems out of place and keeps looking at you and the anesthesia table behind you. You think she may be looking intently at the albumin bottles. The other circulating nurse holds your endotracheal tube and is positioned to assist you with the endotracheal intubation by the patient's head. The new nurse seems disinterested in observing what the circulating nurse is doing. A sixth sense tells you that something may not be right. You stop your preoxygenation and turn to the new nurse.

What is it that bothers you and what would you say to her?

This situation happened to me. I stopped the preoxygenation and turned to the nurse and asked her: "Is the patient a friend and/or relation of yours?" She blushed and muttered: "No." I then said to her: "Kindly tell me if you are or are not a Jehovah's Witness?" She admitted she was and that the patient was someone she knew from the church. On further questioning, she said that she had been asked by the family to observe in the OR and to make sure the patient did not get any blood or blood products. She normally worked in the hospital but in the outpatient clinic. This was her day off. I asked her to leave; otherwise I would have to call security to have her removed. I told her that I would not anesthetize the patient unless she left. She excused herself and left.

Discussion

There are three "take-home messages" from this case.

1. It is imperative that you always know who is in the OR with you and why they are there.

In the case described above, the circulating nurse had presumed that the nurse was new and needed to see how things were done. The circulating nurse should have made sure she knew who she was and what she was doing there.

- 2. Some hospitals have special forms for people who refuse blood and blood transfusion to sign. The two forms are called conditional refusal or absolute refusal (see below). It is important to note that parents and/or guardians are usually prohibited by courts from making this decision for children, minors, or incompetent patients, if such refusal will threaten the life or health of the child, minor, or incompetent patient. If this situation applies, the advice of a lawyer or court may have to be obtained. Also be aware that different countries have different rules.
- 3. One may think that one can refuse to do the case as an anesthesiologist. Certainly that is one's right, but if you do not do it, then one of your colleagues will have to do it. Will he or she thank you? I have always done these cases as they come along. However, I have managed often, when I got the patient alone, to convince him or her that conditional refusal is the way to go. If that does not work, I try to convince them to receive cell-saver blood and/or albumin. In most cases they accept these options. But you can be unlucky. A very good friend and colleague lost a patient like this on the table from blood exsanguinations. But as he said: "I was assigned this case, I did not like it, but what should I do? Give it to you?"

Refusal to Permit Blood or Blood Products Transfusion
I request that no blood or blood products to administered to:
(name of patient).
1. Conditional Refusal.
I request that no blood or blood products be administered unless in the opinion of my physicians,
serious injury or death may occur if such transfusion is not administered.
Date Time
Signature
Relationship(Patient, parent, conservator or guardian)
Witness
Witness
2. Absolute Refusal
I refuse to have any blood or blood products administered under any circumstances including the
possibility that death may occur if blood or blood products are not administered. This refusal is
absolute.
I hereby release the hospital, its personnel and my physicians from any responsibility whatsoever
for unfavorable reactions or any untoward results due to my refusal to permit the use of blood of
blood products. The possible risks and consequences of such refusal on my part have been fully
explained to my by my physicians and I fully understand that such risks and consequences may
occur as a result of my absolute refusal to receive blood or blood products.
Date Time
Signature
Relationship (Patient, parent, conservator or guardian)
Witness
Witness

Lesson

Always be observant of new faces that are working with you in the OR. If you see a new face, then you should introduce yourself and find what their reason for being there is. Below is a suggested form to be used with patients who are Jehovah's Witness:

Suggested Reading

 Rollins KE, Contractor U, Inumerable R, Lobo DN. Major abdominal surgery in Jehovah's Witnesses. Ann R Coll Surg Engl. 2016;98:532–7.

Case 28: Laparoscopic Achalasia Surgery



A laparoscopic surgery for achalasia is just about completed. At that point the surgeon often asks the anesthesiologist to insufflate the stomach with air. This is done to make sure sutures are doing their job and no leak is seen. The anesthesiologist usually does this with a large syringe attached to the proximal end of the patient's nasogastric tube. This technique is not easy to do and has limitations. The main problem with this technique is that it is very difficult to maintain a steady pressure in the stomach.

The surgeon you are working with today has had two patients recently where the gastric sutures have slipped postoperatively. Peritoneal leaks led to reoperations, etc. You attempt bravely to inflate the stomach with your large syringe, but you fail miserably. He turns to you and says: "I don't really want to close up before I can be assured that there is no leak in my suture line. Is there nothing else you can do?"

What will you suggest?

Use the emergency jet ventilator.

Discussion

Most modern OR's have an emergency jet ventilator attached to the back of the anesthesia machine or directly into the oxygen wall outlet. We have used this technique for many years and found it efficient and safe [1]. A 14-gauge catheter is attached to the distal end of the jet ventilator. The catheter is then placed in the proximal large lumen of the nasogastric tube. The stomach is gently distended by pressing on the lever of the jet ventilator. One can then observe the stomach being distended on the TV monitor. It is important to remember that the pressure that the jet ventilator can generate is 50 psi. I always insist that the anesthesiologist practices on a surgical glove initially to get a feel for the pressure that is produced, before using it to distend the patient's stomach. There is always a theoretical potential to cause overdistension of the stomach. However, if you watch the stomach's response to your insufflation on the TV monitor, this technique is both safe and efficient.

Lesson

A jet ventilator can safely and efficiently achieve intraoperative insufflation of the stomach, to the surgeon's satisfaction.

Reference

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Case 29: Sudden Intraoperative Hypotension



An 18-year-old male (55 kg) with a history of spina bifida and developmental delay is admitted for surgical treatment of a recurrent right ischial and peroneal pressure sore. He has undergone multiple previous general anesthetics for urological problems without incident. Of special note is the fact that he has no allergy of any kind, especially no latex allergy. He is seen in the preoperative holding area and an IV inserted in his arm without much difficulty. The patient is very interested in the tourniquet. He is given one and he immediately starts chewing on it for comfort. He carries on chewing for approximately 30 min until anesthesia is induced. After he falls asleep, it is removed. A routine general anesthetic is given and then he is turned prone. Muscle relaxation is achieved with vecuronium 6 mg. You listen to his lungs and confirm that there is bilateral air entry. He is given Kefzol 1 gram and the operation starts. One hour after the induction of general anesthesia, the patient suddenly develops high peak inspiratory pressures (45 cm H₂O), hypoxemia (85%), hypotension (systolic 70 mmHg down from 120 mmHg), and a dramatic decrease in endtidal CO₂. You increase the FIO₂ to 1. You listen to the lungs and discover that there are no breath sounds over the left lung and only very minimal sounds from the right. The endotracheal tube (ETT) is withdrawn from 23 to 20 cm at the lips, with no change in the above respiratory findings. You now request that the patient is turned supine. When that is done, you palpate the cuff in the trachea. Peak inspiratory pressure remains elevated (over 50 cm). You can no longer feel the superficial temporal artery, indicating that you have a systolic blood pressure below 60 mmHg. The heart rate is increasing to 150 bpm. You give epinephrine (50micrograms). There is some improvement in his cardiovascular status, and the chest exam now reveals profound wheezing bilaterally. An additional 20 µg of IV epinephrine, 100 mg of IV hydrocortisone, and 10 puffs of albuterol through the ETT cause near resolution of his bronchospasm. The vital signs return to normal. You are delighted with this turn of events but wonder what the cause could have been for this sudden severe intraoperative hypotension. You discount an overdose of inhalation anesthetic, but you cannot ignore an allergic reaction to, e.g., muscle relaxants and/or antibiotics.

Are there any other causes you would consider? If so, what will you do?

Solution

Consider a latex allergy [1, 2]. The suggested management of a potential latex allergy when it occurs intraoperatively is as follows:

- 1. Remove any latex materials. Surgeon must change gloves.
- 2. Ventilate with 100% oxygen.
- 3. Consider aborting the procedure.
- 4. Administer fluids.
- 5. Administer epinephrine 50–100 μ g/kg IV bolus for hypotension and/or severe bronchospasm.
- 6. Take several samples of blood for tryptase measurements [2–4], IgE antibodies, and complement C3 and C4 [3].

In our case [2], with the vital signs returning to normal, the surgery continued with full latex-free precautions. The operation was concluded uneventfully. Tests later revealed that the patient was highly sensitive to latex.

Discussion

Latex is the milky sap that is extracted from trees in the Amazon and used to manufacture natural rubber products. Latex products include surgical gloves, catheters, etc. So-called hypoallergenic gloves have latex in them, only in a smaller amount.

The timing of latex anaphylaxis presentation during anesthesia is from 30 to 60 min [4]. This coincides with a delayed airborne exposure or with mucous membrane exposure at the beginning of the surgical procedure. In this case, which is similar to one that we have previously described [2], we believe that the extremely high exposure, over a 30 min period, to chewing on a latex tourniquet contributed to the development of the acute life-threatening latex allergy. Slater et al. [5] found that 34% of children with spina bifida had antibodies in their serum specific for latex rubber protein.

In all cases where there is a sudden cardiovascular collapse from an allergic cause, an attempt should be made to see if there is any generalized rash or flushing of the skin. Although not specific to latex allergy, it will indicate that you are dealing with a serious intraoperative anaphylaxis. Although cutaneous manifestations of latex are more common in nonsurgical cases [6], these can easily be explained by the fact that the anesthetized patients are mostly draped [7].

Lieberman [4] compared 1158 cases of latex anaphylaxis that were not associated with anesthesia with 583 cases during anesthesia. There were no reported cases of cardiovascular collapse in nonsurgical patients, while surgical patients had over a 50% incidence of cardiovascular collapse. Respiratory problems, on the other hand, seem more equally distributed between the anesthetized and non-anesthetized patients.

A recent review [8] has confirmed the relevance of several clinical features as risk factors for anaphylactic reactions induced by anesthetic agents: older age, asthma, hypertension, and antihypertensive drugs. The authors observed increased levels of serum basal tryptase in severe reactions: this finding may signify that this biomarker is useful for the identification of patients at risk.

Lesson

In this case, which is similar to a previously described case [2], we learnt three things:

- 1. Patients with no history of latex allergy can develop a life-threatening latex allergy at any time. This is especially true in repeat urological procedures, as these have a high propensity to latex allergy.
- 2. Patients who have a potential theoretical risk of developing latex allergy should be advised and prevented from chewing on and/or be exposed to latex products prior to general anesthesia.
- 3. Finally, all hospitals should now have all latex-free anesthetic equipment, including latex-free tourniquets.

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Case 30: Blood Pressure Difference Between a Noninvasive and an Invasive Blood Pressure Measurement



A 54-year-old male (ASA3) is scheduled to undergo an automated implantable cardioverter-defibrillator (AICD) change under general anesthesia. His past history is remarkable for coronary artery disease, hypertension, and insulin-dependent diabetes mellitus. He is 230 lbs. and 5'5''. You meet him in the catheterization laboratory. As usual the place is in partial darkness and you are asked not to put on the light. So in the darkness you place noninvasive monitors. A right radial arterial line is secured prior to induction of anesthesia (Transpac IV monitoring kit. 84" disposable transducers with a 3 ml squeeze flush. Abbott Critical Care System, Abbott Laboratory, North Chicago, IL.60064). A seemingly normal-appearing arterial waveform is present with a blood pressure of 149/116 mmHg and a mean of 129 mmHg. A simultaneous noninvasive blood pressure of 105/70 with a mean of 82 mmHg is obtained in the left arm. You change the noninvasive cuff to the right arm and get the same reading as in the left arm. The transducer is located in the midaxillary line and has been zeroed by you just before the arterial line was placed. Since you have just zeroed the transducer, you dismiss that as a cause for this difference in blood pressure reading. Furthermore, you did not see any offset when the stopcock was opened to atmosphere.

Besides re-zeroing what else would you do?

Put the light on in the laboratory. Now you see that on closer inspection of the transducer assembly, it is noted that the stopcock lever arm is directed upward from the horizontal by approximately 15 degrees. In the darkened room, somebody may have bumped inadvertently into the transducer stopcock.

Discussion

When the lever is placed in the correct horizontal position, the arterial waveform shifted downward on the display monitor (Fig. 1). The arterial pressure is now 100/65 with a mean of 77 mmHg and similar to the values obtained from the noninvasive blood pressure measurement. The reason for this increase is that the pressure from the flush bag admixes with the arterial line pressure when the stopcock lever arm is misaligned, creating an overestimation of arterial pressure. The admixed arterial waveforms crease an artifactual waveform which appears realistic but numerically is elevated. This case is akin to one previously described [1].

Never anesthetize anyone in the dark. There should be very few reasons that necessitate a darkened room when you anesthetize your patient. During the operation it is a different matter.

Based on this case, we did a study with institutional approval for human research and with informed consent. Each patient had an identical arterial monitoring setup as in the described above case report. Baseline waveform and pressure values were strip chart recorded. The stopcock lever arm attached to transducer assembly was turned 90 degrees upward from the horizontal position. The stopcock lever arm was then slowly turned toward the horizontal position. At approximately 15 degrees above horizontal, an arterial-like waveform was still present which produced a significantly higher pressure than the baseline value. When the stopcock lever arm was turned back to the horizontal, the waveform and pressure values returned to baseline (Fig. 1). The results of this study showed that in 8 out of 10 patients, the arterial

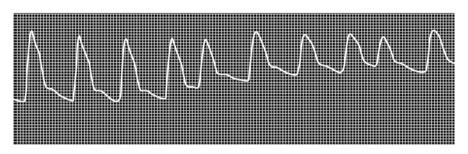


Fig. 1 Right radial arterial pressure monitoring catheter showing normal appearing arterial waveform with a blood pressure of 149/116 mmHg and a mean of 129 mmHg (From Eggen and Brock-Utne [1]. Reprinted with permission from Wolters Kluwer Health, Inc.)

blood pressures were artifactually increased by manipulation of the stopcock lever arm. It was noted that the degree of overestimation was in direct relation to the flush bag pressure. It was also noted that the pulse pressure of the artifactual waveform was narrower than the baseline waveform [1].

Another problem with the arterial line can be "damping," i.e., the magnitude of the difference between the input pressure and the transfused pressure. We had a case some time ago when a radial artery trace was working very well until it suddenly plummeted to zero. The cause of our dilemma was a clamp that the nurse had used to secure wires etc., from a microscope, along the side of the patient. Unfortunately, the clamp had inadvertently clamped our radial artery line [2].

Lesson

- 1. There are many reasons for obtaining artifactual arterial blood pressure measurements. One reason is if you leave the transducer stopcock at about 15 degrees from the horizontal. Inappropriate treatment may be given if unrecognized.
- 2. Remember that if you work in the dark, this can potentially interfere with patient care.

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Case 31: Severe Decrease in Lung Compliance During a Code Blue



An 80-year-old, weight 90 kg and height 5'7', male with a past medical history of coronary artery disease, diabetes mellitus, right inguinal hernia, hypertension, chronic obstructive pulmonary disease, and end-stage renal disease requiring hemodialysis is admitted to an intermediate ICU with upper gastrointestinal bleeding. He is conscious, pale, cold, and clammy. Since he complains of being cold, he is covered with several blankets. His BP is 90/40 and his heart rate is 120 sinus rhythm. His Hct is 20, and he is transfused with three units of packed red blood cells over a 40-min period. Following this he becomes unresponsive with very weak respiratory efforts. A one-stick radial arterial blood gas shows a pH 6.9, pCO₂ 107, and paO₂ of 53 on a 100% O₂ non-rebreather mask.

At this point you are called to manage the airway. When you arrive, a nurse is vigorously ventilating the patient with an Ambu bag via a face mask, but despite this his O₂ saturation remains in the mid-70s. He is supine in his bed and still covered with several blankets from his nipple down to his toes, with the exception of the right inguinal region, which reveals a balloon-like mass in the right inguinal region. You notice this because the intern has just placed an arterial line in his femoral artery. His femoral arterial line shows a blood pressure of 80/40 mmHg and a heart rate of 120 sinus rhythm. You confirm the blood pressure is not below 60 mmHg as you can palpate the superficial temporal artery. You turn your attention to his airway and note there is hematemesis on his pillow. You immediately place an endotracheal tube (ETT) in his trachea without sedation or relaxation. You ventilate his lungs with an FIO₂ of 100%. End-tidal CO₂ is seen (Easy Cap II CO₂ detector, Nellcor, Pleasanton, CA, USA), and bilateral breath sounds are heard. You are very concerned as you notice that the pressure required to hand ventilate the patient is extraordinarily high and seems to get higher and higher. Unfortunately, the oxygenation only improves to mid-80s. You repeat the laryngoscopy and confirm the correct placement of the ETT. A suction catheter is passed throughout the whole length of the ETT, and no secretion of note is obtained. You give the patient 6 mg of vecuronium and spray down the ETT with albuterol; there is only marginal improvement. You obtain a capnometer and confirm that CO_2 is present in the expired air, and as expected the CO₂ trace shows an obstructive pattern. A chest x-ray has been called for but not yet done.

Is there anything else you could do to attempt to find the cause of your ventilatory dilemma?

You undrape the abdomen. You see a very distended abdomen and the balloon-like mass in the right inguinal region points to a diagnosis of a tension pneumoperitoneum. A post-intubation chest x-ray (taken after the abdomen is undraped) reveals a massive amount of free air under the diaphragm. You insert an 18-gauge angiocath into the midline, 2 cm subxiphoid. A large amount of gas is heard evacuating through the catheter. Immediately the patient's chest compliance improves and his O_2 saturation rises quickly to 100%. His vital signs also improve, with BP 160/90 and heart rate of 90 bpm. In a previous case report [1], the patient was extubated the following day. Investigations found no extravasation of contrast, and a conservative approach was decided upon. He was discharged 15 days after his admission.

Discussion

It is postulated that the patient initially developed respiratory failure secondary to fluid overload. The vigorous mask ventilation sent much of the air into the stomach, distending it and causing an air leak through his gastric ulcer into the intraperitoneal space and into his right inguinal hernia. Despite exposure to the chest for auscultation, the abdominal distension was not obvious until the abdomen was uncovered. The abdomen should always be seen in these cases, especially in cases of perforated viscus, so as to alert the anesthesiologist to inadvertent pneumoperitoneum. This distention, if it had remained undiagnosed, could have proved fatal.

The blankets that covered his abdomen, delayed the diagnosis of pneumoperitoneum. The main causes of accidental pneumoperitoneum are intestinal perforations [2–4]. The balloon-like hernia was a clue that was missed by the medical team. However, when the blankets were removed, correct and definite treatment was instituted.

Patient's draping can potentially cause a disaster in other situations too. This can happen when, unknown to the anesthesiologist, the endoscopist injects large amounts of air into the esophagus and stomach of an anesthetized patient for better visualization. Previous reports have shown that excess air into the stomach can markedly decrease chest compliance, especially in children [5] but has also been known to do so in adults [6].

Lesson

Always listen to the epigastric region following endotracheal intubation. If this had been done, most likely the distended stomach with the tension pneumoperitoneum would have been discovered.

Always also consider tension pneumoperitoneum if ventilatory pressures are excessive after successfully placing an ETT in a patient's trachea.

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Case 32: Shortening Postanesthesia Recovery Time After an Epidural. Is It Possible?



You are assigned to anesthetize a patient for a 30-min knee arthroscopy. The patient is a 38-year-old man, weight 75 kg, height 6'0, and ASA 1, and is scheduled to go home the same day. He is terrified of having a general anesthetic and would like a regional block. The surgeon feels that a lidocaine infiltration of the knee joint will not give sufficient analgesia and suggests a spinal or an epidural block. Since your experience with a sciatic/femoral block is limited, you do not discuss this option with the patient. The patient is agreeable to an epidural or spinal but states that he needs to be out of the hospital by 10 am. He is the first case of the day and you are seeing him at 7 am. You tell him that he may be in the recovery room for 2–3 h before being discharged from the hospital, as the recovery room staff has to follow standard criteria for discharge. You give him an epidural, which works fine. The surgery commences at 7:35 am. Unfortunately, the surgery takes longer than anticipated, and the time of arrival in the recovery room is 8:30 am. On arrival in the recovery room, the patient is awake, cooperative, and with 66% motor block according to Bromage scales [1]. (The scales state that inability to move toes, knees, or hips equals 100% motor block; ability to move toes but not knees equals 66% motor block; ability to partially move knees equals 33% motor block; ability to fully move knees equals no motor block.)

You realize that you may have a problem in getting him discharged from the recovery room by 10 am. Is there anything that you can do to shorten the time he spends in the recovery room, other than having used a short-acting drug like chloroprocaine or telling the nurse that the patient can go even though he may not meet the hospital criteria for discharge?

We have previously shown that one can shorten the postoperative recovery time for knee arthroscopy patients by injecting 20 ml of saline into the epidural space at the end of surgery [2]. No complications were noted in our study especially due to the possibility of cephalad spread of the drug.

Discussion

Same-day discharge from the recovery room following a regional block depends also on the recovery from the block. Johnson et al. [3] were the first to show that unwanted motor block caused by epidural anesthesia can be reversed by the epidural injection of saline. Unlike Johnson, who used three separate injections of 15 ml of saline, we used only one-time 20 ml saline bolus. In our study we found that the saline injection at the end of the surgery reduced the overall recovery room stay by 40 min. It is possible that, had we used the dosage regime suggested by Johnson, we may have had a different or even better result.

The mechanisms of action of this reversal are thought to be several. Neural block can be reversed in vitro by washing nerves with crystalloid solutions [4, 5]. Injection of epidural saline may reduce the intensity of neural block by diluting the local anesthetics [2]. Local anesthetic can also be spread by the saline both caudal and cephalad. This enhances the clearance of the drug by vascular and lymphatic uptake. Removal of the drug could potentially occur because the saline 0.9% is acidic (pH = 5). This pH change could promote ion trapping of the charged local anesthetic molecule in the epidural space.

The economic pressures are increasingly important in the delivery of surgical services. Anything that can decrease the time in the recovery room leads to decreased staffing needs. In our study [2], we speculated that this technique could save the hospital \$12,650 per year (1998) if there were 10 arthroscopic procedures performed under epidural per week. Our study [2] has been validated by other researchers [6].

Lesson

The saline epidural injection technique at the end of a surgical procedure decreases the overall length of stay in the recovery room and may have a positive impact on patient satisfaction and staffing levels.

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Case 33: At Times You Need to Be a MacGyver



You find yourself in a foreign land on a medical mission with plastic surgeons repairing facial deformities. As the only anesthesiologist, you are in charge of the anesthesia equipment. You are requested to anesthetize a large man (180 kg) for removal of scars from his face. His neck is over 40 cm in diameter. Unfortunately, your box with airway equipment like a fiber-optic scope, gum elastic bougies, etc. has not yet arrived. Through a translator (you don't speak his language) you tell him them you would like to attempt an awake intubation under local anesthesia. The patient is terrified and wants to be asleep. You realize that you may need to have a gum elastic bougie as a backup. You start looking for possible ways of making a bougie from what you have available. The 18 Fr nasogastric tube would be too soft and the suction catheters you have are too short.

Is there anything you could do to make the nasogastric tube stiffer and therefore use it as a bougie?

The nasogastric tube with some modification can be used as an alternative to the gum elastic bougie [1]. The modification consists of placing one part of a paper clip into the distal orifice of the nasogastric tube via the small air vent channel and advancing proximally (Fig. 1). The distal end of the paper clip is then pushed into the distal blind pouch of the air vent channel to minimize any danger of inadvertent trauma by the paper clip (Fig. 2). The other end of the nasogastric tube is then cut, making the remaining tube with clip 60 cm long. The end of the nasogastric tube, with the clip in it, is bent to a desired curvature and the whole tube placed in a basin full of ice. Within a minute, the now converted nasogastric tube is rigid and can be used as a bougie.

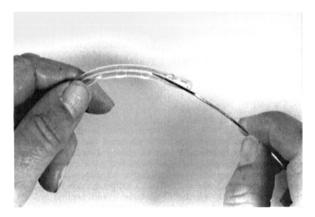


Fig. 1 Insertion and proximal advancement of paper clip via the distal orifice of the nasogastric tube into the small air vent channel (From Manos et al. [1]. Reprinted with permission from Wolters Kluwer Health)

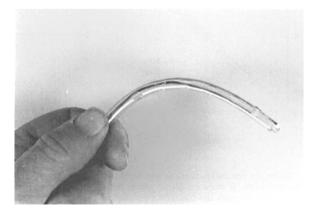


Fig. 2 Distal end of paper clip backed into the distal pouch of the air vent channel to minimize any danger of inadvertent trauma by the clip (From Manos et al. [1]. Reprinted with permission from Wolters Kluwer Health)

Discussion

This solution is meant to assist anesthesiologists only in a third-world environment when they find themselves without a bougie and need to manage a potentially difficult airway. Only by working in the third world can one begin to understand the enormous difficulties and frustrations that our colleagues in these places face on a daily basis [2].

An 18 Fr nasogastric tube is suitable for 8.0 mm and larger endotracheal tubes (ETTs), while the 14 Fr tube can be used for 6.0–7.0 mm ETTs. Nasogastric tubes have the added advantage over a gum elastic bougie because they can be converted into jet stylets providing oxygen. This can be done by inserting a 14-gauge intravenous catheter (Cathlon IV, Criticon, Tampa, FL) into the proximal end and attaching it to a transtracheal jet ventilation system. For the 18 Fr nasogastric tube, the catheter should be inserted all the way to the neck to ensure an adequate seal. The nasogastric tube can also be used to provide oxygen to the spontaneously breathing patient with the help of a standard 3 mm ETT adapter for the 18 Fr size and a 2.5 mm ETT adapter for the 14 Fr size. When the adapter is inserted into the proximal end, the tube can be attached to a conventional anesthetic circuit to insufflate oxygen.

Lesson

This alternative to a bougie may prove useful when a difficult endotracheal intubation is anticipated and no other adjuncts to secure the airway are available. The added advantage of the nasogastric tube is that it allows one to insufflate oxygen.

Please also review Chap. 12 for history and how to successfully use the bougie.

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Case 34: Delayed Cutaneous Fluid Leak from a Puncture Hole After Removal of an Epidural Catheter



You are called after hours to evaluate clear liquid leaking from an "epidural catheter puncture hole." The patient is a 50-year-old Asian male (ASA 2) who had an exploratory laparotomy with gastrostomy for gastric cancer 5 days previously. He had an epidural catheter through which epidural hydromorphone had been used until the second postoperative day, when it was removed intact. The patient is sitting up in bed and not complaining of anything, not even a headache. His past history and examination are noncontributory.

On examination, the dressing applied to the area of the leak is soaked, and the patient's bedding is very wet. At the site of a L4–5 puncture, clear liquid is dripping out of the puncture site at a rate of about 10–12 drops/min. Closer inspection of the patient's back reveals evidence of minimal dependent edema.

How would you proceed to establish if this is cerebrospinal fluid (CSF) or subcutaneous edema fluid without resorting to CT scan, radionuclide myelography, or measurements of CSF-specific isoenzymes?

You collect fluid from the puncture hole and send it for analysis of glucose, chloride, and protein. Thereafter you insert an 18-gauge intravenous catheter 4 cm lateral to the epidural site, and insert it 1 cm into the tissues. In a previous study [1], fluid was seen dripping from that site too. Fluid was again collected, and the values obtained were found to be similar to the ones obtained from the lumber puncture site. A diagnosis of subcutaneous edema fluid leak was made from the puncture hole. The expensive and time-consuming CT and the CSF-specific isoenzymes were not done [2].

Discussion

Table 1 shows a comparison of various biochemical values for differentiating CSF and interstitial fluid.

CSF fluid-cutaneous fistulae are known to occur following trauma, surgery, and infections. On the other hand, fistulae secondary to subarachnoid of epidural punctures are supposedly rare [3, 4]. But this has been challenged [5, 6]. In our case the correct diagnosis was made without resorting to expensive tests. It is important to realize that all CSF fistulae previously reported had occurred within 48 hours after the removal of the catheter. We were very anxious about this case as a CSF leak of this magnitude through a small cutaneous fistula would have required a substantial increase in CSF pressure. Others have recommended a lumber puncture to obtain a CSF sample in a case similar to ours [7]. This is not recommended, as increased intracranial pressure may be present. A diagnosis of a CSF cutaneous fistula may require special radioisotope myelography [8], when sufficient fluid cannot be obtained. If the suspected fluid can be collected in adequate quantity (0.5 ml), simple chemical analysis can avoid time-consuming and often expensive diagnostic tests [1, 5-7]. If the chloride and glucose values for the fluid are ambiguous, then the collected fluid can be tested for CSF-specific acetyl cholinesterase [2]. This may occur when CSF mixes with interstitial fluid as it travels through the fistula.

	Glucose mmol/l	Chloride mmol/l	Protein g/l	Ach
Plasma	4.4-6.6	90–110	60-80	-
CSF	2.75-4.12	122–132	0.15-0.45	CSF. Specific enzyme
Interstitial fluid	3.85-6.05	95–105	60-80	Nonspecific enzyme

Table 1 Comparison of various biochemical values for differentiating CSF and interstitial fluid

Lesson

This simple, inexpensive, and rapid test can prove whether the problem is caused by CSF or interstitial fluid. Expensive and time-consuming tests may prove to be totally unnecessary.

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Case 35: Traumatic Hemothorax and Same-side Central Venous Access



A 35-year-old woman is admitted to the emergency room after attempting suicide by jumping from a bridge. She is in severe pain, orientated for time and place. Her heart rate is 120 bpm and her BP 85/40. She is breathing 100% oxygen through a non-rebreathing mask with safety vent (Hudson RCI, Temecula, CA, USA), and her oxygen saturation is 96%. She has multiple fractures, including the pelvis, right humerus, ninth thoracic vertebra, and ribs 6 through 10 on the right side. She has a right-sided hemothorax. The right subclavian vein is cannulated using the infraclavicular approach and the Seldinger technique. A cordis catheter (PSI kit, Arrow International, Inc. Reading. PA 19605, USA) is inserted into the vein, and blood is easily withdrawn for chemical analysis. These include INR and cross matching for 6 units of red blood cells. After inserting the right chest drain, 1500 ml of blood is drained rapidly without any ill effects. A new chest x-ray shows complete resolution of the hemothorax, and the subclavian catheter is seen in the correct place. The central venous pressure (CVP) is zero and fluctuates with respiration. Blood arrives and 4 units of packed red cells are given rapidly through the subclavian vein via a Level 1 Fluid-system warmer 1000 (Level 1 Technologies, Rockland, MA 02370). The emergency room staff is concerned because despite continuous volume replacement with 31 of crystalloids and albumin 250 ml ×4 through a 16-gauge IV catheter in her right hand, her blood pressure deteriorates, and increased drainage of dark blood is seen from the chest drain. A diagnosis of laceration of major vessels in the chest is made, and you are called to anesthetize this patient for a right thoracotomy.

You arrive and assess the patient and tentatively agree with the diagnosis. You place two additional cannulae into the right internal jugular and right femoral veins. Blood is easy withdrawn from both catheters. You attach the subclavian vein to a CVP monitor, which again shows a value of zero and fluctuates with respiration. More blood is given through the new large-bore catheters and the patient seems to stabilize. Dark blood is still draining out of the right chest drain but at a slower rate. The laboratory reports that the INR is 2.3. The surgeon orders FFP. You are still looking at the dark blood coming out of the chest drain and wondering about the increased INR, as clinically she does not seem to be oozing. You send off a repeat INR this time from the femoral vein. The surgeon is keen to start the operation and you agree reluctantly but you feel there is something wrong.

Why are you concerned?

This case is similar to ones reported earlier [1, 2]. In the above case, minimal bleeding was found in the lung cavity, but the subclavian catheter was seen lying in the pleural cavity. As blood had been given only through the subclavian vein catheter, the color of the drained blood did not raise any suspicion, although it was noted to be very dark. However, the increased INR (the sample taken from the subclavian vein) indicated that the initial blood sample had come from the hemothorax. The repeat INR from the femoral vein showed a normal value of 1.1.

Discussion

Central venous catheter is useful for assessing fluid balance and for rapidly administering large volumes of blood and fluid. Blood reflux and respiratory fluctuations are considered reliable signs of correct placement of the catheter. However, this has been questioned [1-3]. This would especially be true if the central venous catheter is inserted on the same side as the hemothorax and/or blood has already been transfused through the catheter. In these cases, reflux of blood and/or respiratory fluctuation of venous pressure with respiration may not always be reliable signs to confirm correct placement of the central catheter.

It is suggested that, in these cases, blood is taken for INR estimation immediately and also blood is put into a glass tube to observe for clot formation. If there is no clot within 15 min, then you should consider that the catheter may be in the pleural space. If you are concerned, you should observe the chest drain, in particular looking for a correlation between changes in the rate of drainage and the rate at which the fluid is infused through the catheter. The composition of both should be compared for similarities in dilution, volume, and/or color, particularly if substances like methylene blue are being transfused.

Traditional recommendations suggest placement of a subclavian central venous catheter on the same side as a known pneumothorax/hemothorax to minimize risk of bilateral pneumothorax. I agree, but a central line on the same side as the hemothorax should initially not be used for blood transfusion. This avoids confusion and possible wastage of blood [4]. Injection of methylene blue into the central line will make the diagnosis of a pleural catheter easier.

Despite the above problem, I recommend that you place the central lines on the same side as the hemothorax. However, you need to be aware of the problems that can arise when you do that in these cases.

Lesson

When a central venous line is placed inadvertently in the pleural space, reflux of blood, fluctuation with respiration, and even radiological control can be misleading.

A quick way to confirm a catheter in the pleural space is to take a sample of the patient's blood from the CVP catheter into a glass tube to observe clot formation. If the clot does not form within 15 min, then the catheter may be in the pleural space.

If there is no contraindication to giving methylene blue, this will provide a rapid answer to the concern that the subclavian catheter may be in the pleural cavity.

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Case 36: A Single Abdominal Knife Wound. Easy Case?



A 23-year-old man (175 lbs., 6', ASA 1 E) is admitted to the emergency room (ER) with a single stab wound just below his umbilicus. There are no other injuries as per the emergency physician. You, as the anesthesiologist on call, are asked to come to the ER at 8:00 pm. The patient is conscious, orientated for time and place, and vital signs are stable (HR 86 bpm and BP 120/76 mmHg). He states he has not eaten anything for 6 h but his breath smells of alcohol. Blood is taken for Hct, chemical analysis, and crossmatch for blood. His past history is noncontributory. He has no allergies, and a previous general anesthetic for a right inguinal hernia repair is reportedly uneventful. On examination the patient has one stab wound as described and no other injuries as far as you can see. A 16-gauge IV in his left hand is working well. You listen to the anterior part of his chest and heart to midaxillary line with your stethoscope, but find nothing abnormal. His abdomen is soft.

The surgeon wants to do an emergency laparotomy, and the patient is taken to the operating room at 9 pm. Your anesthetic plan is to use a rapid sequence technique. Prior to induction the blood results become available, and his electrolytes, creatinine, and blood glucose are all within normal limits. His Hct is 36%. The laboratory tells you that two packs of red bloods cells are available. Normal routine monitors are placed, including a 3-lead ECG, a noninvasive blood pressure monitor on his right arm, and an oximeter on his left ring finger (see Case 10). As ordered by the surgeon, you give the patient 1 gram of Keflex slowly (over 2-3 min) IV. A prior dose of Keflex in the ER (given at 7:40 pm) showed no evidence of an allergic reaction. You note the vital signs are still the same as they were in the ER and start to preoxygenate the patient. General anesthesia is induced with etomidate 10 mg and succinvlcholine 100 mg, and an endotracheal tube (ETT) is placed uneventfully in the trachea. You hear bilateral air entry and end tidal CO2 is present. The ETT is taped at 22 cm and the surgery begins. Anesthesia is maintained with fentanyl 250 micrograms and isoflurane 0.8% with 50% oxygen in air. Shortly after the abdominal incision is made, there is a rapid decrease in his systolic blood pressure to 40 mmHg. His heart rate goes to 150 bpm and the saturation falls to below 82%. You ask the surgeon to check for bleeding, but he states that there is no bleeding in the abdomen. He also feels the diaphragm and states that it is intact. By now the end-tidal CO2 is nearly at zero despite the fact that you can hear bilateral air entry over the anterior part of his chest. You tell the surgeon to do a thoracotomy, but the surgeon feels this must be an allergic reaction or an anesthetic mishap. Large doses of epinephrine are given IV with no improvement. Despite heroic attempts on your part and that of your colleagues who come to help, the patient is pronounced dead 30 minutes later.

If the patient did not die from an allergic response to drugs, from an anesthetic mishap, or from an undiagnosed surgical bleed in the abdomen, what was the cause of his demise?

This happened to a colleague of mine, long before TEE became a routine use. At postmortem, a 2 cm long and 2 mm wide stab wound, which had been missed, was found high in the left axilla.

The pericardial sack was full of blood, and there was a large ragged wound in his left ventricle and left atrium.

Discussion

Delayed cardiac tamponade after a penetrating chest injury is a rare complication [1, 2]. Therefore, you must have a high index of suspicion if a patient is admitted with only one stab wound and is also alive. In my experience and that of others, then there is at least one more stab wound somewhere else. My good friend John W. Downing, Professor and Head of Department of Anaesthetics, University of Natal Medical School, Durban, South Africa, warned me of this, and he was proven right again and again. The above case was not his. In my 17 years in Africa, I found that in at least half the cases of a single stab wound where the patient was still alive, another stab wound in the patient's back was present. This I would have missed if I had not sat the patient up. So the lesson in these cases, examine your patients thoroughly. Remember that the neck, shoulder, and chest regions are the most vulnerable to single fatal stab/ slash wounds [3]. Multiple stab/slash wounds often resulted in exsanguination [3].

In my experience from Africa, a patient with one stab wound is usually dead, as the person wielding the knife is an expert in killing people with a one stab. These people do the gruesome deed by an abdominal stab and then twisting and advancing the knife at 90° . With this maneuver they cut potential both the IVC and the aorta.

As an aside, in 1975 we had collected the largest series in the world (over 40 cases) of the anesthetic management of stabbed hearts. We had prospectively successfully anesthetized over 40 cases of stabbed hearts in Durban, South Africa. All patients were stabbed within 30 min to 2 h prior to admission.

The study had taken 4 months. One Friday evening I put all the patients' data sheets into my briefcase to review at home over the weekend. On my way home, I stopped over at the Yacht club and left the briefcase in a locked car. Tragically my briefcase was stolen in the car. All the data sheets were lost. I never found the bag or its contents. Since I had not made any copies of the data sheets and I had no other identifiers as to the patients' name or medical record numbers, we had no results and hence no publication [4].

I learnt a valuable lesson, always make copies of data sheets etc. This is especially true if you are transporting the data from one place to another. Also never give a co-worker any research data sheets before making copies [4].

Lesson

Remember an alive patient with one stab wound has one or more stab wound somewhere else on his or her body. Sitting the patient up and examining every bit of the patient are imperative; otherwise bad things can happen to the patient.

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Case 37: A -Over Vaporizer with a Non-rebreathing Circuit



You are the only anesthesiologist who is part of a medical team to visit an outlying village in Colombia, South America. Unfortunately, all your anesthetic equipment has not arrived, and today is the day for operations on 12 orthopedic patients. You are presented with an Ohmeda Cyprane PAC (portable anesthesia complete) isoflurane draw-over circuit. This is the only available anesthesia delivery system (Fig. 1). The anesthesia nurse, who is a Colombian and works in the hospital, says she had been recommended this equipment by an overseas anesthesiologist some years ago. She has used it with great success both for spontaneously breathing patients and those she had to paralyze. In the latter case, she used an Ambu bag attached to the draw-over circuit. She tells you that she was recommended to use the Penlon Oxford ventilator (POV) when she needs to ventilate the patient instead of the Ambu bag. However, she has not used the above ventilator yet as she not familiar with it. She never saw it in use and is very keen for you to show her how to use the Penlon ventilator. You have never seen this equipment before but ask for any information she may have on this equipment. She hands you a paper [1] describing the use of the draw-over vaporizer and the Penlon Oxford ventilator (the latter is to be placed at F in Fig. 1). You read the text and study the diagram below. The vaporizer D is fitted with a 900 ml corrugated anesthetic tubing (B) with a dust filter at the inlet (A). Oxygen can be given through inlet C. Air is drawn in and through the vaporizer by the patient during inspiration. B acts

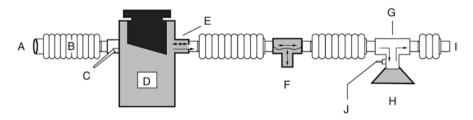


Fig. 1 The Ohmeda Cyprane PAC isoflurane draw-over circuit (Ohmeda, Madison, WI). For explanation see text (From Ali and Brock-Utne [2]. Reprinted with permission from Elsevier)

© Springer International Publishing AG 2017 J.G. Brock-Utne, *Clinical Anesthesia*, https://doi.org/10.1007/978-3-319-71467-7_37 as a reservoir of oxygen between inspirations if oxygen is used. However, in a healthy patient, you may elect not to use oxygen. The outlet E of the PACU vaporizer incorporates a one-way valve that is connected via a second anesthetic tube to a T-connector F. This T-connector is the ventilation port to which an Ambu bag or a ventilator can be attached. From the T-connector right exit, another anesthetic tube leads to a one-way Ruben's valve (G) and the patient's mask (H) or to an endotracheal tube. An exhaust tube (I) carries expired gases from the patient to a scavenging system or into the air. A sidestream adaptor (J) is used for end-tidal CO₂ and/or gas analyzer. Unidirectional flow during ventilation is maintained in the PAC draw-over circuit by the one-way valves at E and G, such that negative pressure at F draws only carrier gas through the vaporizer and into the circuit. Positive pressure applied at F directs the flow only to the patient, as reverse flow is blocked at the E. Back flow of exhaust gas into the circuit is also blocked during all phases of respiration by the Ruben's valve. You also discover that the Penlon Oxford ventilator is fed by a compressed gas source and thus provides only a positive pressure cycle.

You have read all this, but you are still concerned that combining the PAC vaporizer and the Penlon Oxford ventilator may not work as intended. What should bother you about marrying these two items?

In a previous study [2], we showed that the Penlon ventilator did not provide any vapor to a test lung. This is explained by the fact that the Penlon ventilator is only fed by a compressed gas source and thus provides only a positive pressure cycle. Therefore, it is impossible to draw any carrier gas and therefore any vapor from the vaporizer (Fig. 2).



PENLON VENTILATOR AT POSITION "F"

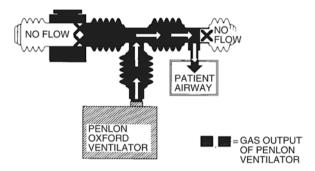


Fig. 2 Penlon ventilator attached at position F. This ventilator provided only a positive pressure cycle and was unable to draw any carrier gas (and, therefore, vapor) through the vaporizer. When this ventilator was attached at position A, ventilation produced excessive airway pressures and vapor concentrations dangerously in excess of the vaporizer settings. Ventilation port A is not recommended by the manufacturer (From Ali and Brock-Utne JG [2]. Reprinted with permission from Elsevier)

Discussion

When this anesthetic circuit is used with a draw-over vaporizer for controlled ventilation, it is essential to use a self-inflating bag or ventilating equipment that can draw the carrier gas through the vaporizer and into the circuit to ensure delivery of the anesthetic gases. A previous article [1] mentioned that this ventilator can be used with a draw-over vaporizer circuit. It is obvious that awareness may occur if the Penlon is used as recommended [1]. Since the Penlon ventilator uses compressed gas, it is not useful in a situation where no compressed gas is available. Hence under these circumstances, manual ventilation with an AMBU bag may be the only means available if the patient needs to be paralyzed.

In our study [2], we also found that attaching an Ambu bag wrongly at position (A) led to the production of a vapor concentration dangerously in excess of the vaporizer settings. In addition, the distal one-way valve (G) became stuck, creating extremely high pressures to the patient airway.

Lesson

When faced with anesthesia delivery systems that you have never used or seen, it is imperative that you be aware of the pitfalls and problems that can occur when using this equipment.

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Case 38: Unexpected Intraoperative "Oozing"



A 64-year-old woman (ASA 3) is scheduled for a craniotomy for clipping of a large aneurysm. Her past history includes hypertension, obesity (130 kg, 5'6", ASA 3), and insulin-dependent diabetes mellitus. She is alert and orientated and moves all limbs. She claims her exercise tolerance is good, meaning that she can walk half-aflight of steps without stopping or getting breathless. The size of the aneurysm worries the surgeon. Therefore, he has requested that a femoral arterial sheath be placed by the neuroradiology team in the femoral artery prior to the surgical incision. The reason for this is that, should clipping of the aneurysm prove to be impossible or dangerous, coiling of the aneurysm can then be attempted via the femoral artery. You anesthetize her in a routine manner, and the anesthetic proceeds according to plan with stable vital signs. The interventional radiologist cannulates the femoral artery. After that he slowly infuses the femoral sheath with a heparinized solution (500 ml normal saline with 2000 units of heparin) under pressure via a pressure bag (Infusable Pressure Infusor, Vital Signs, Totowa, NJ 07512, USA). The solution drips via a 60 drops/ml Piggyback Microdrip with a Clair clamp controlling the rate (one to two drops every minute) (LifeShield, Hospira Inc., Lake Forest, IL 60045, USA). There is no transducer system attached. The patient is turned 180 degrees from the anesthetic machine and the surgery begins. After 90 minutes the aneurysm is exposed. The surgeon complains that there is a lot of oozing in the surgical site and that he has difficulty maintaining adequate hemostasis. He asks you if the preoperative coagulation was normal. You answer that it was and tell him that the liver enzymes, etc. were also within normal limits. He says: "Please give her some fresh frozen plasma (FFP) and tell me when you have given it." You are at a loss to understand the cause of the oozing but order the FFP ASAP. You send off new coagulation studies (INR, PT, and PTT) and an arterial blood gas (ABG). You also place 5 ml of her blood in a glass tube. The results from the ABG, including electrolytes and blood sugar, come back within normal limits. However, the blood in the glass tube is still liquid even after 15 min. Five minutes later, the FFP arrives and you infuse it quickly. The surgeon reports no improvement. You tell him that you are still waiting for the coagulation results from the lab. You are at a loss to understand what is going on and the surgeon is now really unhappy.

What do you think could be the cause of the oozing and what will you do?

You check the bag with the heparinized solution that is being infused into the femoral artery. You discover to your dismay that the 500 ml bag it is totally empty. You immediately do an ACT and discover that it is prolonged. You inform the surgeon and give protamine IV which quickly reverses the effect of the 2000 units of heparin that had inadvertently been infused. The clipping of the aneurysm is concluded successfully, and the patient makes an uneventful recovery. This case is similar to a previously reported case [1].

Discussion

Coagulopathy and severe bleeding are associated with high mortality [2].

This case highlights the importance of keeping a watchful eye on all healthcare personnel performing procedures on anesthetized patients under your care. In this case, the interventional radiologist did not use a transducer system to keep the femoral artery patent. Instead he relied on a simple IV line with a stopcock controlling a pressurized heparinized saline bag. When we discovered that the cause of the intraoperative oozing was due to the excess heparinized saline solution given through the femoral sheath, we closed it and replaced their IV set with our own transducing system.

In our case report [1], we did object that the neuroradiology team did not use a transducer system. That was a big mistake. We now routinely hand the interventional radiologist our transducer system when they insert a femoral sheath.

Laboratory coagulation studies in the operating room and sending blood to the laboratory does take time. If you are concerned and have no thromboelastography (EG) or rotational thromboelastometry (ROTEM) machine readily available and/or people knowledgeable to use them, then place some blood in a plain glass or plastic tube. You will know within 10–15 min that you have a problem. This test is very useful during obstetric emergencies and trauma where DIC can develop. The result from this simple tube test is usually available up to an hour before the central laboratory coagulation studies are available. If there is no coagulation in your tube after 10–15 min, then you can start the DIC treatment much earlier.

Lesson

Three points are worth noting about this case:

1. Keep a watchful eye on all healthcare personnel working on your anesthetized patients. After all this is your patient.

- 2. Understand the limitations and potential problems of equipment and/or techniques that other healthcare providers use.
- 3. The tube test mentioned above can be a lifesaver.

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Case 39: Central Venous Access and the Obese Patient



You are scheduled to an esthetize a 54-year-old 160 kg, 183 cm tall (BMI = 47.8 kg/m^2 , ASA 3) man with vasospasm for cerebral angiography. He had been admitted to the ICU 3 days earlier, following an emergency craniotomy for a subarachnoid hemorrhage. When you meet him in the radiology suite, he responds sluggishly to verbal commands but reacts to pain. His heart rate is regular at 66 beats per minute and the blood pressure is 160/90. The neurosurgeon is happy with that pressure. An endotracheal tube had been removed 12 h previously. He is breathing spontaneously on an oxygen mask, providing 2 liters of oxygen per min. His vital signs are within normal limits. His only IV access is a triple lumen right subclavian venous catheter (16 cm long) (Arrowgard Blue Plus Multi-lumen CVC, Arrow International, Inc. Reading, PA 19605). This had been inserted at the time of the original operation. Through the proximal lumen of this catheter IV, propofol 100 mcg/kg/min is being infused for sedation. Via the medial lumen, neo-synephrine 200 mcg/kg/min is given for pressure support. Maintenance fluid is given through the distal port. You review the previous anesthesia team's note in their anesthesia record. They report no problem with airway management. Despite that, you make sure you have a fiber-optic cart, bougie, etc. available. You check the anesthetic machine and make sure your anesthesia equipment and all your anesthesia drugs are ready. You call for a colleague to come and give cricoid pressure. He arrives and you look set to start the anesthetic.

However, is there something else that you should do prior to induction of general anesthesia?

You should always check the patency of any IV you are using prior to induction of a general anesthetic. This is especially true when you using someone else's IV. We had a case like this recently [1]. In that case we could aspirate blood from the distal end of the triple lumen catheter but not from the other ones. Since we were in the radiology suite, contrast medium was injected in all three lumens under direct fluoroscopic control. The only lumen that was noted to be intravascular was the distal lumen. Propofol (proximal lumen) and neo-synephrine (medial lumen) were both seen to extravasate into the subcutaneous tissue of the neck. Rather than pulling this catheter out, we placed, under fluoroscopic control, a long guide wire into the right atrium through the distal lumen. The triple lumen catheter was then removed, and a double lumen catheter (20 cm long), also made by Arrowgard, was inserted over the guide wire. After that we used fluoroscopy with dye injection, to confirm later that both lumens were in the vein. Anesthesia was induced uneventfully and the patient was taken back to the ICU in a stable condition.

Discussion

The triple lumen catheter, which was originally placed to its hub, was 16 cm long. The patient had a large neck (>70 cm in circumference), and due to the distance between his skin and the vein, only the distal lumen was intraluminal. The replacement catheter was 20 cm long, and the additional length was needed to allow both lumens intravenous access. We could have used the 20 cm long triple lumen Arrowgard, but it was not available at the time.

Since peripheral intravenous (IV) access may be limited in obese patients [2], cannulation of a central vein (subclavian, internal jugular) is often recommended for IV access [3]. A similar problem as the one described here has been reported with a much shorter 10.8 cm pulmonary artery catheter introducer in an obese patient [4].

Of interest was the fact that the blood pressure in the right arm was very different from the left. The systolic blood pressure in the right arm was 20 mm Hg higher than in the left arm. When checked 24 h later, the blood pressure in the two arms was equal. This temporary difference is attributed to the subcutaneous infusion of neo-synephrine.

Lesson

It should always be your routine to ascertain the patency of any IV prior to induction of general anesthesia. This is especially true for lines that have not been inserted yourself. In the case of central lines, I would say checking is mandatory. When using

any multi-lumen catheter, all lumens should be checked to confirm patency following initial placement. Remember that in morbidly obese patients, catheter position can change with movement [4], so intravascular position must be confirmed periodically in these patients.

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Case 40: Check Your Facts



You are just out of residency and have joined your first anesthesia practice group. This evening you are on fourth call. There is nothing for you to do, so you are told to leave. Just then an emergency laparotomy is booked. The patient is a morbidly obese patient (BMI 34) with an acute abdomen. She is 4 days out following a gastric bypass procedure. You offer to help and the first call accepts your offer. The patient arrives within 5–10 min in the operating room. She complains of severe abdominal pain. Her respiratory rate is over 40 and shallow. Her heart rate is 130 sinus tachycardia, with a blood pressure of 100/60. She has a cold periphery and her oxygen saturation is 91% on room air. The patient is placed on the operating table with a ramp as previously described [1] to improve your view on laryngoscopy. Preoxygenation and a rapid sequence induction commence. You provide cricoid pressure. The anesthesia induction drugs are given by your colleague. It appears to you that your colleague is using fentanyl, etomidate, and rocuronium, in that order, for anesthetic induction. You see no fasciculation. Your colleague performs the laryngoscopy and tells you that he has a grade 1 view. The endotracheal tube is seen to pass between the cords and bilateral air entry is heard. An obstructive end-tidal CO₂ pattern is seen. For general anesthesia maintenance, isoflurane is added to the oxygen. At this point, your colleague is called urgently to another room where there is a code blue. You say you will stay and look after this case. He leaves in a hurry. You place the patient on the ventilator and the vital signs, including the respiratory parameters, are stable. However, the heart rate is still 130 sinus tachycardia. You start getting ready to place an internal jugular vein line, but the oxygen saturation is seen to fall to 88% from 98%. The airway pressure has risen from 40 to 56 cm H_2O . You start to ventilate the patient manually and notice that it is very difficult to do so. It is now 5-6 min since the anesthetic induction. The airway pressures continue to be high but the saturation is improving. You listen to the chest and confirm bilateral, equal air entry. You pass a suction catheter down the whole length of the endotracheal tube without any problem. Sucking on the catheter yields no secretion. Since the oxygen saturation is improving, you place the patient back on the ventilator. Unfortunately, the peak pressures are still high (58 cm H_2O), and the oxygenation saturation starts to fall again. The patient is not moving and the pupils are pinpoint. You are at a loss as to what to do next. You can feel the pressure mounting in the room, as everyone is looking at you and wondering what is the "new boy" going to do? So what will you do?

You look at the syringes that are still stuck in the line attached to three-way stopcocks. To your surprise you see the syringe that you thought was rocuronium is actually labeled succinylcholine. You draw up some rocuronium and give 80 mg. Within 2 min the ventilator airway pressures fall and the oxygenation improves.

Discussion

When assisting a colleague with a case, it is imperative to know what he or she is doing and what drugs are being administered.

This is also important when you are taking over a case that has begun. In those cases, you must also assess the anesthesia machine, like checking that there is emergency oxygen, the vaporizers are full, the suction is working, etc. This is because you have not had the opportunity to check the anesthesia machine prior to the case. I have seen several near misses with this scenario where the primary anesthesia provider had not checked the machine and bad things did happen.

Lesson

When you do not know what is going on, always check your facts. In this case, information as to what drugs had been given proved vital for safe patient care.

If you had known it was succinylcholine, then you would have given a nondepolarizing muscle relaxant, and this problem would never have occurred.

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Case 41: Intraoperative Epidural Catheter Malfunction



A 62-year-old obese male (wt, 120 kg; ht., 190 cm; body mass index, 33.2 kg/m²), ASA 2, is scheduled for a colectomy secondary to ulcerative colitis. You plan a combined general anesthetic technique with an epidural. When the patient is in the operating room, you place an epidural catheter (20 G closed tip) (B. Braun Medical Inc., Bethlehem, PA 18018, USA) uneventfully in the L4–L5 interspace. The length of the epidural catheter in the epidural space is 4 cm. As is your practice, the epidural catheter, as it leaves the patient's back, is led in a semicircle over a folded 2×2 inch swab. This is then covered with an OpSite (Flexigrid, Smith and Nephew, Hull HU3 2BN, England). The catheter is attached to the patient's back and over his right shoulder using Compound Benzoin Tincture USP (The Clinipad Corporation, Guilford, CT 06437, USA) and tape (Hytape Surgical Produce Corporation, Yonkers, NY 10704, USA). There is a negative test to 3 ml of lidocaine 2% with epinephrine 1; 200,000. A total of 22 ml of lidocaine 2% is then injected and a block to T-4 is achieved. The patient is placed in a supine position and routine general anesthesia commenced. Further two additional boluses are injected into the epidural space with good effect and no problem. Forty minutes after the last dose, the surgeon requests that the patient be placed in a steep head-down position. Shortly thereafter, it becomes impossible to inject anything through the epidural catheter, despite the use of a small syringe. You suggest that the surgeon puts the table back in the original position, but he is unwilling to do so at that time. You are reluctant to abandon your epidural.

Is there anything else you could do to get the epidural to function adequately again?

We have previously described two such cases in obese patients [1]. The solution was to get another colleague to place both his hands underneath the patient's lumbar and thoracic area and pulled the catheter, tape, and the subcutaneous tissue toward the head. The other person, at the same time, attempted to inject lidocaine into the epidural space with a syringe. Immediately after the catheter/tape was pulled cephalad, the plunger descended into the syringe. This indicated that the catheter was functioning again.

The patient remained pain-free for the next 18 h. At 18 h the catheter had stopped working. We attempted again to do the traction maneuver, but this was unsuccessful. The epidural catheter was removed. On examination of the nonfunctioning epidural catheter, we discovered that a 4 mm section of the catheter was severely narrowed. The narrow section was 22 cm from the tip. Attempts to inject saline through this catheter, after its removal from the epidural space, were unsuccessful. It is possible that our last attempted forceful traction, applied to tape and tubing, caused this severe narrowing of the catheter. Hence the traction maneuver should not be done with excessive force.

Discussion

This is another example of an anesthetic complication that can occur in obese patients that are under your care. An intraoperative epidural malfunction is a real problem for patient care, since a change in an anesthetic plan must be made if the epidural cannot be made functional again. We speculated that, in our case, the steep head-down position caused the catheter to be kinked or stretched so that it temporarily occluded the lumen.

A total obstruction of an epidural catheter has been reported in the postoperative period [2]. In that case [2], the catheter became severely stretched and permanently obstructed when the patient was moved from the operating room table to the trolley. It was made functional again by cutting the catheter distal to the stretched portion and reattaching the catheter connection. The stretching of an epidural catheter leading to an obstruction has also been reported on the withdrawing the Tuohy needle over the catheter [3]. To prevent this from occurring, the needle should be withdrawn slowly and in line with the catheter. An epidural catheter can also become kinked at the point of entry into the supraspinous ligament, if the insertion of epidural is done with the patient in a very flexed position. When the patient then straightens out, the catheter gets obstructed [4]. An epidural catheter can also be knotted in the epidural space [5]. This probably occurs when the catheter forms a loop in the epidural space and then doubles back on itself and forms a knot. These authors recommend that the catheter should not be introduced more than 2.5 cm into epidural space. However, the majority of anesthesiologists leave the epidural catheter at least 3 cm in the space [6].

Manual stretching (pulling the catheter with both hands) of a Braun epidural catheter shows that an enormous force must be produced before damage to the catheter is seen [1].

Other causes of total intraluminal obstruction of an epidural catheter have been reported as a manufacturing defect [7], a knotting of the catheter [8, 9], or when connector assembly is not placed correctly with the epidural catheter [10]. This misplacing can lead to a clamping of the catheter and therefore an inability to inject anything through the catheter.

Lesson

It may be prudent, in obese patients, to initially tape the epidural catheter laterally to the mid-auxiliary line and thereafter in front of the arm to the shoulder region. In this way, the movement of the subcutaneous tissue of the back is less likely to kink, stretch, or bend the catheter. In the recovery room, the catheter can be re-taped.

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Case 42: Breathing Difficulties After an ECT



You are scheduled to do an outpatient electroconvulsive therapy (ECT) on a 36-yearold female. This is her fourth ECT in a series of at least 8. She tells you that the previous ones have been uneventful and that her mood is better. She is 90 kg and 5'6'' in height. She works in her husband's restaurant where she is a cook. Her medical history is significant for depression, hypertension, insulin-dependent diabetes mellitus, and hypothyroidism. She takes nifedipine, enalapril, insulin, thyroxine, paroxetine, and fluphenazine. After she arrives in the treatment room, an IV is started. You examine the patient and find nothing abnormal, including her airway exam. Her chest is clear to auscultation. Her heart rate is 90, BP is 130/70, respiratory rate is 12, and room air oxygen saturation is 97%. You place noninvasive monitors on the patient and start to preoxygenate her. Anesthesia is induced with etomidate 16 mg, followed by succinvlcholine 1 mg/kg. The patient is hyperventilated, and the ECT procedure is done uneventfully, except excess salivation is noted after the treatment. The patient awakens from the anesthetic but suddenly sits up and starts to cough violently. Her saturation falls to 86%. You diagnose laryngospasm and provide positive pressure mask ventilation with 100% oxygen. After a few minutes, the laryngospasm is broken and she is now breathing easier. She sits up, and although her oxygen saturation is now 96% on nasal oxygen (6 liters/min), she complains that she does not get enough air. The nurse tells you now that this difficulty with breathing happened last time she had an ECT, but she slowly improved over a 20-min period and was discharged home. The nurse tells the patient to lie down, but she refuses. She again states that she cannot breathe. She rips off all her monitors and refuses to have them replaced. The nurse tells you she did that last time too.

Should you be concerned? If, yes what will you do?

If you are concerned, then stay concerned.

In this case, with all the monitors off, you decide to examine the patient's chest and cardiovascular system. This case is similar to one we have previously described [1]. Actually this was the first reported case of negative pressure pulmonary edema (NPPE) following ECT. In our case we found that the patient had bilateral crepitation over both lung fields. A chest x-ray showed moderate pulmonary edema and a diagnosis of NPPE was made. The patient was given furosemide, 20 mg. One hour later, there was a rapid clinical improvement. She was admitted for observation and discharged home the next day. The patient found the complication frightening and elected not to return for further ECTs.

Discussion

It was Oswalt et al. [2] who first described this complication. The pathophysiology of NPPE has been outlined, but the exact mechanism has not yet been defined [3]. Two conditions must be present for NPPE to be produced: inspiratory air obstruction and strong spontaneous inspiratory efforts. Is it noteworthy that NPPE can develop in a matter of seconds? The main differential diagnosis is pulmonary aspiration mimicking the signs of NPPE (i.e., hypoxia and decreased compliance). However, the presence of these signs, after correction of airway obstruction in a spontaneously breathing patient, merits the presumptive diagnosis of NPPE. If frothy, pink fluid appears, the diagnosis is almost certainly correct. In our patient, the clinical examination, the chest x-ray, and the rapid resolution of her symptoms with treatment helped confirm the diagnosis.

In our case [1], we postulated that excess secretion, most likely due to a large dose of succinylcholine, resulted in laryngospasm followed by pulmonary edema. There is really no need to give more than 0.5–0.7 mg/kg of succinylcholine to prevent the breaking of bones during the ECT. The routine uses of glycopyrrolate or atropine might have prevented the excess secretion. However, anticholinergic medications may exacerbate the tachycardia associated with ECT. Bear in mind that when I started anesthesia, atropine was routinely given prior to ECT because of the concern of a short-lived bradycardia/cardiac stand-still often seen following ECT.

There is no way of clinically predicting who will develop NPPE. However, it is more likely to occur in strong healthy adults [4]. Since our report, two other reports of NPPE following ECT have been published [5, 6].

Lesson

If you are worried about your patient, stay worried. Even though you are told this postoperative concern/problem has happened before and there is nothing to worry about.

In these cases, *always* examine your patient and make your own assessment. This is, after all, your patient. Although NPPE is uncommon following ECT, being aware of this complication allows for early recognition and prompt treatment.

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Case 43: White "Clumps" in the Blood Sample from an Arterial Line



You are scheduled to anesthetize a 67-year-old woman for a lower abdominal resection of a rectal cancer. This is your first day in this hospital. The patient has a past history of coronary artery disease, hypertension, obesity, and insulin-dependent diabetes mellitus. A previous anesthetic 6 months before for a laparoscopic cholecystectomy was, according to the patient, uneventful. You have no previous anesthetic record to refer to. She proves to be a difficult IV stick, but you manage to put a 20 G IV in the back of her hand. You sedate her and take her to the operating room. Prior to induction of anesthesia, you place an arterial line in the patient's right radial artery. The anesthesia technician hands you a pressure transducer containing 1000 U heparin in 500 ml of normal saline. Thereafter you put the patient sitting up and place an epidural for postoperative pain relief. The test dose is negative and again the patient is placed supine. Anesthesia is induced uneventfully. Since the patient has very poor IV access, you elect to put in a central line. A right internal jugular catheter is placed uneventfully and seen to work well. Anesthesia is induced in a routine manner with etomidate, sufentanil, vecuronium, and isoflurane in air. The surgery starts and you elect to send an arterial blood sample to the laboratory for estimation of the usual parameters, including blood sugar. To your dismay, you note that white "clumps" precipitate out of the arterial blood sample. You take a new sample but unfortunately with the same result. However, blood from the central line does not show any "clumps," and you are wondering if you should be worried about this and if so why?

This is an example of heparin-induced thrombocytopenia (HIT), also known as heparin-induced thrombocytopenia thrombosis syndrome (HITTS) [1, 2]. Heparin is now recognized as the leading cause of drug-induced thrombocytopenia in the perioperative period [2]. Platelet aggregation or clumping is the primary mechanism, leading to the development of life-threatening thrombotic events in cerebral and myocardial circulation. It is also known as the "white clot syndrome" [2].

In the case mentioned above, the outcome was disastrous. The HIT panel came back positive. The platelet count dropped from 153,000 to 23,000, and the patient suffered a stroke on the first day postoperatively, from which she never recovered. This was thought to be due to the severe cerebral thrombosis, as spontaneous intracranial bleeding is considered rare. We attempted to publish this case in 2002 but were told by various editors that there was nothing new in our submission. At that time, I would contend that HIT and its serious complications were not well known.

Discussion

It is important to remember that the HIT reaction can occur in patients who have previously received heparin without any problems, just like latex allergy [3]. Patients with a previous diagnosis of HIT/HITTS should have no heparin or heparin analogs under any circumstance. These include heparin-coated catheters, Lovenox or other low-molecular-weight heparins, heparin flushes of catheters, and subcutaneous heparin. The following drugs should be used as alternatives if anticoagulation are required:

- Bivalirudin (Angiomax). This is given 0.15–0.20 mg/kg/h without an initial bolus. It is broken down enzymatically and excreted via the kidney. Its half-life is 25 min. A reduction in the infusion dose of bivalirudin should be considered in patients with moderate or severe renal impairment. Coagulation times return to baseline approximately 1 h following cessation of bivalirudin administration.
- Argatroban (Novastan outside of USA). The dose is 2 mcg/kg/min without an initial bolus. The drug is excreted by the liver and has a half-life of 40–50 min. It is interesting to note that the drug increases the INR so one must aim for a higher therapeutic range.
- 3. Danaparoid sodium (Orgaran). This drug was withdrawn by Organon International for use in the USA in 2002. It is available in several other countries. There have been rare cases of crossover reactivity with anti-heparin antibodies and possible asthma exacerbation.
- 4. Lepirudin (Refludan): recombinant hirudin. The drug is given as a bolus 0.4 mg/kg and the infusion is 0.15 mg/kg/h. The drug is excreted by the kidney and the half-life is 80 min with normal kidney function. In kidney failure the half-life rises considerably. Bayer ceased the production of lepirudin in 2012.

Lesson

So should heparin be removed as a drug to prolong vascular patency? In a study by Tuncali et al. [4], they found no statistical significant difference in pressure waveform dampening and arterial occlusion after catheter de-cannulation. However, they did identify the risk of vascular occlusion. This could be correlated with the presence of a hematoma at the puncture site, the duration of cannulation, and the age of the patient. They concluded that the use of heparin flush solution did not improve the arterial catheter patency in the perioperative period. Until definitive evidence is available, the routine use of heparin flush solution in vascular access should be considered carefully both in adults as well as children [1]. However, a recent meta-analysis [5] found that heparin with saline was not superior to normal saline in reducing central venous lines occlusions. In the short term, the authors found that the use of heparin saline is slightly superior to normal saline for flushing catheters from a statistical point of view. In 2010 Stanford hospital no longer uses heparin in their arterial or central line flush.

Lesson

- 1. Proper positioning of the arm, nursing care of the catheter, and continuous normal saline flush under pressure may prove to be adequate to maintain patency, especially in high-risk patients [1].
- 2. In a case where you suspect the patient is developing HIT, you immediately do the following:
 - (a) Stop all heparin and heparin-containing products, including low-molecularweight heparin and heparinoids.
 - (b) Send a blood sample to the laboratory asking for HIT tests. These are the heparin-induced platelet aggregation assay (HIPAA) and the ELISA test for heparin_PF4 antibodies. It is important to note that if the first test comes back negative, you carry on treating the patient if he/she has HIT and send another test. If that is also negative, then carry on and monitor for possible delayed HIT for at least 24 h. The ELISA test can give a 15% false-negative rate, but if the platelets count is dramatically down from a normal baseline, then the patient should be treated as if he/she has HIT.
 - (c) If anticoagulation is required, then use alternatives to the heparin mentioned above. Warfarin (Coumadin) must never be used unless a direct thrombin inhibitor is given first to maintain anticoagulation. Warfarin alone can precipitate a massive thrombosis due to depletion of protein C and S [2].
 - (d) If possible, avoid platelet transfusions. There is a higher risk for these patients to develop thrombosis than to start bleeding. This is seen even in the face of significant thrombocytopenia. Platelet transfusion can lead to significant thrombosis in these cases [3].
 - (e) Early intervention with a heparin substitute may minimize the permanent damage from thrombosis.

(f) The patient must be told that he or she has a heparin allergy. A wristband and a sign over the bed should warn everyone of this drug allergy. Lastly, this allergy must be recorded in the patient's medical record.

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Case 44: Anesthesia for a Surgeon Who Has Previously Lost His Privileges



You have just completed residency and have joined an anesthesia group that has many surgical locations to cover. Today you are assigned to a free-standing surgical clinic. This is your first time in this clinic. Your first case is a 42-year-old male who is a drug abuser and positive for HIV. According to the surgical history and physical exam, he is otherwise healthy. He is scheduled for a cervical discectomy under MAC (monitored anesthesia care). Upon arrival at the clinic, you introduce yourself to the head nurse. She tells you that the surgeon always does these cases under MAC and has refused in the past to have them done under general anesthesia. You look at the operating room list and discover that this is (fortunately) the only case he is doing today. You arrive in the change room, and one of your anesthesia colleagues tells you that the surgeon you are working with today has lost his privileges in many hospitals and clinics in the area. He also lost his privileges in the one you are in now but has recently been reinstated. This is his first time back after a 12-month absence. You ask why he lost his privileges in this clinic, and you are told that he punctured the trachea in one patient during a cervical discectomy under MAC. This led, in the postoperative period, to severe surgical emphysema of the neck with obstruction of the airway. Even though he was aware that the patient had breathing difficulty due to surgical emphysema in the postoperative period, he did nothing and left the hospital. The patient stopped breathing 30 min later. Without the timely intervention by an anesthesiologist, who was passing by in the recovery room, the patient would have died. Adding to your concern is the fact that you are told by your anesthesia colleague that the surgeon told the patient to sue the anesthesiologist as it was his fault. You are now really apprehensive and wonder what you should do.

What would you do?

Solution/Discussion

There is no simple solution to this problem. There are three options:

- 1. You should remind the surgeon that you have the right to provide an anesthetic technique that you think is the safest for this patient. You have no doubt that this case must be done under general anesthesia, with an endotracheal tube. Should the surgeon refuse your anesthetic plan then you can refuse to provide the anesthetic. If he agrees to a general anesthetic and you do it but are concerned at the end of the case that the surgeon may have traumatized the trachea, then you could remove the ETT over a tube exchanger or bougie [1]. Should your patient develop respiratory complications that necessitate rapid reintubation, then you can use the tube exchanger or the bougie. Plans must then be made to transport the patient to a nearby hospital with an ICU.
- 2. You can delay/cancel the case by stating that abnormal cardiac and/or chest exams require a preoperative ECG with or without a cardiac consult, chest X-ray, etc. This to give you time confirm that the surgeon has indeed had his privileges restored. One would hope that the administration of any hospital or clinic would check before they allow him to schedule a case. In addition, if you reside in California, you can ask for the surgeon's medical record from the California Board of Medical Quality Assurance. If he has lost his privileges, for what you consider is well below the standard of care, then you can approach the administrators of the surgical clinic with your anesthesia colleagues to discuss your concerns.
- 3. In most academic centers, you have the right to refuse to work with a specific surgeon, just as he has the right to refuse to work with you. However, if you do refuse, then one of your colleagues will be asked to do it and I doubt they will thank you. In private practice the situation can be very different. In the latter case, your anesthetic group may have a contract with a hospital/surgical clinic to provide anesthesia care. Hence you may not be in a situation to refuse to do the case, as it could be seen as a break in contract. However, if you decide to do the anesthetic, then it must be as you want it. No surgeon should dictate to you what anesthetic the patient should have, nor should we dictate to them how their surgery should be done.

Lesson

Always do what is best for the patients, even if this means you do not follow the surgeon's preference for the type of anesthetic. In a case such as the one above, if you manage to cancel the case, then you can check if the surgeon has indeed lost his privileges and has been reinstated. Information as to why he lost his privileges can prove to be vital to you and your anesthesia group's future association with this surgeon and/or the clinic that supports him.

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Case 45: Airway Obstruction in an Anesthetized Prone Patient



Today you are anesthetizing a 58-year-old man (82 kg and 5'11'') with a cerebellar tumor. He is otherwise healthy and classified as an ASA 2. General anesthesia is induced uneventfully, and the patient is turned prone after having been placed in a Mayfield pin holder. The patient's head is 180 degrees away from the anesthesia machine. His neck is flexed so that there is a one finger gap between the mentum of the mandible and the sternal notch. You would prefer to have two finger gap. A BiteGard (Hudson RCI, Teleflex Medical, Research Triangle Park, NC 27709) has been placed between the upper and lower molar on the right side. The operation proceeds uneventfully for 6 h, when there is an increase in the peak inspired pressure from 24 cm H20 to 42 cm H2O over a 2-4-min period. All the other parameters are unchanged. You call for help and one of your colleagues comes to your aid. With his help you confirm bilateral air entry with no adventitious sounds. You inspect the endotracheal tube at the mouth and confirm that it has not moved and is still taped at 22 cm H20. Your colleague places the patient on 100% oxygen, and after a few minutes you attempt to pass a suction catheter through the endotracheal tube (ETT), but it only goes in 15–20 cm. You manipulate the ETT, but there is no improvement. Your colleague suggests you let the endotracheal cuff down as the cuff could have herniated into the lumen. You do so, but you are still unable to pass the suction catheter all the way through the ETT. You diagnose a partial kink in the ETT. The vital signs are still within normal limits but the peak airway pressure has gone to 48 cm H2O. You are concerned. At the moment you can ventilate the patient, but should the kink be total, this could have serious consequences for the patient. There are 30-45 min left, and the surgeon is unhappy to reposition the patient's neck before the end of the surgery. Nor is he happy about your request to turn the patient supine and reintubate the trachea. You get an LMA ready in case you are unable to ventilate the patient.

What else could you do to improve the ventilation without extending the neck or turning the patient supine?

Replace the angle connector at the end of ETT with a Bronchoscopic Swivel Elbow Adaptor (PriMedico, Largo, FL) [see Case 12 for a picture of the Bronchoscopic Swivel Elbow]. Through the bronchoscopic port (at the top end of the connector), you advance a gum elastic bougie or semirigid Sheridan Jet Ventilation Catheter/tracheal tube exchanger (Rusch Inc., Duluth, GA 30096) [1] or a Cook's exchange catheter (Cook Critical Care, Ellettsville, IN). With any of these three devices, you should be able to straighten the kink. With the two latter devices, you can ventilate, using the orifice in the middle of the Sheridan Jet Ventilation Catheter or the Cook's exchange catheter.

Discussion

Sudden airway obstruction in a prone patient can end in disaster. The above solution can be lifesaving.

Many years ago I had an episode where an ETT fell out onto the floor from the mouth of an anesthetized prone patient. Luckily the head was in a neutral position, and I managed to intubate the trachea by placing myself on the floor under the head of the patient. With the laryngoscope in the right hand, I found it very easy to secure the airway with an ETT in my left hand. However, you could also have had the laryngoscope in your left hand, and advance the ETT with your right arm by crossing over your left arm.

Lesson

An obstructed airway in the prone patient is fortunately rare, but the suggested solutions may prove to be lifesaving in these critical situations. Having a Bronchoscopic Swivel Elbow Adaptor may prove to be invaluable.

Reference

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Case 46: A Question You Should Always Ask



You are just out of residency. Today you are scheduled to do a laparoscopic tubal ligation. The patient is a 28-year-old diabetic with an insulin pump placed. She has had a baby boy 2 weeks earlier and wishes now to have her fallopian tubes tied under an epidural anesthetic. She prefers not to be asleep.

She is healthy and weighs 260 lbs. and is 5'6". You meet the patient for the first time in the preoperative area and discover to your dismay that the insulin pump is on. She has been fasting since the night before. You check the blood sugar and find it is 25 mg%. By now you have a working IV and you are infusing 50 ml of 50% dextrose. An epidural for her delivery had been a great success and she wonders if she can have the same again. You explain to her that because the surgeon will be distending her peritoneal cavity with gas and tilting her head down, it will be difficult for her to breath. She will also get a "referred pain" to her shoulder which could be very painful for her. The surgeon does not want to do a local with some sedation, and he leaves you with two options either a GA or an epidural/spinal.

You elect to do an epidural and the surgical procedure goes uneventfully and the patient is very grateful. How did you get away with doing an epidural in this case?

This situation happened to me when working in private practice. The surgeon, when asked how long he is going to be, responded: "Less than 15 min." Actually the operation took only 13 min. From skin to skin. However, at 10 min, while in full Trendelenburg, the patient complained of severe bilateral shoulder pain. She had no pain from the abdomen incision nor from the peritoneal distension. Fortunately, the operation was finished and the patient placed supine, the abdominal incision closed, and the shoulder pain subsided over a 2-min period. She was discharged home after about 2 h in the postoperative recovery area.

Discussion

It is always important, especially when you do not know the surgeon, to enquire how long he or she will be. The way I recommend you deal with this matter is to say: "Where I trained, the surgeons were known to take over an hour (or whatever) to do this case." So when the surgeon responds and says he will be 15 min, you get valuable information but also make the surgeon feel good.

Lesson

If you do not know your surgeon, it is imperative to find out how long he/she thinks they will be.

Case 47: Postoperative Vocal Cord Paralysis



You are scheduled to anesthetize a 65-year-old woman (ASA 2) for a left carotid endarterectomy (CEA). She has hypertension, which is well controlled medically. Her past surgical history is significant for a thyroidectomy 17 years previously. Otherwise she is well, with no known allergies. Her physical exam is normal and she has no stridor or hoarseness. She prefers to be awake for the surgery, and you do a deep and superficial cervical plexus block with 1.5% lidocaine with 1:200,000 epinephrine. Six ml is injected at C3, C4, and C5, while 14 ml is injected into the superficial plexus. The surgery starts with the patient awake, as she has only received midazolam 2 mg and 50 ug of fentanyl for the block. After a Pilling retractor is inserted, pushing the internal jugular vein and sternocleidomastoid laterally and thyroid and trachea medially, the patient coughs repeatedly. The surgeon injects 6 ml of plain lidocaine 1% around the common carotid artery, but this does not help as the patient now develops stridor and becomes agitated. The retractor is removed and within a few minutes she feels much better. The surgeon inserts the retractor again, but once again the patient develops severe coughing and stridor. The patient is told that a general anesthesia is needed and she reluctantly agrees. She is anesthetized without any problems with fentanyl 200 ug, etomidate 18 mg, and vecuronium 7 mg. She is easy to mask ventilate. You have a grade 1 view of interest is the fact that your resident notes that the right vocal cord seems to less away from the midline than the left. You have a look and agree with his assessment. An endotracheal tube (ETT) is placed uneventfully. The anesthetic is maintained with oxygennitrous-isoflurane. The surgery is completed uneventfully and a special note is made by the surgeon that the vagus nerve is intact. Both a nonrecurrent laryngeal nerve and a recurrent laryngeal nerve are not seen.

At the end of the surgery, you are concerned about the vocal cord mobility and hence a bit hesitant to remove the ETT. But the surgeon does not share your concern and suggest you remove the ETT from the trachea.

What do you think? What should your "modus operandi" be in this case?

To assess the vocal cord mobility, the patient is allowed to breath spontaneously on sevoflurane after the effect of vecuronium is reversed by glycopyrrolate and neostigmine or sugammadex. With the patient deeply anesthetized and breathing spontaneously on sevoflurane, the ETT is removed. The patient is breathing unaided with minimal evidence of airway obstruction. Immediately thereafter a #3 laryngeal mask airway (LMA) is inserted, and a fiber-optic bronchoscopic (FOB) is inserted through the LMA. In a similar case to the one described above [1], bilateral vocal cord adduction (vocal cord paralysis) with moderate supraglottic edema was seen. An ear-nose-throat (ENT) surgeon recommended that a tracheostomy be done, especially in view of the supraglottic edema. Serial FOB, performed on days 2 and 5 after the tracheostomy, revealed bilateral vocal cord paralysis. On the ninth day, movement of the left vocal cord was noted. The tracheostomy was removed 2 weeks after the surgery and the patient made an uneventful recovery.

Discussion

Airway obstruction following CEA can occur from several causes, including tissue edema, nerve injury, and neck hematoma [2].

In this case [1], the authors postulate that the right recurrent laryngeal nerve was damaged during the thyroidectomy done many years previously. This led to paralysis of the right vocal cord. As long as the left vocal cord was working, the patient had no complaints, and the physicians were unaware of this problem. However, when during this surgery the left vocal cord was temporarily paralyzed, the effect was acute bilateral vocal cord adduction and acute stridor.

Thyroid surgery is reported to lead to permanent and/or transient palsy of the vocal cords in 1-2% of patients [3], while the incidence of unilateral vocal cord paralysis under CEA is reported to be up to 6% [4]. In a previous case [5], acute respiratory distress was caused by a combination of deep cervical plexus block and a preexisting unknown asymptomatic contralateral recurrent laryngeal nerve paralysis.

Lesson

A patient scheduled for a CEA, after a thyroidectomy or any neck surgery, should have a preoperative vocal cord examination. The use of only a superficial cervical plexus block may lower the risk of respiratory complication in cases like this.

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Case 48: This Is a Serious Problem



You are a new attending anesthesiologist at a university hospital. It is late in the evening and you are on second call. A craniotomy has been ongoing for 4 h and there is 1 h to go. Your resident (32-year-old married male), although competent, does not seem particularly interested in what you are trying to teach with him. You have worked with him before but at that time he had seemed much more interested and willing to learn. Today he also complains about being cold, although you feel warm. He is wearing a long-sleeved gown. He has been to the rest room at least three times in this period. He now wants to go again. You ask him if there is anything wrong, but he states that he is fine but needs to go to the bathroom. He is in the rest room when your colleague, who is first call, comes into the OR to send you home. You tell him about the case and remark to the first call attending that the resident seems to have TB (tiny bladder). He tells you that this has been his impression also. No more comments are made and you leave the OR. As you get out into the corridor, a nurse calls you urgently to say that there is a person lying unconscious in the hallway outside the men's rest room. You run to the scene. The collapsed person is your resident. He is cyanotic and not breathing. With a firm jaw trust he starts to breath, albeit slowly. You call for an oxygen cylinder and give 100% oxygen via a face mask. With the help of your nurse you attach an oxygen saturation monitor and BP cuff and establish that his vital signs are within normal limits but he still unconscious. You pinch his arm and he pulls the arm away. You are very concerned and call for more help. Aided by the nurse, you quickly establish an IV with Lactate Ringer. You give him an ampoule of 50% glucose as you think he may be hypoglycemic, but there is no improvement in his consciousness level.

What will you do?

This happened to me. When help came, another colleague discovered that his pupils were pinpoint. The resident was taken to the emergency room where a "tox screen" was positive for narcotics. The next day he was in the chairman's office and then sent onto a treatment center. After treatment was completed, he was permitted back to the department to continue his residency. However, after another episode with drug abuse, he decided to change specialties. As far as I am aware, he is still alive.

Discussion

The definition of drug addiction, as defined by the American Society of Anesthesiologist, is:

The overwhelming compulsion to use drugs in spite of adverse consequences. It is a chronic, progressive disease that results in loss of control of one's life. Unless it is recognized and treated skillfully, addiction will result in disability and will often end with death. Physical dependence frequently develops but is not present in all drug addictions.

Although the exact rate of substance abuse among physicians is unknown, conservative estimates are that 8-12% of physicians will develop a substance abuse problem at some point during their career [1]. Drug abuse has been shown to be a major risk factor for medical malpractice and negligence lawsuits [2], the development of physical and psychological illnesses, and adverse effect on his or her family [3]. This does take into account the harm that can be done to the patient and to the profession as a whole. It is a fact that physicians with substance abuse problems often remain undetected for many years before any intervention is made and then it may be too late [4]. Left untreated, the mortality rate among drug-abusing physicians has been reported to be as high as 17% [5]. Anesthesiologists are thought to be especially at risk [6], but this has been questioned [1].

Unfortunately, most anesthesia care providers do not know how to identify or help an impaired colleague [7].

I have known five anesthesiologists who have died, after given themselves a drug overdose. Three died in the hospital while on duty and two died at home using propofol and/or thiopental. Of these five, two had been known to use anesthesia drugs to an excess in the past, but despite that they were allowed to practice anesthesia. Another six (including the case above) were discovered before they killed themselves and they were sent to treatment centers. Of these six, three were residents who managed to complete the anesthesia residency. I have lost contact with these latter six. But I do know that one had a relapse while in private practice but is doing fine now.

Lesson

The American Society of Anesthesiologist has produced two very good pamphlets entitled "Chemical Dependence Guidelines for Departments of Anesthesiology" and "Chemical Dependence in Anesthesiology." In the latter booklet is a list of what to look for to try and identify a potential drug-abusing physician.

Among these are:

- 1. Charting which becomes increasingly sloppy and unreadable.
- 2. Preference to work alone.
- 3. Unusual behavior changes, i.e., mood swings with periods of depression followed by euphoria.
- 4. Difficult to find between cases, as he takes short naps.
- 5. Requests frequent bathroom breaks.
- 6. Wears long-sleeved gowns to hide needle sticks.
- 7. Often states he is cold and hence wears long-sleeved gowns.
- 8. Pinpoint pupils.
- 9. Patients anesthetized by a drug abuser may complain of pain which is out of proportion to the amount of narcotic charted on the anesthetic record.

It is essential the anesthesiologist becomes familiar with presenting signs and symptoms of substance abuse in their fellow colleagues. In this way the morbidity and mortality associated with the abuse of drugs can hopefully be reduced [7], not to mention the safety of the patients in their care.

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Case 49: A Leaking Endotracheal Tube in a Prone Patient



You are to anesthetize a 30-year-old man (200 lb) who is donating his bone marrow to an unknown person. He is classified as an ASA 1 with a class 1 airway. The patient has had no previous anesthesia/surgery, and his family history is negative for anesthesia-related complications. He takes no medication and has no drug allergy. An IV is placed in his left hand, and midazolam (2 mg) is given in the preoperative holding area with good effect. He is anesthetized in a routine manner and mask ventilation is uneventful. After the airway is secured with an endotracheal tube, the patient is turned prone. His head is turned to the side so that you can see both eyes. Everything is progressing as planned until 30 min before the end, when you notice that there is a progressive cuff leak in the ETT. With 10 min to go, you are eventually unable to blow up the cuff. At this point, the patient's vital signs remain stable, but the oxygen saturation has decreased from 100% to 96%. The peak pressure has fallen from 36 cm H₂O to 22 cm H₂O.

You inform the surgeon and suggest that you change the ETT either with a tube changer or by turning the patient supine. The surgeon tells you that he has only a few more minutes to go. But you have worked with him before and you know that 10 min could be 30 min.

Is there anything else you could suggest, so as to stop the leak in the cuff and thereby ventilate the patient adequately?

The problem can easily be rectified by packing the throat with a 2-in. moist vaginal pack [1]. This was done and the surgery was completed uneventfully.

Discussion

The use of a vaginal pack can be extremely helpful in these cases. They are especially useful in cases where the cuff develops a leak after a successful but difficult endotracheal intubation. Your reluctance to remove a correctly placed ETT in these circumstances is understandable. Changing the ETT over a tube changer could be dangerous. Packing the throat and thereby sealing the leak may prove to be the safest move when facing a similar situation. I have used it many times with great success.

The use of these packs to "buy time" in the emergency room can prove lifesaving. The pack can then be used to stabilize the patient's vital signs before you change a leaking ETT. I have used this technique in the emergency room with great success. A leaking cuff in the ER occurs with regular monotony. When you as an anesthesiologist arrive in the ER to help change a leaking ETT, then you are invariably handed a laryngoscope and an ETT. The ETT you are given is usually bloody. Furthermore, this cuff too is most likely damaged by the patient's teeth from previous attempts at laryngoscopy. I always select my own ETT and check it, but there are times when you have no such luxury and a rapid endotracheal intubation is called for.

Lesson

The throat packs come in two sizes: 1" or 2". Moisten the pack in water and squeeze the water out. Tie a single knot in what is to be the distal end of the pack. This is done to tell you when the pack has been completely removed. Since it is seldom that you place a throat pack, I make it a rule to always write "PACK" on a piece of tape and place it on the patient's forehead or ETT. In this way I hope to prevent leaving a pack behind when I remove the ETT.

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Case 50: An Impossible Situation?



You are stationed in a cottage/mission hospital in Africa as the anesthesiologist. You are called to anesthetize a 35-year-old healthy male who has been stabbed in the neck with a wooden knife. You run to the emergency room and find the patient in a stable condition. He is awake and cooperative. The wooden knife's handle is visible sticking out of his neck with the wooden blade disappearing under the clavicle directed toward the heart. He is otherwise healthy with no previous surgeries or allergies to drugs. The surgeon wants to remove the knife under general anesthesia. You send off blood for crossmatch and place two large bore IVs. The patient is taken to the operating room. After 4 units of blood are made available in the operating room, you anesthetize him with a rapid sequence technique. He tolerates the anesthetic induction well and his vital signs remain unchanged. The surgeon cleans the neck and pulls out the wooden knife. There is sudden tachycardia with a dramatic drop in blood pressure. Severe bleeding is seen from the neck wound. The surgeon now does a sternotomy and discovers to everyone's dismay that there is a 0.5-1 cm hole in the aortic arch between the innominate and the left common carotid arteries. Your training dictates that the patient be placed immediately on cardiopulmonary bypass, but you know there is no such thing available.

What do you think the surgeon can do to save this patient's life?

This case happened to a friend of mine in Empangeni, Zululand, South Africa, in 1974. The surgeon clamped the aortic arch proximal to the hole but in such a way that blood was still flowing through the innominate artery (Fig. 1). He started to repair the hole while at the same time instructing my anesthetic colleague, Dr. Michael Grant, to look for dilatation of the pupils. Michael told him when they started to dilate and then the clamp was let off for a few seconds. This process was completed several times until the hole was repaired. The importance of pupillary dilatation can be argued in this case. I have seen pupils dilate dramatically after severe intraoperative bleed, only to return to normal size with adequate resuscitation. Barbiturates could have been used in this case if the patient's blood pressure would have tolerated it.

The patient was taken to the ICU for postoperative ventilation and that is when I met him. By lunchtime I had removed his ETT. He made an uneventful recovery.

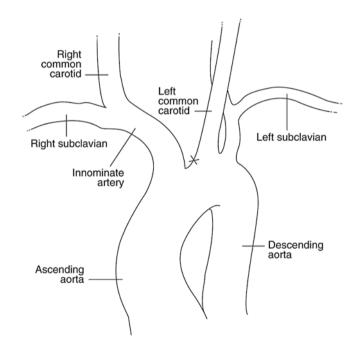


Fig. 1 "X" marks the entry wound of the wooden knife

Discussion

The surgeon was Mr. Rex Henderson FRCS of Empangeni, Natal, South Africa, one of the very best surgeons I have ever had to pleasure to work with. This fellow could do anything. I remember vividly, after he had successfully repaired a severe eye injury, we were called to the scene of an accident where a man was trapped under his tractor. He had lost a lot of blood from a crushed leg and other soft tissue injuries. We were able to place a subclavian IV into the patient while he was upside down. It took some time before we got him to the hospital, but Rex saved the man's leg by repairing the damaged popliteal artery. Later that night we operated on a 1.5-year-old child that had fallen off a train and needed a hind-quarter operation. Rex knew more about pediatric anesthesia than I will never know.

The above patients were saved by a great surgeon and by the presence of a blood bank. But what do you do when you do not have a blood bank? The following story illustrates a remarkable way to solve this problem. During a plenum session in the World Congress of Anesthesiologist in Sidney, Australia, a friend of mine, Brother John from the Australian Franciscan Order, was talking to a large audience about rural anesthesia and trauma. At the time he was the only doctor working in a large rural area of Borneo. He joked by telling us that he was "the one-armed bandit" (being both the surgeon and the anesthesiologist). When asked what he did when the patients needed blood and he had no blood bank, he said: "That is easy, I am the blood bank, I am 'O' negative." The response of the large audience was total silence – you could hear a pin drop.

Lesson

Unusual circumstances require unusual methods.

Case 51: An "Old Trick" but a Potential Serious Problem



You have anesthetized an 18-month-old child with a facial deformity which requires surgical correction. He is otherwise healthy and is classified as an ASA 1. Unfortunately, there is more blood loss than anticipated, and the blood pressure is beginning to drop to nearly unacceptable levels. You call for blood and start infusing albumin 5% (250 ml glass bottle), but it is going very slowly through your 22 G IV in the hand. By aspirating 20 ml albumin from the albumin bottle via an in-line stopcock, in the IV administration set, you are able to inject the albumin more rapidly into the patient. But this is time-consuming, minimizes your ability to observe the vital signs, and can potentially damage the vein leading to extravasation.

Besides telling the surgeon to stop the surgery until you can get better IV access, what else would you suggest to get the albumin quicker into the patient?

The disposable intravenous administration set for the albumin has an air vent, which has a one-way valve. With the bottle upside down (rubber stopper facing down), air can be forced into the bottle via this valve from a 20 ml syringe. This results in a faster flow of albumin.

Discussion

However, there is a downside to this technique, as air embolism can occur if one is not observant. When the albumin bottle is empty, then one must immediately close the giving set with the IV administration set clamp. If you do not do this, the patient could get a lethal air embolism. In cases where the albumin administration set has no air vent, a needle with a three-way stock-cock and a syringe can produce the same effect.

Before the present soft blood bags were used, blood was given from a 500 ml glass bottle. The bottle was hung upside down, and a non-vented giving set was inserted through the rubber stop at the neck of the bottle. Normally blood would flow for a little while until the pressure above the blood surface in the bottle became negative. To overcome this problem, all glass blood bottles had a small glass tube inside the bottle. This tube acted as a vent and went from the neck of the bottle to nearly the top of the bottle. Hence, when the blood bottle was turned upside down, there was an air connection from the outside to the inside of the bottle above the surface of the blood. In this way, when blood was leaving the bottle, it was replaced with air. Without this small glass tube, blood would not flow out of the bottle.

Therefore, the "old" way of rapidly infusing blood from the blood bottle was to apply positive pressure, using a sphygmomanometer pump [1]. For those of you who have watched the film and TV series MASH (Mobile Army Service Hospital) from the Korean War, you will have seen this technique being used.

Unfortunately, I have seen this technique leading to a death on an operating table. The anesthesiologist was not paying attention to the fact that the blood bottle was empty of blood, but not of pressurized air, most likely a liter, was therefore rapidly infused into the circulation with the result that the patient died. It was the anesthesiologist's first day on the job. Next day he resigned and left the specialty.

I have also seen a blood bottle which was full of blood being pressurized with air as described above. The blood bottle exploded, sending pieces of glass and blood all over the operating room. Luckily no harm was done.

Fortunately, blood bottles were replaced in the 1980's with the polyvinyl chloride bags which we now use. Thus the incidence of air embolism from the above cause has been dramatically reduced.

The disadvantage of the "in-line syringe technique" is that the vein can be damaged by the intermittent pressure exerted on the vein. It can also be messy and time-consuming. In addition, if you are by yourself, you may not be able to monitor the vital signs as often as you would like.

It is important to realize that air embolism can occur from plastic bags too [2]. In this case, air was seen in a half-filled 6% Hetastarch bag (Abbott Laboratories, North Chicago, IL) but ignored when the bag was pressurized. This led to an air embolism in a child with an episode of cardiovascular instability, but no mortality.

Recently venous air embolism has been reported to occur in athletes receiving pressurized IV fluids [3].

Lesson

Pressurizing with air, especially an IV glass bottle, can lead to venous air embolism after the blood or fluid has been infused. If you use this technique, you better be vigilant.

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Case 52: A Loud "Pop" Intraoperatively and Now You Cannot Ventilate



Today you are to anesthetize a 58-year-old man, ASA 2, for an anterior resection of a rectal cancer. He is otherwise healthy but is a big man of 150 kg and 5'8". His airway is classified as 2. You bring him to the operating room and place him on a "ramp" for easier endotracheal intubation [1]. Noninvasive monitors are placed and you anesthetize him in a routine manner. He is easy to ventilate after being given succinylcholine. However, it is a grade 3 view. You place a gum elastic bougie blindly in the trachea and try initially a #9 endotracheal tube (ETT), but it will not go, neither will a #8 ETT. Eventually you settle for a #7 ETT. You make a mental note that his trachea must be big since you need to put a much larger than normal air volume into the ETT cuff to prevent a leak. With the airway now secured, the patient is put in a steep Trendelenburg position and the surgery starts. His arms are placed out at 90° from the body, and an upper body Bair Hugger blanket is placed. This is the variety of Bair Hugger that has sticky sides to the see-through face cover. The operation proceeds uneventfully, until a large "pop" is heard and the bellows of your Narkomed 2B, North American Drager, descend and stay down. You attempt manual ventilation by using the anesthesia machine's collapsible breathing bag. This is unsuccessful. No air fills the bag, even though you use the oxygen flush control button and close off of the "pop of valve." You ascertain that the anesthesia hoses are intact and in the correct position. You decide that the loud pop was the bursting of the ETT cuff. You immediately replace the "burst" ETT over a gum elastic bougie with a new one and blow up the cuff. However, there is no improvement. The oxygen saturation is now 76% and the surgeon has stopped working and is looking at you. You have no Ambu bag in the room and you call for one. While waiting, you mouth ventilate through the ETT. It is then that you see the problem. What is it?

One of the anesthesia breathing hoses coming into the wye at the patient's mouth looked like it was in place, but in actual fact it was not. It was barely kept in place with the sticky tape of the Bair Hugger. There was therefore a large leak. When this was fixed, the ventilator worked again satisfactorily. The case was completed uneventfully.

Discussion

When suddenly during an anesthetic you cannot ventilate either with the ventilator or manual ventilation, despite the fact that you have oxygen pipeline pressure and gas in the rotameter, then there is only one way to deal with this matter. If you do not have oxygen, then the situation is different [2].

If you have ascertained that you have oxygen coming out of your anesthesia machine, then disconnect the ETT from the anesthetic tubing at the wye and do a pressure test. In this way you can ascertain if it is the circuit or the ETT that is at fault. It is unlikely that both will fail at the same time. If this had been done in the above case, you would have discovered that the leak was in the circuit and not in the ETT. It is so easy to jump to conclusions. Never do that. In these cases always do the pressure test. It will save you a lot of heartache.

This happened to me, so I speak with experience.

Lesson

There is only one modus operandi when this happens to you. You must pressurize the anesthetic circuit. In this way you can easily find out if it is the ETT or the anesthetic breathing circuit that is the cause of your inability to ventilate.

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Case 53: Postoperative Median Nerve Injury



You get a call from your orthopedic surgical colleague to say that the patient that you anesthetized, for an 11-h spine surgery operation yesterday, has developed a left median nerve conduction block. The diagnosis has been confirmed by a neurologist who is at a loss to find any reason for this. There is no evidence of infection, hematoma, or vascular insufficiency of the hand. The patient has no other problems and is otherwise happy with her surgery. You tell your colleague that you will get back to him. In reviewing your anesthetic record, you see that the anesthetic was uneventful with stable vital signs throughout. Your IV access included two large bore IVs in the right forearm and back of the right hand, respectively. A right subclavian triple lumen catheter had also been inserted. In addition, she had a left radial intra-artery catheter. The arms were both placed forward alongside the head. The elbows were bent <90°.

You go and see the patient and apologize for what has happened. You ask to see her hand. There is nothing abnormal noted with the overlying skin of the left hand and wrist. Nor do you see any scars from any surgeries on her whole limb. Scar tissue can cause excess pressure on nerves while a patient is undergoing surgery [1, 2]. The blood pressure cuff is on the right arm. Nor is there any evidence of a left shoulder dislocation [3]. The oxygen saturation attached to the left ring finger reads 100%.

You are also at a loss to explain why this has occurred and tell the patient that you have not seen anything like this before. Therefore, you inform the patient that you believe the median nerve conduction block should go away hopefully within a few days.

Can you think of a reason why this has happened to his particular patient?

Wrist hyperextension for arterial line placement and stabilization is likely to result in profound impairment of median nerve function, although transient [4].

Discussion

It is common practice to supinate and hyperextend the wrist during insertion of a radial intra-arterial catheter to facilitate arterial puncture and cannulation. Many anesthesiologists leave the wrist in this position for the duration of the surgical procedure or as long as the catheter is in place. The reason being that the arterial line should not be accidently dislodged in this position. Unfortunately, median nerve conduction block can occur with hyperextension although transient [4].

Postoperative median nerve injury has, in some cases, been ascribed to direct needle trauma associated with intravenous catheters inserted at the wrist [2]. The mechanism of position-related median nerve injury remains undefined but may be multifactorial [1, 5]. Factors mentioned are obesity. In all of Silverstein's four cases, all patients had a BMI of 34 and greater [3]. Other factors are peripheral neuropathy, diabetes mellitus, chronic kidney disease, alcoholism, and HIV [3]. In cases where wrist hyperextension is prolonged, it is possible that a stretch-induced focal neuropathy may be the cause [6].

Lesson

To minimize or prevent median nerve problems in the postoperative period, it is advisable to return the wrist to a neutral position following arterial line placement.

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Case 54: A Patient in a Halo



A 73-year-old (89Kg 5'8") female is admitted, following an MVA, for surgical stabilization of an unstable cervical neck. You find her in the preoperative holding area where she is wearing a halo (PMT model 1233, DePuy Spine, The Bremner, Jacksonville, Florida, USA) (Fig. 1). The halo is a vest that covers her upper body and shoulders. The halo is attached to the head on both sides of the skull with adjustable headlocks. These can be adjusted in three ways, A/P positioning, flexion/extension, and traction/distraction. You manage to examine her chest with your stethoscope, although this is not easy due to the vest that is covering her back and front of the chest. You are comforted that the chest x-ray taken 1 h ago is normal. She has no other health problems. She tells you that she underwent an elective cholecystectomy 6 months previously under general anesthesia without any problems.



Fig. 1 PMT® Corporation's (Chanhassen, MN) Halo System, model 1233 (Image courtesy of PMT Corporation)

© Springer International Publishing AG 2017 J.G. Brock-Utne, *Clinical Anesthesia*, https://doi.org/10.1007/978-3-319-71467-7_54 She can open her mouth and you can clearly see her uvula. She is very nervous and does not want a fiber-optic endotracheal intubation. You say you will put her to sleep and secure the airway after she is asleep. She is very grateful. You induce anesthesia in a routine manner and find that you can easily place a #7 endotracheal tube (ETT) in the trachea with a Macintosh #3 blade. You have a grade 1 view. The operation concludes satisfactorily within 2 h and the patient wakes up still in the halo. As she follows commands and is breathing adequately, you remove the ETT from the trachea. You take her to the recovery room, where initially her vital signs are within normal limits. You are next door in the preoperative holding area talking to the next patient, when a nurse tells you to come quickly to the recovery room as your halo case is not breathing and her oxygen saturation is 87% and falling. You run to see the patient. A nurse is attempting to mask ventilate the patient with an adequate size Guedel airway, with no success. You ask for and get a Macintosh #3 blade and insert the blade into the mouth of the now semi-unconscious woman, but to your dismay you see nothing. You ascertain that this is indeed a Macintosh #3 blade and try again, but again you see nothing. You secure the airway by blindly passing a bougie into the trachea. You are at a loss to understand why, within 2.5 h, with no evidence of airway edema, you now can see nothing during laryngoscopy while previously you had a grade 1 view.

What do you think the problem is?

Unbeknown to you, the surgeon adjusted the headblocks forward so that the anatomy of the airway changed. This adjustment resulted in you having no view of the larynx.

Discussion

A patient with a preoperative halo represents a potential anesthetic problem, as the above case, which happened to a great friend of mine, can attest to. Should this happen to you and the patient is allowed to wake up with the halo, then you must verify that the halo has not been adjusted; if so then you may not be able to reintubate the patient should you require to. Being aware of this problem is imperative for the patient's safety. It may be prudent to leave a bougie in place when you remove the ETT from the trachea, especially if there is a possibility that the surgeon may have moved the halo during the surgery even if it is ever so slightly.

Lesson

Be aware that a surgeon may not tell you that he has adjusted the halo position. The result of this adjustment may be that a patient with a grade 1 airway during induction of anesthesia has a very different and potentially difficult airway after surgery.

Case 55: It Is Now or Never



You have started a new job as an anesthesiologist in private practice. Your first case on your first day seems a simple one. The patient is a 27-year-old male (weight 90 Kg and height 5'8", ASA 1) scheduled for elective arthroscopy of the shoulder, knee, and ankle, all on the left side. He is an unmarried construction worker accompanied by his father. He is a motorbike fanatic and has fallen of his bike in the past. He claims he is perfectly healthy except that in the last 6 months he has developed a hoarseness of his voice. He tells you that it has not got worse. His father nods in agreement. He went to an ENT surgeon 3 months ago, who told him that he has vocal cord polyps. The ENT surgeon indicated that there was no problem. However, you do not agree and you speak to the orthopedic surgeon to relay your concern. You suggest that a new ENT consult be obtained and that he be cancelled today. The surgeon informs you that it is "now or never" for this surgery as the patient's insurance expires tomorrow. You consider regional blocks, but the patient refuses them and states he wants to be asleep. He would rather leave without the surgery than having it done under regional blocks.

If you decided to go ahead with a general anesthetic, how would you do it for this patient, remembering that he has to be on his right side down for the shoulder surgery?

The best solution is to cancel the case. However, this happened to me on my first day in private practice and this is what I did. Since the patient wanted the surgery, I warned him and his father that going ahead with a general anesthetic could result in a tracheotomy, and/or postoperative ventilation in an ICU for maybe a day or two, or even death. I wrote that in the chart and had them sign my note. In a court of law, I don't know if it would have made much difference, but it made me feel better. In the preoperative holding area, I gave him atropine 0.5 mg IV, to reduce salivary secretions, together with 6 mg of midazolam IV. He was taken to the OR and noninvasive monitors were placed. After a smooth IV induction with propofol, a #3 LMA was inserted. He was positioned on the OR table with his right side down. Ventilation was manually assisted throughout the shoulder surgery. When the surgery was completed, the patient was placed on his back and spontaneous ventilation resumed. At the completion of the surgery, the LMA was discharged home the same day.

Discussion

I felt this was a difficult case to manage. Others may not agree. However, I believe strongly that the correct thing to do was to cancel; however when "forced" to do the case, regional blocks would have been a good option. But again, an overdose of local anesthetic, with respiratory/cardiac arrest, could have proven disastrous.

I elected not to place an endotracheal tube (ETT), since that could have made the polyps bleed, etc. Using a fiber-optic scope, to look at the vocal cords prior to anesthesia, would not be of any use either, as in a court of law the question would be: "How many vocal cord polyps have you seen, etc.?"

Lesson

Always do what is best for the patient and don't take risks. In this case a risk was taken, but I was fortunate that the outcome was a good one for the patient.

Case 56: General Anesthesia in a Patient with Daily Use of Prescribed Amphetamine



You are assigned to anesthetize a 59-year-old female for a total hip replacement. Her past history is significant for depression, and she has taken dextroamphetamine 15 mg bd. for over 3 years.

Would you proceed or would you recommend discontinuing amphetamine therapy prior to general anesthesia and thereby delay the surgery?

We have recently reported nine cases of patients who daily took prescribed amphetamines and underwent an uneventful general anesthetic [1, 2].

The conclusions [1, 2] suggest potential stable anesthesia management in patients on chronic prescription amphetamines. However, it is recommended that directacting vasopressors, including phenylephrine or epinephrine, should be readily available intraoperatively.

In the nine cases above, none required any vasopressor drugs.

Discussion

Amphetamines are non-catecholamine, sympathetic amines with powerful central nervous system (CNS) stimulation activity. Their action is thought to be associated with the local release of biogenic amines such as norepinephrine from nerve terminal storage sites [3]. Peripheral actions from amphetamine include an increase of systolic and diastolic blood pressure and a weak bronchodilator and respiratory stimulant action. Chronic amphetamine exposure with stimulation of the adrenergic and peripheral nerve terminals causes a depletion of catecholamine receptor storage [4]. This reduction in catecholamine, especially norepinephrine, is thought to contribute to a blunted physiologic and sympathetic response to hypotension, as has been reported in anesthesia [5, 6]. Hence intraoperative refractory hypotension with or without bradycardia in patients taking amphetamines should be treated with direct-acting vasopressors, such as epinephrine (IV 50–100 microgram) or phenyl-ephrine (IV 50–100 microgram). Ephedrine has been reported to have a decreased or absent pressor response after chronic amphetamine use [6].

A single case report in 1979 [5] of cardiac arrest and death during a cesarean delivery in a chronic amphetamine abuser has become the reference in the anesthetic literature to warn against the use of general anesthesia in these cases. It is interesting to note that the authors [5] admit that it was difficult to prove the association of the patient's drug use and the general anesthesia, as there were other concurrent clinical factors that could have contributed to her demise. Unfortunately, the case report has been incorporated in many anesthesia textbooks and thereby has contributed to the belief that amphetamine must and should be discontinued prior to general anesthesia and surgery, to avoid patient morbidity and mortality.

In our two reports [1, 2] dealing with nine elective surgical patients, in which the prescribed amphetamine was not stopped preoperatively, the general anesthesia proceeded successfully with no cardiovascular instability in any of the patients. No dramatic fluctuations in arterial blood pressures were observed, at induction or during the course of the surgeries, which necessitated the use of pressor treatment.

A recent study in pediatric patients diagnosed with addition deficit hyperactivity disorder (ADHD) found no evidence to suggest that continuing preoperative stimu-

lant medication should be stopped preoperatively [7]. The study showed no cardio-vascular instability in this pediatric population.

Lesson

We believe that patients on chronic amphetamines may not need to discontinue the drug prior to elective surgery but that direct-acting vasopressors should be readily available intraoperatively.

References

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- Cartabuke RS, Tobias JD, Rice J, Tumin D. Hemodynamic profile and behavioral characteristics during induction of anesthesia in pediatric patients with attention deficit hyperactivity disorder. Paediatr Anaesth. 2017;27:417–24.

Case 57: What Is Wrong with This Picture?



You are sent to investigate an increased incidence of difficult endotracheal intubations in a mission hospital in Africa. On the first morning you are there, you observe an anesthesiologist giving a general anesthetic to a patient for an elective cesarean section. A rapid sequence induction with etomidate and succinylcholine is followed by the insertion of a Macintosh blade #3 inserted into the mouth using the left hand. What follows then is contra to everything you have ever seen before. The anesthesiologist, instead of placing the endotracheal tube (ETT) to the right of the blade and into the trachea, places his right hand over the left hand and guides the ETT successfully into the trachea. You are amazed, but after careful inspection of the blade, you understand the reason for this unorthodox way of placing the ETT in the trachea.

Without seeing the blade, what will you say the problem could be?

A RIGHT-handed laryngoscope blade for a RIGHT-handed anesthesiologist (Fig. 1).

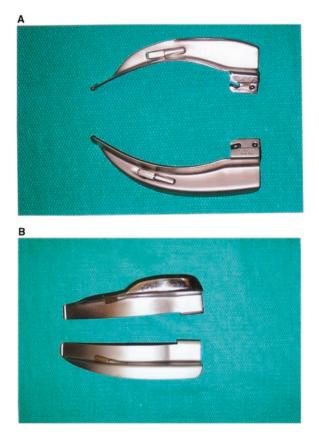


Fig. 1 (a, b) The RIGHT-handed laryngoscopy blade is on the bottom

Discussion

This happened to a friend of mine Dr. Mannie Mankowitz in the 1970s. He was never tired of telling the story. The interesting part of the story was that this was the only blade they had in the hospital. It had been donated by a RIGHT-handed anesthesiologist who no longer worked there.

The normal or conventional laryngoscope is held in the left hand and has the flange on the left as one looks down the blade. The flange is used to sweep the tongue to the left and allows one to use the right hand to intubate. The right-handed laryngoscope is held in the right hand. The scope is the mirror image of the conventional one. When using this blade, the ETT is held in the left hand.

After the first edition of this book in 2008 came out, an old friend of mine, Dr. Rajend Maharaj, from King Edward VIII Hospital days (1977–1985) informed me that I had written LEFT-handed laryngoscope when I obviously meant RIGHT. Thank you Rajend, for putting me right.

Lesson

Be aware that there are RIGHT-handed laryngoscopes. They can appear when you least expect it.

Case 58: The One-Eyed Patient



A 78-year-old female is scheduled for a right upper lobectomy under general anesthesia. She is 54 kg and 5 feet 10 inches. Her weight has been steady for as long as she can remember. Her past history is significant for hypertension, a 50-year smoking history and an enucleation of the right eye for tumor 10 years previously. She is unsure what the tumor was. She has a glass eye but prefers to wear a large black (pirate) eye patch that covers the whole orbit including the eyebrows. Otherwise her medical history and physical exam are unremarkable. She has a class 2 airway.

After sedation with midazolam 1 mg IV, she is taken to the operating room where a routine general anesthetic, with fentanyl, thiopental, and vecuronium, is induced uneventfully. Mask ventilation with sevoflurane in 100% oxygen proves to be difficult, as there is a large leak detected around her face. A larger and well-inflated face mask is placed over her mouth and nose, but a large leak is still present. By increasing the fresh gas flow to 12 liters, the patient is easy to ventilate, but the leak is still present and everyone in the room can smell the sevoflurane. You again perform a pressure leak test on the anesthetic absorber circuit (you did it before you started), but you find nothing wrong. Another anesthesiologist is brought in to hold the mask with both hands while you elect to manually ventilate the patient with the reservoir bag. Despite this, the leak is still there. Her vital signs remain stable, but you are concerned as this unexplained leak is something you have not encountered before.

What will you do now?

Remove the eye patch and in this case, you see the orbital cavity without a glass eye. The reason for the leak is now quite apparent as there is an anatomical passage from the mouth cavity to the orbit of the eye. Having discovered the cause of the leak, tracheal intubation with double lumen tube is done atraumatically on the first attempt with the patient fully paralyzed. The operation proceeds uneventfully and she is discharged home after 4 days in hospital.

Discussion

This is the only case like this I have seen [1]. At the time we were at complete loss as to the cause of the large leak. If the patient had been bigger, with or without lung pathology, mask oxygenation of the patient could have been a real problem.

A possible solution could have been to use a moist 4-inch vaginal pack to pack the orbit and in so doing hopefully minimize the leak.

Lesson

Be aware of the patient with an eye patch. Should it be difficult to mask ventilate due to a large orbital leak, then a moist vaginal pack can be used.

Reference

1. Brock-Utne JG. Beware of the one eyed patient. Anesth Analg. 2007;104:1615.

Case 59: A Near Tragedy



After a long week in the operating room, you are relaxing on a beach south of Durban in South Africa. The weather is wonderful. Just behind the beach is a lagoon. When the tide comes in, the Indian Ocean enters the lagoon at the end of the beach where there are many rocks.

Suddenly there is a cry for help, and you rush to the rocks to see a 6-year-old boy in the water to his armpits with the tide coming in. He is screaming that he cannot get his foot out. Two men are both busy trying to release his foot. It will not come out. Over 200 people are now gathered around. You introduce yourself, offer help as an anesthesiologist to the two men, and discover that one is an orthopedic surgeon and the other a cardiac surgeon. They say they may need your help as in less than 10–15 min the water will be over the boy's head, and in that case, they will need to amputate the foot.

You run to your car. In the booth you have all the anesthetic drugs and equipment (including a draw over vaporizer) you will need to anesthetize the child. Since you have no IV, you consider sedating the child with intramuscular injections of any of the anesthetic drugs you have in your doctor bag. The other option is to do an inhalational induction using the isoflurane draw-over vaporizer.

What do you suggest?

I administered ketamine 6 mg/kg intramuscularly to the child. This provided sufficient pain relief for the surgeons to remove the foot intact while I looked after his airway. With the patient breathing spontaneously and with no sign of pain, the foot came out intact but badly bruised and bleeding. Happily, the child made an uneventful recovery.

Discussion

This happened to me on Easter in 1980. The beach was South Broom, 2-h drive south of Durban, Natal. An unknown orthopedic surgeon and the thoracic surgeon Dr. Bruce Henderson (no relation of Rex Henderson of Empangeni, see Case 50) and I were left with this dilemma.

Lesson

1. When living or traveling in faraway places, always carry adequate medical equipment with you if you feel that you would like to render medical help. If you want to assist, you will never know when and what you will need.

Shortly after arriving in South Africa, I was called to help a neighbor who had collapsed. I came with my normal GP bag which had no laryngoscope or endotracheal tubes, face masks, or Ambu bag. I found myself hopelessly unprepared. If I had been able to control his airway properly, I might have saved his life. As it was, the patient died. It was very distressing. Shortly after that, I invested in a Laerdal (Stavanger, Norway) bag (with laryngoscopes, endotracheal tubes, IVs, etc.). I always carried it in the trunk of my car and used it at least nine to ten times in a period of 17 years. The chest drain which I had was used only once.

- 2. Based on my experience, my recommendation is that you do not want to be caught out like a "carpenter without his tools."
- 3. Lastly always think outside the box and ketamine in this case was a "foot saver."

Case 60: Robot-Assisted Surgery: A Word of Caution



Today you are anesthetizing a 3-month-old 6.1 kg infant with biliary atresia. The operation is scheduled for laparoscopic Kasai using a robot-assisted surgical system. The system consists of a remote-operating console and a wide-base surgical cart. Inhaled anesthesia with sevoflurane, peripheral IV access, securing the airway with an endotracheal tube and a radial line inserted is performed in the OR. The endotracheal tube position is confirmed by auscultation, and a precordial stethoscope is placed over the patient's left chest. An orogastric tube is used to decompress the stomach. The patient is elevated about 4 inches off the OR table on blankets and egg crate to allow the greatest range of motion for the robotic arms. The OR table is positioned in a 30° reverse Trendelenburg to facilitate surgical exposure. The operation proceeds with placement of the robotic cart and arms.

Thirty minutes into the case, there is a gradual drop in blood pressure. There is no obvious bleeding, but you are concerned. A medical student suggests that you put the patient in Trendelenburg, as he observes that the child has his feet down.

Is this a good idea?

No, this is not a good idea.

If you are to change the OR table in these cases, the robotic instruments must be disengaged [1]. The reason is that the robotic arms are solid structures and moving the patient with the robot and its appendages in place can and will cause major injuries to the patient.

In case of an airway emergency or severe hypotension or cardiac arrest, resuscitation of the patient necessitates that the robotic instruments be quickly removed.

Discussion

For the patient to be safe during robot-assisted surgery, advanced planning is essential as to what to do should a problem arise. All team members must familiarize themselves with the issues that are specific for this type of surgery. The biggest problem with these robots is that access to the patient is severely limited. It is essential that the OR team practices the crisis scenario of removing the robotic equipment and gaining access to the patient quickly should it be necessary. The limited access to the patient requires special monitoring. A left-sided precordial stethoscope can monitor inadvertent right main-stem intubation. Core temperature should be monitored and maintained with a warm OR, warm IV fluid, and forced air warming. The latter can be difficult to place because of the need for surgical access and the placement of the robotic arm. The placement of an intra-arterial catheter allows for continuous arterial blood pressure monitoring and blood gas sampling. The latter is important as the laparoscopic operations lead to decrease in lung volumes, impaired ventilation, and increased CO2 absorption [1].

In the case above, an inadvertent gradual increase in pneumoperitoneum decreased venous return with an increase in airway pressure. When this was corrected, the child's blood pressure returned to normal. It is also important to realize that with the use of the reversed Trendelenburg position, a 50% reduction in cardiac index can be expected [2].

Lesson

Advanced planning and understanding of the robot equipment are essential prior to anesthetizing a patient undergoing robot-assisted surgery. Failure to do so can lead to disaster.

- Mariano ER, Furukawa L, Woo RK, Albanese CT, Brock-Utne JG. Anesthesia concerns for robot-assisted laparoscopy in an infant. Anesth Analg. 2004;99:1665–7.
- Joris JL, Noirot DP, Legrand MJ, et al. Hemodynamic changes during laparoscopic cholecystectomy. Anesth Analg. 1993;76:1067–71.

Case 61: An Airway Emergency in an Out-of-Hospital Surgical Office



Today you find yourself in a free-standing oral surgery office which is 3 miles from the nearest hospital. You are to provide conscious sedation to a 38-year-old man (80 kg and 5'10''). He is scheduled for a 3-h oral surgery procedure, consisting of multiple dental extractions, alveoloplasty, and placement of multiple dental implants in the mandible. He is otherwise healthy and classified as an ASA 1. The conscious sedation consists of midazolam, ketamine, meperidine, and propofol in multiple divided doses. He is monitored using standard monitoring, including a precordial stethoscope. Supplemental oxygen is provided throughout the procedure via a nasal cannula. After 2.5 h of an uneventful surgical procedure, the surgeon notices a rapidly expanding hematoma in the floor of the mouth as well as a rapid enlargement of the posterior part of the tongue. The patient begins to complain of difficulty in breathing. His oxygen saturation remains in the mid-90s. The surgeon's attempt to control the bleeding fails. He now believes the reason for the hematomas is an arterial bleed in the floor of the mouth caused by one of the implants. The hematoma continues to expand and the saturation is now falling to 85%. You stop all IV sedation and attempt a blind nasal intubation but that fails. A jaw thrust with a face mask and ventilation with 100% oxygen does not improve the patient's saturation. Any attempt at an LMA insertion or oral intubation is deemed impossible due to the degree of mechanical obstruction caused by the hematoma.

What is the most important thing to do now, and what other airway maneuvers can you think of?

The first thing to do is to call 911 so that you can get the patient transported to a hospital. Remember this is only a free-standing oral surgical office.

As regards the airway, sit the patient up and place bilateral French nasal airways. But most importantly, place a tongue suture to retract the tongue away from the posterior pharynx. This should help alleviate the airway obstruction.

Discussion

This problem happened to a friend of mine, Dr. Terri Homer (personal communication). In her case the surgeon had placed the tongue suture during an earlier stage of the procedure. The paramedics arrived within 5 min, and the patient was transported to the hospital via ambulance in a sitting position, with nasal airways placed and Dr. Homer maintaining constant forward retraction of the tongue with the tongue suture. The oxygen saturation was maintained in the low 90s. On arrival at the hospital emergency room, the ENT team was called. A successful fiber-optic nasal intubation was performed. The patient was sedated and taken to the OR where he underwent surgery and the arterial bleeding of the floor of the mouth was successfully sutured. Postoperatively, the patient remained intubated overnight in the ICU and was discharged from the hospital 48 h later.

If the airway had been lost despite the maneuvers mentioned above, then a cricothyrotomy kit should be available. Failing that, then a tracheostomy must be done [1]. It is also important to know that many dental surgeons place a tongue suture (under local anesthesia) at the beginning of the procedure. This is used to intermittently or continuously gently retract the tongue during the surgery. As the IV sedation is given intermittently, there may be occasional respiratory obstruction. This can easily be treated with a gentle pull on the tongue suture.

Lesson

Know the various options available to you should you have to manage a rapidly obstructing airway in the out-of-hospital surgical setting.

Also remember to call 911, as soon as you realize you have an airway problem.

Reference

1. Sadda R, Turner M. Emergency tracheotomy in the dental office. Int J Oral Maxillofac Surg. 2009;38(10):1114–5.

Case 62: A Case of Recent Hip Replacement Coming for a Cystoscopy



A 75-year-old frail lady (ASA 3, weight 50 kg, and height 5'7") is scheduled for cystoscopy and hydro-dilation of the bladder. Her medical history includes severe osteoarthritis, cardiovascular disease, and hypertension. Three weeks before this scheduled surgery, she had undergone a successful right hip replacement. Her left hip is also very painful and stiff and has a lack of mobility. She also complains about painful and stiff shoulders. She takes various pain medications but cannot remember what they are.

You see the patient with your resident in the preoperative area. The patient has had a bad experience with a spinal in the past and does not want another one.

Your resident plan is to give her some sedation with midazolam and fentanyl prior to placing her in lithotomy. After she is comfortably placed, he plans to induce a general anesthesia and use an LMA. You agree that this would seem like a good plan as you have seen a frail patient break her neck of femur when being positioning in lithotomy after general anesthesia was induced. Also, in this case, the chance of dislocating the newly replaced hip should also be a consideration. However, there is also a potential serious anesthetic concern when anesthetizing a patient who is in the lithotomy position.

What can that be?

Should the patient, already in lithotomy, regurgitate during induction of general anesthesia, how are you going to safely and quickly turn her onto her side?

This case was presented to us not long ago. Although she had a bad experience with the spinal, we convinced her to have another spinal. She agreed.

Discussion

The patient was brought to the operating room after having been given ½ mg of midazolam in the preoperative area. In the operating room, monitors were placed while the patient was sitting up. After that she was given 25 mg of fentanyl intravenously. Luckily for us the spinal went in smoothly on the first attempt, and an adequate bupivacaine dose was given. Immediately thereafter she was gently with her cooperation, placed in the lithotomy position. The procedure was concluded uneventfully.

Hydro-dilation is very painful and cannot be done without a spinal/epidural or general anesthesia. (In certain countries, dilating the bladder is an effective torture method to get prisoners to talk.)

When I started anesthesia, most surgeons would insist that the patient be placed in lithotomy position prior to inducing general anesthesia for many different surgeries. This was done to save time. Luckily for the patient, this is not the practice today. I remember as an operating room orderly, prior to starting medical school, I was instructed to place a fully awake patient for an abdominal/peritoneal operation in a lithotomy position prior to induction of anesthesia. Being my first day and nobody was available to show me how to strap the patient properly, it nearly ended in disaster. After induction of anesthesia, the patient was placed in Trendelenburg, with the result that the patient started to slide off the operating table. Only the quick acting anesthesiologist saved the patient from landing on the floor. That was my last and only day as an orderly. The next day I was made an ambulance driver – a job I kept for 3 months before starting medical school.

Lesson

Always treat frail patients very carefully when placing them on the operating table. This is especially true if they are to be placed in the lithotomy position. Failing to do that can lead to broken bones and/or dislocating a newly replaced hip.

Inducing anesthesia in a patient in the lithotomy position could lead to regurgitation of stomach content. Failure to quickly treat this successfully by Trendelenburg, suction, and turning the patient preferably to their right side could lead to aspiration into both lungs.

Case 63: A High Glucose Concentration in an Epidural Catheter Aspirate: Should One Be Concerned?



A 62-year-old man had undergone an uneventful colostomy surgery for bowel obstruction. He received a routine GA and epidural infusion. His past history was significant for colon cancer, hypertension, and cardiovascular disease. Of note was that he did not have diabetes mellitus.

After an uneventful operation, he arrived in the post-anesthesia care unit (PACU) pain-free and with vital signs stable. In PACU a continuous epidural infusion of 0.25% bupivacaine (95 ml) with buprenorphine 0.5 mg (2.5 ml) and droperidol 2.5 mg (1.0 ml) was commenced at 2 ml/h. Two hours after the start of the epidural infusion, with the patient still in PACU, an aspiration test was performed. This was done since there was a concern that the epidural catheter might have migrated into the subarachnoid space [1, 2]. Clear fluid (2.5 ml) was easily aspirated through the catheter, and the aspirate was sent to the laboratory for glucose estimation. The catheter sample was 83 mg/dl, while the patient's blood glucose concentration was 73 mg/ml.

Since the difference was small, it was decided to keep the infusion going and wait 2 h. Again the aspiration of clear fluid was easy. This time the sample contained 100 mg/dl of glucose, and the patient's blood glucose concentration was 76 mg/ml. The patient was pain-free and very happy with the pain management. The level of the block was T-8. On exam there was no definite motor block of the lower limbs.

Since subarachnoid catheter migration could not be completely ruled out due to the high glucose concentration in the aspirate, you decide to remove the epidural catheter.

But was this the right decision?

This was not the right decision [3].

In the case report by Katori et al. [3] who after removing the catheter checked the packed insert for buprenorphine, they found that it contained "50 mg per ml of glucose." In their case [3], they calculated the glucose concentration of the epidural infusion fluid to be of 127 mg/dl (125 mg of glucose in 98.5 ml). Hence this could explain the high glucose concentration that was obtained.

Discussion

It is imperative to make sure that an epidural catheter does not migrate into the subarachnoid space at any time during the infusion period. The daily aspiration test is done to identify this problem. If a higher glucose concentration is found in the aspirate than in the patient's blood, then the possibility exists that the catheter may have migrated. Other tests to distinguish between CSF and local anesthetic fluid are temperature of the fluid dropped on the back of the investigators' hand (CSF is warmer), pH of the solution, and turbidity when mixed with sodium thiopental [1]. In that study [1], we found that anesthesiologist could distinguish CSF from local anesthetic fluid, correctly with temperature on the back of the hand (84%), with glucose (97%), and with pH (91%). Using sodium thiopental, only 50% had it correct.

Lesson

Watch out when drugs are added to an epidural local anesthetic infusion. It is imperative that you know what the added drug solution contains beside the active drug.

- 1. Walker DS, Brock-Utne JG. A comparison of simple tests to distinguish cerebrospinal fluid from saline. Can J Anaesth. 1997;44:494–7.
- 2. Ennis M, Brock-Utne JG. Delayed cutaneous fluid leak from the puncture hole after removal of an epidural catheter. Anaesthesia. 1993;48:317–8.
- 3. Katori K, Shigematus K, Higa K. Buprenorphine contains glucose. Anesthesiology. 2003;98:799.

Case 64: A General Anesthesia in a Patient Who Has Had a Recent Eye Operation



A 76-year-old man (ASA 2, weight 70 kg, and height 5'9") is scheduled for a right total hip replacement under both general and spinal anesthesia. He has declined a pure regional block and wants to be asleep. Twenty days ago, he underwent an uneventful vitrectomy and internal drainage of a large subretinal hemorrhage under monitored anesthesia care. He is otherwise healthy with moderate hypertension which is well controlled.

Based on the information above, are there any concerns when anesthetizing this gentlemen?

Nitrous oxide must not be given.

Discussion

Visual loss has been reported after the use of nitrous oxide gas for general anesthesia in patients who have had intraocular gas instilled up to 30 days after vitrectomy [1]. The authors [1] reported three cases that had undergone vitrectomy and who all developed visual loss postoperatively after a non-ophthalmic surgery where nitrous oxide had been used. The authors [1] recommended that anesthesiologists should be aware that the intraocular gas bubble introduced by the ophthalmologist can persist in the eye for up to 10 weeks after ocular surgery.

Nitrous oxide is much more soluble than other gases and therefore exerts a potent effect on all intraocular gases. A more than threefold volume increase may be seen over 1 h when nitrous oxide is used [2]. This increase is sufficient to close the central retinal artery [1] with potentially disastrous results.

The authors [1] recommend that patients who have undergone a vitrectomy should be given a bracelet to wear. This should describe the eye operation and when it would be safe to use nitrous oxide.

Lesson

In patients with a recent eye vitrectomy, nitrous oxide should not be used in any surgery until the gas has been seen to be reabsorbed. If one does not know if the gas has been reabsorbed, then it is best to avoid nitrous oxide in all patients that have had a vitrectomy in the last 3 months.

- 1. Vote BJ, Hart RH, Worsley DR, Borthwick JH, Laurent S, McGeorge AJ. Visual loss after use of nitrous oxide gas with general anesthetic in patients with intraocular gas still persistent up to 30 days after vitrectomy. Anesthesiology. 2002;97:1305–8.
- Stinson TWIII, Donlan JV Jr. Interaction of intraocular air and sulfur hexafluoride with nitrous oxide. A computer simulation. Anesthesiology. 1982;56:385–8.

Case 65: Another Awake Craniotomy



You are scheduled to do provide anesthesia care for an awake craniotomy. The patient is a 73-year-old female who has a left frontal lobe glioma. The patient is classified as an ASA 3 and has a BMI of 35. She had the following comorbidities: hypertension on metoprolol and recent onset epilepsy. The latter is treated with Keppra (levetiracetam), valproic acid (valproate), and Tapclob (clobazam). Her vital signs and ECG are all within normal limits, and she has consented to an awake craniotomy under local anesthesia with intermittent analgesia and sedation with remifentanil and propofol, respectively.

Are there any concerns regarding the successful completion of an awake mapping surgery in this patient?

Valproic acid and its derivatives [1–3] can cause hyperammonemia with an associated encephalopathy.

Discussion

Martinez et al. [1] found that after removal of the bone flap in the above awake craniotomy case and with all sedation stopped, the patient exhibited very slow mentation and excessive progressive drowsiness over a period of minutes. The patient stopped breathing, and the airway was promptly secured and general anesthesia commenced. The surgery was aborted, and the patient was taken to the ICU for ventilator support and evaluation. Her ammonia level was found to be elevated, and she was treated with an infusion of l-carnitine. She recovered quickly with this treatment. No postoperative complications were seen.

Initially it was thought that children and young adults were more prone to develop hyperammonemia with an associated encephalopathy [2], but lately it has been noted that older patients also appear to develop this side effect to the same extent [3].

In a recent review article about awake craniotomy [4], the authors state that some institutions routinely administer anticonvulsant before surgery, whereas others, if the indication of the awake craniotomy is epilepsy, then they withhold anticonvulsant to facilitate cortical mapping.

Lesson

When scheduled to provide anesthesia care in an awake craniotomy, it is imperative to review all the drugs the patients are taking and be fully aware of all the drug's potential side effects.

Ammonia encephalopathy should be considered in patients treated with valproic acid and its derivatives. Preoperative ammonia levels must be done prior to any awake craniotomy. If the levels are found to be high, then all drugs that are known to cause increased ammonia must be stopped and replaced with other appropriate antiepileptic drugs.

- 1. Martinez VG, Fernandez-Candil JL, Vivanco-Hidalgo RM, Terradas PS, Jorba LA, Perz AR. Ammonia encephalopathy and awake craniotomy for brain language mapping: cause of failed awake craniotomy. Rev Esp Anestesiol Reanim. 2015;62:275–9.
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- 4. Meng L, McDonagh DL, Berger MS, Gelb AW. Anesthesia for awake craniotomy: a how to guide for the occasional practitioner. Can J Anesth. 2017;64:517–29.

Case 66: Spinal Fracture and Flail-Segment Rib Fractures Following a Motor Vehicle Accident



A 44-year-old was admitted to the emergency room following a MVA. He had extensive crush injuries, bilateral flail chest, unstable traumatic spine fractures of T10 and T11, as well as other injuries. He had been a smoker and was treated for hypertension. He was classified as an ASA 2 E. His BMI was 28.

The patient was scheduled for an emergent posterior spinal instrumentation and fusion (PSIF) [1]. Chest x-ray taken 1 h ago showed fractured ribs bilaterally but no pneumothorax or hemothorax. He arrives in the operating room. You examine the patient and find no evidence of any lung pathology, and the abdomen is soft.

The flail-segment rib fractures are a concern to you. When placing this patient in the prone position on chest bolsters, the fractured ribs could create a life-threatening pneumo- and/or hemothorax.

Have you any suggestions as to how to position this patient to possibly prevent the above complications?

By using the anterior portion of an oversized back brace like the thoracolumbar sacral orthosis brace (TLSO, Breg, Carlsbad, CA 92010) in conjunction with the Jackson's spinal frame, one can produce a wider, more anatomically shaped surface to the chest and potentially reduce the risks of pneumo/hemothorax [2].

Discussion

In a case report outlined above [2], two PSIF attempts failed. In both cases difficult ventilation occurred, and the end-tidal CO2 was dramatically reduced. The patient was taken back to the operating room later that day, but this time he was fitted with an oversize TLSO brace. Only the anterior portion was used to support his chest, while he was in the prone position for the surgery. The patient had an uneventful recovery and discharged home.

Early fixation of unstable traumatic spine fractures has become more popular since it was advocated with good results in 1976 [1]. As anesthesiologist it is important to attempt to prevent the complications that can arise when these patients are turned prone onto chest bolsters. In these cases, it is imperative to attempt to maintain the patient's cardiovascular stability throughout the operation.

Lesson

Patients with unstable spine factures and flail-segment rib fractures may benefit from an oversize TLSO which should hopefully help to prevent pneumo/hemothorax.

- Bellabarba C, Fisher C, Chapman JR, Dettori JR, Norvell DC. Does early fracture fixation of thoracolumbar spine fractures decrease morbidity or mortality? Spine. 2010;35:S138–45.
- Pennington M, Dagal A, Bransford R. A technique to allow prone-position spine surgery in the patient with unstable spine fracture and flail-segment rib fractures. A A Case Rep. 2016;7:2–4.

Case 67: Angioedema in the Emergency Department



You are called urgently to the emergency department (ED) for an emergent endotracheal intubation. The patient is a 65-year-old African-American female (BMI 32) with a history of hypertension and diabetes mellitus type 2. She takes an ACE inhibitor and metformin. You are told by the emergency physician that the patient was admitted 2 h ago with angioedema. She was treated with epinephrine, methylprednisolone, and diphenhydramine. Unfortunately, the tongue swelling has increased dramatically in the last 30 min.

On examination you find an anxious lady. The tongue is seen to be very swollen and is protruding out of her mouth. When the patient attempts to open her mouth, you see no hard palate but only the swollen tongue. Her chest is clear. She is on a 100% non-rebreather face mask. Her vital signs are oxygen saturation of 93%, heart rate 106 bpm sinus rhythm, BP 170/96, and her respiratory rate is 28 per minute.

You reassure her that you will look after her and you explain what you plan to do. She accepts your suggestion but tells you to get on with it. Through the IV, you give her glycopyrrolate 0.6 mg. Nasal trumpets coated with lidocaine ointment are used for progressive dilation of the nasal passage. After the trumpet is removed, a number 6.5 endotracheal tube (ETT) is placed in warm saline. The ETT is advanced over a red rubber catheter to the back of the pharynx. Topicalization with nebulized lidocaine is done. Through the suction port of the fiber-optic scope, you give 100% oxygen at 4 liters/min. This increases the oxygen concentration in the pharynx and also helps to blow away secretions. Unfortunately, your attempt at nasal fiber-optic intubation proves impossible due to severe airway swelling, excessive secretions, and patient intolerance with gagging, retching, and vomiting. The oxygen saturation drops to 85%.

You remove the scope, reinsert the nasal trumpets, and replace the oxygen mask. The patient's oxygen saturation improves to 91%, but she now looks very tired. Her respiratory rate has increased to 34 per minute.

The ED physician suggests that you:

- 1. Either sedate and then maybe paralyze the patient so that you can secure the airway more easily
- 2. Or take the patient to the operating room with the face mask for an awake tracheostomy

Are these good suggestions? Which one would you do? Or would you suggest something else?

In a patient like this you should NEVER:

- 1. Sedate without knowing that you will be able to secure the airway rapidly.
- 2. Move from the ED. You call the surgeon to come down to the ED and do the awake tracheostomy there.

Discussion

An awake emergency tracheostomy in the ED is the safest way to secure the airway in this precarious case. I have seen a patient like this being transferred from the ED department up three floors to the operating room (OR) via an elevator. But by the time the patient arrived in the OR, there was nothing we could do as the patient had died from airway obstruction. Furthermore in patients with severe airway edema, the administration of sedatives, hypnotics, and/or neuromuscular blockers is contraindicated.

In cases like this, communication between anesthesiologist, surgeons, emergency physicians, and the patient is paramount in successful management of the airway.

In the above case which was successfully treated in the ED, the tracheostomy was done with the patient fully awake, a face mask delivering 100% oxygen and verbal reassurance. Having someone hold the patient's hand (like a handshake) can prove most helpful. Remember also to remind the surgeon of the upper limit of the local anesthetic. You do not want a toxic dose of the local anesthetic given in this setting. It is imperative for a successful awake tracheostomy that more than adequate field block of the pretracheal tissues is performed. Depending on the case, dexmedetomidine can be given in (4–8 mcg boluses) or meperidine 10–25 mg.

In this case, the cause of the angioedema was believed to be the ACE inhibitor which the patient was taking. This is a well-known side effect of ACE inhibitors and occurs more commonly in African-American patients, especially females [1].

Lesson

In these cases, confirm the patient's findings with a through exam, and never listen to well-meaning suggestions that are contra to what you know is unsafe. Lastly always have a backup plan should plan A falter.

Reference

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Case 68: Cranioplasty: Should You Be Concerned?



A 29-year-old, otherwise healthy male presented for a left-sided cranioplasty with replacement of his original bone flap.

Six months before he had been hit by a car while riding his bike. He developed right and left epidural and subdural hematomas. These were both evacuated emergently, and a left-sided craniectomy was performed. Unfortunately the traumatic brain injury left him in a persistent vegetative state, without the ability to track or follow commands and with only minimal response to noxious stimulation.

Preoperatively you find his vital signs were normal. His pupils were equal and small, and they reacted to light. He was taken to the operating room, where you connected him to the anesthesia via his indwelling tracheostomy. Anesthesia was induced with sevoflurane and fentanyl. Maintenance of the anesthetic was with isoflurane in oxygen 30%, vecuronium, and fentanyl. At the end of the case, the neuromuscular blockade was reversed and spontaneous ventilation resumed.

On arrival in the PACU, his vital signs were HR 76 (NSR), BP 114/63, respiratory rate 10, and temperature 36.8C. His oxygen saturation was 100%.

You handed the patient over to the PACU nurse and informed her that his mother and the ambulance were outside waiting to take him home. Furthermore, you tell the nurse that if all vital signs are stable, the patient can go within 30 min.

Would you agree with this discharge plan? If not, then why not?

Close postoperative monitoring of these patients must be done for at least 12 h in an ICU.

In a series of four patients, massive cerebral swelling immediately after cranioplasty has been reported [1]. In that series of four patients, all succumbed.

Discussion

We had a case like the case described above [2]. Within 15 min of arriving in the PACU, the patient began to develop an irregular respiratory pattern with subsequent bradycardia into the low 40s but with his blood pressure unchanged.

On examination, the patient's pupils were now found to be 5 mm dilated bilaterally and nonreactive. An emergent CT scan of his head demonstrated an acute, large intraparenchymal hemorrhage centered within the left parietal lobe with significant mass effect, a 20 mm left to right midline shift.

In this case, after discussion with his family, he was made comfortable and died 16 h later in the hospital.

Lesson

A high index of suspicion is warranted and immediate imaging should be obtained if signs and symptoms of increasing intracranial pressure are observed in these patients.

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- Miller C, Larson B, Brock-Utne JG. Acute intraparenchymal hemorrhage and cerebral herniation following cranioplasty. Western Anesthesia Resident Conference, Seattle, 1–3 May 2015.

Case 69: More Haste Less Speed



It is a Saturday morning. An "urgent" case for a biopsy of a lymph node (Virchow) situated in the left supraclavicular fossa is booked under monitored anesthesia care (MAC). The patient is an 86-year-old female (120 lbs and 5'2'') who is described as frail. Her past history consists of CAD and hypertension. Recently she has lost weight and developed a chronic cough. A CT scan of the chest reveals a mass in the upper left lung. You explain to the patient that you will look after her and that the procedure will be done under local anesthesia. You have decided that you are not giving her any sedation.

The patient is brought in from home by her son. The surgeon who is in an obvious hurry, his child has a baseball game, helps to bring the patient to the operating room. The patient is placed on the operating table, and while you place monitors and an oxygen face mask, the surgeon starts to paint DuraPrep surgical solution (iodine povacrylex (0.7% available iodine) and isopropyl alcohol, 74% w/w) over the left supraclavicular fossa and neck. The prep takes 2 min with another 2 min of local anesthetic injection. While the surgeon goes out of the operating room to wash his hands, the patient's head and upper torso are covered with a drape. The patient's vital signs remain stable, and she says she is comfortable. The surgeon returns, reapplies more DuraPrep, and picks up the cautery (diathermy) pen to start the operation.

Is there something wrong with this picture?

In this case the surgeon did not wash off the excess DuraPrep and dry the skin. Hence when the hot wire cautery was placed on the skin with the wet DuraPrep, enough heat was generated to ignite the DuraPrep. A second-degree burn resulted to the patient's left neck, side of head, supraclavicular fossa, and upper back before the fire was extinguished. The case was aborted. This unfortunate case happened to an anesthesia colleague.

Discussion

DuraPrep is an antiseptic known as iodophors. The antiseptic effect is exerted by slowly releasing iodine. Its big advantage is that it is quick drying. Most of the alcohol dissipates into the air. The directions of use state that you must allow it to dry completely (minimum of 3 min on hairless skin and up to 1 h in hair prior to the use of electrocautery).

When using DuraPrep it is important not to scrub but to paint the solution on the area. Do not allow the solution to pool. If pooling does occur (like in the case above), use the sponge applicator to absorb excess solution. If the solution accidentally gets outside the prep area (like in this case – running down the back), remove with excess gauze. Remember that wet hair is flammable. If the hair gets wet, it must be dried completely. If tucked towels are used before prepping, they must be removed immediately after the prepping.

Hot wire cautery can ignite all alcohol-based antiseptics even if these contain as little as 20% alcohol [1-3].

It is important to remember that alcohol solution should not be used in open wounds like lacerations or gunshot wounds for the above reasons.

If you ever see in the operating room wet or pools of alcohol antiseptic on a surgical site and the surgeon is about to start, just say: Stop. This situation is akin to putting brandy over a Christmas pudding and then lightening it. This for the uninitiated is a common practice in the Great Britain to the delight of the children.

Lesson

All aqueous alcohol-based antiseptic must be carefully dried off before proceeding with cautery; failing to do that may result in fire and serious burn to the patients.

- 1. Briscoe CE, Hill DW. Inflammable antiseptics and theatre fires. Br J Surg. 1976;63:981-3.
- 2. Bruley ME. Surgical fires: perioperative communication is essential to prevent this rare but devastating complication. Qual Saf Health Care. 2004;13:467–71.
- 3. Report by the American Society of Anesthesiologist Task Force on Operating Room Fires. Practical advisory for the prevention and management of operating room fires. Anesthesiology. 2008;108:786–801.

Case 70: A Pregnant Patient for a Carpal Tunnel Operation



It is late in the afternoon in a large university hospital surgical operating suite. You, as a senior anesthesiologist, are stopped by a resident anesthesiologist who asks you to come urgently to an operating room. On the way to the room, the resident anesthesiologist tells you about the patient. The patient is an otherwise healthy 28-yearold pregnant female (24/52) scheduled to undergo a carpal tunnel release. A successfully working supraclavicular block has been placed without any sedation about 5 min ago. Unfortunately the patient now complains of difficulty in breathing. The resident also tells you that the operation is being performed since the patient has complained about intractable pain for several months and is worried that she will not be able to tend to her newborn child.

You enter the room, and the patient is lying on the operating room table with an oxygen mask covering her face. You observe that reservoir bag moving at a respiratory rate of 16. Her other vital signs are heart rate of 78 bpm, BP 135/85, oxygen saturation 100%. The anesthesiologist in charge tells you that a few minutes after the supraclavicular block had been placed, the patient complained of difficulty in breathing. She suggests that the most likely diagnose is a phrenic nerve paralysis. You concur. The head is elevated and the patient is reassured. The surgeon, who is scrubbed and ready to go, has not started the operation. He is concerned about the patient and wants to abort the procedure.

The staff anesthesiologist in charge of the case does not agree. She wants to know what you would recommend; carry on or abort the operation?

So what will you do and then recommend?

You introduce yourself to the patient and examine her. You agree with the assessment of a phrenic nerve paralysis. You explain to the patient that the cause of her difficulty in breathing is a temporary paralysis of the nerve that controls the movement of the diaphragm. You confirm and inform her that the vital signs are within normal limits. You tell the patient that if she was your wife, you would suggest she carry on having the procedure. As the patient has not had any sedation, it is imperative that you ask her what she would like to do. If she says she wants to abort the procedure, then the case is cancelled. If she says to go ahead, you would suggest to the surgeon that he starts this very short procedure forthwith.

Discussion

You may wonder why the anesthesiologist elected to use a supraclavicular block in this case. The reason was that the patient's father had a successful unknown procedure on his hand under a supraclavicular block and she wanted the same. If I had been the anesthesiologist in charge of this case, I would have talked her out of it [1]. There are other regional anesthesia techniques that would have been safer in this case. These include, all without sedation, local anesthesia to the incision site only, a Bier Block, and a regional anesthesia like an axillary block.

The rationale for continuing the procedure would be that no new risks can be foreseen with this block. Actually if the patient would require ventilator support, what better place to secure the airway than in the operating room [2]?

Lesson

When faced with a clinical problem, as above, it is imperative to have all the facts including the primary team's physical finding. If the patient has had no sedation, then the patient must be involved in the discussion to proceed or not.

- Gazmuri RR, Torregrosa SA, Dagnimo JA. Should supraclavicular brachial plexus block be avoided in pregnancy? J Clin Anesth. 1992;4:333–5.
- 2. Noel TA. You paid for the block. Why throw it away? J Clin Anesth. 1993;5:259.

Case 71: A Request to Provide Isoflurane Anesthesia for Treatment of Status Epilepticus



You are the anesthesiologist on call. It is late at night. An attending ICU physician requests that you provide an isoflurane general anesthetic to an ICU patient in refractory status epilepticus (RSE). Volatile anesthetic reliably induces burst suppression within minutes and is considered a reasonable treatment option for RSE [1–3].

You are concerned about the use of volatile anesthetic in the ICU for the following reasons:

- 1. One of your on-call anesthesiologists has to provide the general anesthetic which could take several hours. This task cannot be left to an ICU nurse or to a non-anesthesia provider.
- 2. How are you going to remove the waste anesthetic gases (WAG) from the ICU?
- 3. If WAG cannot be removed, the ICU staff must be informed about the risks of WAG. Any pregnant staff or patients must be deployed elsewhere.

You go and see the patient in ICU. The patient is a 44-year-old male in RSE. The ICU team has tried midazolam, propofol (both bolus and infusion), phenobarbital boluses, and phenobarbital IV infusion without success. You realize that the request for an isoflurane anesthetic has merit.

But is this what you want to do? Anything else you would suggest?

Ketamine infusion was considered a better option when considering the potential concern and complexity of the use of isoflurane in the ICU [4, 5]. These articles have documented the effectiveness of ketamine in seizure suppression. In this case [4] ketamine successfully weaned the patient of the phenobarbital coma and treated the RSE. Ketamine was titrated to burst suppression using EEG with a neuro-technician in attendance. The infusion was controlled by the ICU staff after they had been educated over a 1-h period.

Discussion

Ketamine was a logical alternative when considering the above 1–3 concerns. WAG scavenging would have required a unique adaptor to bypass the existing wall suction diameter index safety system (DISS), and the educational initiatives regarding WAG would have been imperative to ensure staff safety.

Lesson

Ketamine is also effective in seizure suppression.

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Case 72: No Methylene Blue in the Urine: What Would You Do?



You are called as a senior anesthesiologist to a room where an ongoing robotic prostatectomy is in progress.

On the operating table is a 73-year-old man with hypertension and prostate cancer (ASA2). His preoperative laboratory values (hematocrit, electrolytes, kidney, and liver functions) are all within normal limits. You observe the anesthesia monitor and note with satisfaction that his vital signs are stable and the oxygen saturation is 97%. Your colleague tells you it had been 100% at the beginning of the anesthetic. The chest is clear and air entry is heard bilaterally. Peak inspiratory pressure has remained at 28 cm H20 after the patient was put in Trendelenburg.

Your anesthesia colleague tells you that he has given, at the surgeon's request, an IV dose of methylene blue (MB) 10 mg about 75 min ago. Since no discoloration of the urine was seen, another IV dose of MG 10 mg was given 25 min ago. You look at the urine in the urine bag and confirm no blue-green discoloration.

You check that the IV is working. It is. The surgeon tells you that the bladder was not distended when he started closing about 15 min ago. The medical student is now closing the skin where the trocars, etc. have been. Finally, you check that the urine is coming out of the urine catheter. It is.

The surgeon is concerned and wants to reintroduce the trocars and explore for any obstruction to urine flow.

What do you say? Another dose of MB? Reintroduce the trocars, or what?

Try indigo carmine (IC). This drug is thought to be superior to MB since IC is not readily metabolized but is rather freely filterable by the kidneys [1].

If IC fails, re-exploration is needed. This to establish that there is free efflux of clear urine from both ureteral orifices and no sign of obstruction.

Discussion

Urologists use intravenous dyes in diagnosing genitourinary fistulas and in investigating ureteral patency. Methylene blue and indigo carmine are the most common dyes used today. MB has a half-life of 5-6.5 h, while IC has a biological half-life of 4-5 min after an IV injection. Generally, patients with clinically normal renal function demonstrate dye in their urine within 10 min. In this case methylene blue was not visualized after two intravenous injections, due to an increased biliverdin reductase B [1]. Methylene blue can metabolize into leucomethylene blue, which is colorless in urine [1]. This was the reason for why the urine was colorless in this case [1].

Leucomethylene blue is formed from a redox reaction catalyzed by biliverdin reductase B [1]. Biliverdin reductase B is significantly overexpressed in prostate cancer tissue giving these patients a higher than normal blood levels [2]. This upregulation may explain the lack of urine color change in patients with confirmed prostate adenocarcinoma. Since MB metabolism may also vary between patients, it may be an unreliable diagnostic indicator [1].

Indigo carmine, however, is not readily metabolized but is rather freely filterable by the kidneys. Therefore, it is important to appreciate that non-visualization of methylene blue may be a MB metabolism effect and not an anatomic one.

Indigo carmine (IC) may be superior to MB, as it is principally excreted unchanged in the urine. Be aware that if you give IC quickly intravenously, you may see a significant but temporary increase in the patient's blood pressure [3, 4]. Interesting also is that hypotension has been reported [5].

If in this case the IC had not worked, then the only option would be surgical re-exploration.

The decrease in oxygen saturation with MB administration could only tell you that the MB had most likely gone into the circulation.

Another reason for using IC is that methylene blue has been reported to cause prolonged disorientation after a parathyroidectomy [6].

Lesson

Indigo carmine may be superior to methylene blue as a diagnostic indicator to confirm ureteral patency especially in patients with prostate cancer.

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Case 73: A Right Upper Lobe Tumor and Concurrent Tracheal Polyp: What Lung Isolation Technique Would You Use?



Today you are to anesthetize an 86-year-old female (5'3" and 51.2 kg) (ASA3). Her past medical history is significant for hypertension, bladder cancer, and newly diagnosed lung adenocarcinoma of the right upper lobe. She is scheduled for a "flexible bronchoscopy and (R) VATS upper lobectomy." In addition to her (R) upper lobe tumor, her PET scan showed, as an incidental finding, a potentially fragile polyp (approximately 0.7 cm in diameter) attached to the anterior tracheal wall.

General anesthesia is induced without any problem, and she proves to be an easy mask ventilation. A Cormack-Lehane grade 1 view is seen on direct laryngoscopy with a size 3 Macintosh laryngoscope. You place a size 8.5 mm endotracheal tube (ETT) in the patient's glottis opening and leave it there. Through the ETT you now introduce a fiber-optic bronchoscope and see that you cannot place the cuff of the ETT below the vocal cords without the ETT touching/pushing on the tracheal polyp.

Since you are worried about avulsing the polyp and/or causing a serious airway bleed, the ETT is removed.

How would you safely provide anesthesia in this case?

A size 4 LMA Proseal was placed successfully, with a leak at 22 cm H_2O . An orogastric tube was passed through the LMA orogastric port into the stomach and placed on continuous suction. With a fiber-optic bronchoscope through the LMA, the tracheal polyp was biopsied and found to be benign on frozen section. Fiberoptic bronchoscopy also confirmed that the takeoff of the (R) upper lobe bronchus was about 1 cm below the carina making it feasible to use a bronchial blocker.

The only bronchial blocker that could be negotiated into the glottic opening through the curve of the Proseal LMA was a 9F Arndt wire-guided endobronchial blocker (WEB).

Successful lung isolation was achieved by looping the lasso of the Arndt blocker to an Ambu® aScope 3 Slim fiber optic bronchoscope behind the flexible tip. This allowed for placement of the blocker in the (R) main bronchus, avoiding the now friable tracheal polyp. The endobronchial blocker was secured at 50 cm.

The LMA leak pressure remained at 22 cm H_2O in the left lateral decubitus position. The blocker position was reconfirmed. Successful lung isolation was achieved, and an uncomplicated right VATS upper lobectomy was performed.

Discussion

Lung isolation for VATS procedures is most commonly accomplished with the placement of a double-lumen tube (DLT) in the airway. Sometimes a bronchial blocker is placed through a single-lumen tube (SLT) in the airway. When neither tube can be safely placed in the airway, the combined use of an LMA Proseal and Arndt endobronchial blocker can be an effective alternative to conventional techniques. Protection of the airway from gastric contents was helped with the orogastric tube. The combined use of a LMA Proseal and an Arndt endobronchial blocker allowed for achievement of one-lung isolation, without compromising adequate oxygenation and ventilation (personal communication Dr. Christopher Clave and Vivek Kulkarni).

This technique serves as an alternative to conventional lung isolation strategies in cases where such strategies are impractical or potentially harmful.

Lesson

In a case of a tracheal polyp, it is imperative to be inventive. In this case the combined use of a LMA Proseal and an Arndt endobronchial blocker allowed for safe achievement of one-lung isolation.

Case 74: Complete Heart Block During Central Line Placement



An 85-year-old man is scheduled for a mitral valve replacement. He is 180 lbs and 5'9" (ASA 3). His past history includes hypertension, paroxysmal atrial fibrillation, and severe mitral stenosis. The ECG shows a sinus rhythm (SR) with a left bundle branch block (LBBB).

In the operating room routine, ASA monitors are attached, and under local anesthesia an arterial line is placed. Thereafter the patient is anesthetized and the airway secured without any problems. However during the central line placement, using a guide wire, the ECG transitioned from SR to asystole. Chest compressions are started, but the patient remains in asystole.

Is there anything else you should do besides carry on with ACLS guidelines including IV epinephrine?

Pull the guide wire back.

In this case, by just pulling the guide wire back and the concurrent IV administration of epinephrine 20 mg, the patient went to a junctional rhythm with a pulse of 35–55 bpm and systolic BP of 85–95. The operation was concluded successfully [1].

Discussion

Guide wire tips are more rigid and less flexible than PA catheters and therefore potentially more arrhythmogenic [1]. If the guide wire is inserted less than 22 cm, the incidence of complications is decreased by 70%. The typical safe upper limit of guide wire insertion in an adult patient is 18 cm [1].

Complete heart block is seen in only 0.1% of patients undergoing catheterization of the right heart [2]. In this case, the patient was thought to develop catheterinduced AV nodal block or, more likely, a right bundle branch block (RBBB) which caused bilateral bundle branch blocks. The patient did not have an escape rhythm and therefore developed a cardiac standstill. In patients with preexisting LBBB, it has been suggested to prophylactically temporary pace these patients prior to undergoing right heart catheterization (Thornton and Ford, personal communication).

Transient RBBB is occasionally seen during guide wire insertion. This is thought to be due to mechanical trauma to the conduction system with an incidence of 3-12% [1].

Lesson

If you are using a guide wire during catheterization of the right heart, it is recommended not to introduce more than 18 cm in an adult patient.

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Case 75: Cervical Hematoma Following Neck Surgery



A 48-year-old man was scheduled for left parotidectomy under general anesthesia. He was 190 lbs and 6'0 (ASA 2) and had no significant past medical history. His vital signs in the operating room and after sedation were HR 78 bpm, BP 140/85. The parotid mass was 17×11 mm and diagnosed as a benign salivary neoplasm. No airway compromise was seen on imaging, and the patient denied preoperatively any symptoms of dyspnea, shortness of breath, or cough.

On the day of surgery, he underwent a routine general anesthesia induction with fentanyl, propofol, and succinylcholine. A grade 1 view of the airway was seen on direct laryngoscopy, and a 7.0 endotracheal tube (ETT) was placed in the trachea and taped at 22 cm at the teeth. Maintenance was with sevoflurane/fentanyl, and the patient's vital signs remained stable throughout the anesthetic. Upon emergence and with the patient breathing spontaneously at a rate of 24 per minute, HR 92 bpm and BP 170/95, he suddenly bucked, and a large left-sided cervical hematoma developed. The patient was reanesthetized with propofol, and the surgeon explored the wound. No source of the bleeding was seen. The BP at the time of the exploration was 90/70. You suggest that the surgeon put a drain in the wound, but he ignores you. "No reason," he says, "as there is no bleeding." He steps away from the operative field and tells you to wake the patient.

Questions:

- 1. Are you happy to wake the patient now? What are your concerns?
- 2. What anesthetic technique would you employ prior to extubation which could potentially prevent coughing/bucking during emergence?

- 1. You should be very concerned, as the BP was lower than normal during the reexploration. That may be the reason why the surgeon did not see any obvious bleeding. I remember, in the "old days," all surgeons before they started to close asked "What is the BP?" Always be aware of a low BP during closure.
- 2. The anesthetic technique that I would suggest is the following: Bring the respiratory rate down to 8–10 per minute, with the use of IV meperidine 10–20 mg in divided doses [1], maximum 150 mg. If you do not succeed, using meperidine alone, you can add in morphine or hydromorphone (Dilaudid). I do not recommend fentanyl, as it has a much more potent respiratory depressant effect and is of short duration. Turn the sevoflurane off, and leave the patient on N2O 70% in oxygen. Observe the gas monitors, and when no volatile gas is seen, turn the N2O off. Make sure the BP comes up to preoperative levels, and observe the neck for any evidence of bleeding. With N2O turned off, ask the patient to open the mouth, which in most cases they will do without bucking, etc. Talk to the patient, and tell them all is well and that you will now gently suck out their oropharynx. The latter can be difficult, as the surgeon in neck cases has often bandaged the entire neck, making it difficult for the patient to open their mouth.

Discussion

Prevention of bucking/coughing is of paramount importance in cases like this. I normally use the Laryng-O-Jet Kit (4% lidocaine hydrochloride topical solution (160 mg/4 ml (40 mg/ml) (International Medication Systems Limited, South El Monte, CA 91733) inserted into the trachea prior to the ETT placement. With the Laryng-O-Jet Kit in the trachea, a total amount of lidocaine 160 mg can be deposited onto the tracheal surface. In addition, I also place 2 cm, on each side of the ETT cuff, lidocaine ointment (5%) (Taro Pharmaceuticals Inc. Brampton, Ontario, Canada). A total of 4 cm. I have used the above technique with great success, and I therefore recommend it to you.

We have previously shown that by using a modified ETT (laryngotracheal installation of topical anesthesia (LITA), Sheridan Catheter Corp, Argyle, NY, USA), during neuroanesthesia, 75% of patient was found to have complete cough suppression upon emergence [2]. This ETT had a port for injecting topical anesthesia below and above the cuff.

The control group and the group that received saline only had a cough suppression of 14% and 13%, respectively [2].

Lesson

Be acute aware of a low BP during closure of a most surgical operations.

Coughing/bucking should be minimized in all anesthetic emergences. This is especially true in head/neck and neuro cases.

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Case 76: Transient Language Disturbance Following General Anesthesia



A 70-year-old lady (ASA1) with weight of 160 lbs and height of 5'8" has had an uneventful general anesthetic for an arthroscopy of her left knee. Several general anesthetics in the past have also been unproblematic. She is healthy and does not take any medications. She was given midazolam 1 mg IV, fentanyl 50 microgram, and propofol 150 mg and had an LMA. Her anesthetic maintenance was with sevo-flurane in N₂O 70% in oxygen. The operation lasted 30 min.

On arrival in the recovery room, around 15:30 pm, she is asleep, and her vital signs are stable. You, as the anesthesiologist, hand over the case to the nursing staff and leave to see your last case of the day. After having induced the next patient, the phone rings in your operating room and informs you that the patient has awoken but is talking in a foreign language. The language is recognized as German. The nurse also tells you that the patient seems to have had a "turn," with some movement of the left arm, and also she was staring at the ceiling just prior to speaking German. The "turn" had lasted for only 15 s. The nurse says the patient is fine now except she is still speaking German, and the nurse cannot communicate with her.

The patient vital signs are normal.

The nurse asks you what she should do? What would you suggest?

Take a blood sugar.

Discussion

Transient language disturbance has been reported with hypoglycemia [1].

The above case happened to me many years ago. In my case the blood sugar was found to be very low 25 mg/dL. This may have been caused by a long fast of more than 18 h. After 50 g of IV glucose, she suddenly started speaking English again. On questioning, the patient said that she was born in Germany in 1934 but had come to the United States in 1938 as an orphan. She was adopted by American parents who only spoke English. She had not spoken German since she left Germany. In a similar case report, Ward and Marshall [1], postulated that a temporal lobe seizure induced by hypoglycemia allowed the mother tongue to present itself. It would be totally unethical to induce hypoglycemia at a later date, without general anesthesia, to see if the foreign language would reappear.

There are many reports of transient language switches after cerebral insults in bilingual and polyglot patients [2]. There is a neuropsychological explanation for the disturbance. It is thought that the mother language is in part stored and acquired by "implicit memory systems" represented in the subcortical regions while the second language in contrast is displayed diffusely in the cerebral cortex [3]. Functional brain imaging has found distinct areas in the cortex associated with the mother tongue and the later acquired language [4].

A case by Kati et al. [5] describes a case of a 56-year-old woman who developed somnolence and mutism for 11 days postoperatively after total intravenous anesthesia with fentanyl, propofol, atropine, and vecuronium for a femur fracture fixation. The authors conclude that this mutism may have been caused by an unknown side effect of propofol. However, it is more likely that, in this case, the mutism was caused by the central anticholinergic syndrome (CAS) as described by Srinivasa et al. [6]. These authors felt that CAS was a distinct possibility, in this case, rather than propofol, since the patient in [5] had received atropine. The signs and symptoms of CAS are those described by sometimes even relatively small doses of atropine including somnolence, confusion, amnesia, agitation, ataxia, hallucinations, delirium, stupor, and coma. This is in addition to tachycardia, dry mouth, visual disturbances, and dysarthria. CAS symptoms can last from hours to days [7].

Following general anesthesia, unexpected neurological consequences may occur. The following story illustrates the many strange things that may happen. Many years ago I gave a spinal to an 89-year-old German-born lady who was going to have a below-knee amputation because of gangrene of her right leg. I gave her no sedation as she was resting peacefully after the surgery began. However, when the surgeon started sawing, she started singing German war songs from the First World War.

Lesson

There are several anecdotal accounts of patient postoperatively transiently reverting to a mother tongue. Should this happen, it is imperative to rule out hypoglycemia as a treatable cause.

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Case 77: A Flexible Suction Catheter Complication



A 10-year-old boy (ASA 1, weight 35 kg, height 4'8'') has just undergone a right inguinal hernia repair under general anesthesia. The operation has taken 30 min. The boy is still in the operating room. He is breathing spontaneously on 70% nitrous oxide in oxygen through his endotracheal tube (ETT). His respiratory rate has been titrated to 10 per minute with the use of meperidine (30 mg) IV. Your resident is performing the anesthetic under your supervision. You, as the attending anesthesiologist, are pleased with his performance to this point.

With the patient as described above, the resident places a 14 Fr soft flexible suction catheter in the oropharynx to suction any secretions. Some secretions are encountered, but his attempt to remove the catheter is meeting with resistance and is stuck at least 3 inches from the teeth. He is now trying hard to pull it out, but you intervene and say "Don't do that." You are equally unsuccessful in removing the catheter, despite an attempt to gently manipulate it by sticking your finger blindly into the oropharynx.

Now what will you do?

Turn the nitrous oxide off, wait 2 min, and then remove both the ETT and the suction catheter.

Discussion

In a previous case report [1], Onsiel and Shah describe a similar case in which the suction catheter could not be removed from the oropharynx. It was successfully taken out by removing both the ETT and the catheter at the same time. By examining both the ETT and the catheter, the cause of the dilemma was discovered. The suction catheter had formed a tight knot around the ETT explaining why the catheter could not be removed.

Nasogastric tubes have been known to knot around ETTs [2, 3]. Unintentional endotracheal extubation by orogastric tube removal [4] has been reported. Hence if your nasogastric tube and/or oropharynx suction catheter gets stuck in the oropharynx and cannot be removed, then do not pull hard, as you may damage oropharyngeal structures and also pull out unintentionally the ETT, too.

Lesson

Knotting of oropharynx suction catheter and nasogastric tubes can occur, albeit rarely. It is important to recognize this rare complication and not to make vigorous attempts to remove the catheter. This can induce more complications.

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Case 78: A Neurosurgical Case with a Sudden Disappearance of the Arterial Line Waveform



A 57-year-old female (ASA3, weight 148 lbs and height 5'6") is scheduled for the clipping of a left temporal arterial venous malformation. Her past history is significant for COPD, hypertension, and diabetes mellitus type 1. Her laboratory values and preanesthetic vital signs are all within normal limits.

The patient is seen in the preoperative area, sedated with IV midazolam, and taken to the operating room. Here noninvasive monitors are placed. You, as the attending anesthesiologist, notice that the medical student is putting the pulse oximeter on the left ring finger and the noninvasive BP cuff on the upper left arm. You make a mental note to speak to him about this, as putting both these two monitors on the same limb is something you do not do, mainly because of the intermittent decrease of the oxygen saturation. As an aside, I make it a point of not putting a disposable pulse oximeter on the right index finger but rather on the ring finger, on either hand. This helps to avoid corneal abrasions [1, 2].

You decide to change the left pulse oximeter to the right side after the patient is asleep. You do not want to delay the case by explaining this to the medical student, nor do you want him to look bad in the eyes of the operating room staff.

The operating room table is turned 180°. The patient's feet are now close to the anesthesia machine. In this position the patient is induced with a routine general anesthetic, and the patient's trachea is intubated with an endotracheal tube (ETT). Maintenance of anesthesia is with 70 nitrous oxide in oxygen, isoflurane, and remifentanil. A right radial artery is cannulated for continuous blood pressure monitoring. As per surgeon preference, a femoral central venous line is placed in the left groin. The patient is positioned in the left lateral park-bench position, and all pressure points are padded. The surgeon, who is in a hurry, starts to scrub the operating site, while the nurse drapes the patient from the neck down.

You turn to your monitors and observe the pulse oximeter (still attached to the left index finger) is 98%. The heart rate is 68 beats per minute (bpm) sinus rhythm. The noninvasive BP on her left arm has just recorded a BP of 130/72. The end-tidal CO2 is 32 mmHg, and tidal volume and peak pressures are normal. But to your chagrin, you notice that your right radial arterial line trace is now a straight line recording 0 mmHg.

You do the following: confirm the presence of the left dorsalis pedis pulse which is 68 bpm and with a good volume. Next you recycle the noninvasive BP and get the same as the preoperative reading, namely, 130/72. You successfully flush and aspirate blood from the arterial line, change the transducer, and relevel it. However all this is to no avail. Your resident suggests that she puts in an arterial line in the dorsalis pedis artery.

Do you agree, or what will you do?

Take the patient's drapes off.

You now notice that the right arm is white, pulseless, and cold.

Repositioning the patient into a supine position improves the circulation of the arm, as seen clinically and by the now replaced oximeter on the right ring finger. In such a case I would ask for another oximeter for the left ring finger as it is essential that you continuously monitor the circulation of the right arm during the operation.

Discussion

In a previous report [3], the authors with the patient still asleep called for a vascular surgeon. He performed a duplex Doppler of the right subclavian, brachial, and axillary arteries. Triphasic signals were recorded with the arm in a normal position, but with the patient in the park-bench position and the shoulder pulled down into the position required for surgery, no arterial blood pressure trace could be seen. The vascular surgeon diagnosed a thoracic outlet obstruction of the right subclavian artery. Using the oxygen saturation monitor, the patient was repositioned carefully to prevent recurrence of the problem, and the surgery concluded uneventfully.

The park-bench position provides a good surgical approach to the temporal lobe. Brachial plexus injuries, nerve pressure palsies, stretch injuries, and increased ventilation perfusion mismatch problems have all been documented as risks of this position [4, 5].

Had the saturation monitor being placed on the right ring finger, the diagnosis of the obstruction to the subclavian artery would have been diagnosed earlier.

Lessons

Thoracic outlet obstruction of the subclavian artery can occur in susceptible patients during park-bench positioning.

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Case 79: Not Another Corneal Abrasion



It is 9 p.m., and you are the attending anesthesiologist in a university hospital and on your last case. With 20 min to go, you are asked to see a female patient in the ward that has undergone a thyroidectomy about 8 h previously. She is complaining of a sore eye. You send your resident to go and see her.

He returns to tell you that the patient is a 62-year-old female (ASA 2, 170 lbs and height 5'8", BMI 25.8). She is otherwise healthy and on no medication for any medical condition. This was her first general anesthetic. It consisted of midazolam as a premedicant, propofol, fentanyl, and vecuronium for induction and 70% nitrous oxide in oxygen with sevoflurane for maintenance. Endotracheal intubation was achieved with a 7.0 endotracheal tube (ETT) using a conventional laryngoscope. The intubation was described as uneventful and the airway secured on the first attempt. Her eyes had been taped after induction. Mechanical ventilation was employed throughout. The patient's head was near the anesthesia machine. Shortly after induction, the patient became severely bradycardic, and blood pressure fell. Atropine 0.6 mg and ephedrine 15 mg were given with good effect. There had been two surgeons, one on either side of the patient's head. Low intraoperative blood pressure continued to be a problem and was treated with a total of 70 mg of ephedrine and IV fluids. The operation had lasted 2 h and 15 min. Toward the end of the operation, meperidine was titrated IV to a respiratory rate of 10-12 per minute. With the operation finished and the patient breathing spontaneously and awake, the ETT was removed. IV Toradol (ketorolac tromethamine) 30 mg was administered for postoperative analgesia. No other additional drugs had been given.

The resident reports that on examination, the patient was sitting semi-reclined in the bed complaining of right eye pain (pain scale 3/10) and frontal headache (pain scale 7/10). Cranial nerves are normal, and the extraocular movements are intact. He tells you that he has made a tentative diagnosis of corneal laceration and has ordered 5 mg of morphine subcutaneously.

What is your response?

Tell the resident to go back up and ask if the patient has:

- 1. A subjective vision loss (this is seen in acute angle glaucoma), not just blurred vision (the latter seen in corneal abrasions)
- 2. A feeling of a foreign body in the eye (the latter seen in corneal abrasions)

In this case the patient did have vision loss, very swollen eyelids, and did not have a feeling of a foreign body in the eye. An ophthalmologist was called, and he diagnosed acute angle-closure glaucoma.

Discussion

Acute angle-closure glaucoma after nonocular surgery is a rare complication of anesthesia [1-3]. Unfortunately, it can lead to optic nerve and cornea damage [1-5]. As anesthesiologists we should always refer to an ophthalmologist any patient who has a red eye and subjective vision loss postoperatively. This is especially true if the patient has no sensation of a foreign body in the eye.

In this case, the intraoperative ephedrine administration, combined with stress and surgery, precipitated the acute event in a patient with unrecognized severe angle-closure glaucoma. The right eye was treated with oral acetazolamide and eye drops of pilocarpine and betaxolol for 24 h with good effect. Later an iridotomy was performed, first in the right eye and then in the left eye. It is interesting that in the case reported by Lentschener et al. [2], the patient denied any previous eye symptoms suggesting subacute glaucoma. Hence it is imperative to be aware that patients who suddenly have postoperative severe visual loss and eye pain may have an undiagnosed subacute glaucoma.

Corneal laceration is the most frequent postoperative diagnosed eye concern. It is caused when an unexposed eye is exposed to the atmosphere with or without a known direct trauma. Antiseptic solution can also produce a corneal abrasion. The diagnosis is mainly made when the patient has a red eye with a sensation of a foreign body. If there is no feeling of a foreign body in the eye, then acute angle glaucoma may be a possible diagnosis, and an ophthalmologist must be contacted.

One should also be aware that orbital compression can lead to occlusion of the retinal artery blood flow and result in permanent or temporary blindness. Compression of the supraorbital nerve may also cause local numbness [6].

A group of patients with an autoimmune disease may be particularly prone to corneal abrasions [7]. Orr and Park [8] have recommended that one place an inverted surgical mask over the patient's eyes to protect them during sedation/general anesthesia. The mask with elastic ear loops appears to be the most convenient type for this technique. The contour strip marking becomes the inferior border allowing coverage of the eyes.

Lesson

ALL painful postoperative eyes should immediately be referred to an ophthalmologist [6].

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Case 80: A Maxillofacial Operation



Today you are to anesthetize an 8-year-old girl (88 lbs and 4'7'') for dental work under general anesthesia in an outpatient surgical center. The patient has developmental delay, but otherwise her past history and physical exam are unremarkable. You meet the surgeon for the first time. He is an elderly gentleman and requests that the patient gets a nasal intubation. He managed with the help of the mother to put vasoconstrictors into the right nostril, after having deemed the right to be the best opening.

With the mother in the operating room, all noninvasive monitors are placed, and a successfully inhalational induction follows. After the mother has left, you place an IV in the back of her hand. Rocuronium 25 mg is given. The mask ventilation is easy. Nasal intubation is done on the first attempt and the airway secured. No bleeding is seen. The table is turned 180°, and you hand the patient over to the surgeon.

What have you forgotten to do?

- 1. You should always ascertain what vasoconstrictors the surgeon is using, its concentration, and the expiratory date.
- 2. Then you should remind the surgeon that the recommended dose, for example, phenylephrine in children up to 90 lbs should not exceed 20 ug/kg [1]. An overdose of nasal phenylephrine can lead to life-threatening adverse events like severe hypertension with increased cardiac afterload.

Discussion

I have had frightening experiences with both adults and children who were given an unknown amount of nasal phenylephrine. One such case is presented below.

A 60-year-old man (ASA3) with CAD, HTN, diabetes mellitus, and obesity was anesthetized for a maxillofacial operation. Nasal intubation was accomplished, and the table turned 180°. I was at the machine end and doing charting, etc. and was not paying much attention as the surgeon administered nasally an unknown amount of oxymetazoline, a potent alpha-adrenergic 1 and 2 agonist. Suddenly I notice a dramatic increase in the heart rate from 68 to 115 bpm. The BP increased dramatically from 120/75 to 220/140 mmHg. His BP prior to induction was 130/85. ST segments depression were seen on the intraoperative ECG monitor. Immediate treatment with increasing sevoflurane and IV esmolol brought the BP down to normal levels over a few minutes. I asked the surgeon if he had done something to cause this increase in pulse and BP. But he said: "Nothing!" That was when the scrub nurse said: "But you gave oxymetazoline."

After the pulse and BP had stabilized, I brought in an intraoperative 12-lead ECG. It showed T-wave inversions in lead 11, 111, AVF, and V2-V-6. Based on that finding, I insisted on canceling the case. Reluctantly the surgeon agreed, and the case was aborted. His neuromuscular blockade was revered and the patient extubated and admitted to the ICU overnight. The patient was discharged the next day with normal vital signs, normal ECG, normal troponins, a clear chest x-ray, and no change in his mental status.

In the above case, the surgeon admitted that he did not know how much he had given of oxymetazoline.

This drug has replaced phenylephrine in many centers. Oxymethazoline is an over-the-counter decongestant and is available in 0.025% and 0.05%.

Lesson

- 1. It is imperative that you always ascertain in any ENT case if nasal vasoconstrictor is to be employed.
- 2. Always be sure that both you and the surgeons know the dose limits for vasoactive nasal medication.

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Case 81: A Patient with a Transplanted Heart for Cholecystectomy



You are an attending anesthesiologist in a university hospital. Today, together with a resident, you are scheduled to anesthetize a 52-year-old man for a laparoscopic cholecystectomy. He is 160 lbs and 5'10'' tall. His past history is significant for a heart transplant for cardiomyopathy 3 months previously. He is back at work in an office but claims that he has a good exercise tolerance. He is afebrile. At present he takes immunosuppression medication with a low-dose prednisone. He is also on antifungal prophylaxis (itraconazole).

You see him in the preoperative area. His vital signs are within normal limits. For sedation you give him midazolam 2 mg and 100 mg of Solu Cortef (hydrocortisone sodium succinate) as a stress dose. He is induced with fentanyl, etomidate, and vecuronium. The airway is secured, and vital signs remain stable throughout the 45-min operation.

Your resident has drawn up a mixture of glycopyrrolate 0.6 mg and neostigmine 3 mg and is about to give it.

Any comment you will give as the attending anesthesiologist?

Be very careful in giving neostigmine to a patient with a transplanted heart [1–4]. These reports mention slowing of the heart and even cardiac arrest when a cholinesterase inhibitor neostigmine is used despite concurrent treatment with an anticholinergic.

Discussion

After cardiac transplant, autonomic physiology and pharmacology are altered [5]. The most important being the interruption of cardiac autonomic innervations. In the absence of neural-mediated cardiac reflexes, the heart beats at a rate of 90–100 bpm. Luckily the incidence of posttransplantation arrhythmias decreases over time.

As regards giving neostigmine to reverse the effect of the neuromuscular blockade, most will in these cases allow time for resolution of the block. If one did use neostigmine, then one could have used a small dose of epinephrine to support the heart rate during the injection of neostigmine. Otherwise sugammadex could theoretically be used to reverse the blockade.

An alternative for neuromuscular blockade would have been a succinylcholine infusion for muscle relaxation.

Lesson

Be careful in using neostigmine to reverse the effects of a neuromuscular blockade in patients with a transplanted heart. Severe bradycardia or even cardiac arrest has been reported.

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Case 82: A High Total Spinal in an Obstetric Patient



You have placed an epidural in a 30-year-old (180 lbs 5'10", gravida 2, para 1) female in active labor. She is otherwise healthy. In her first delivery 14 months ago, the labor epidural was, according to the patient, amazing, and she had an uneventful delivery. This epidural was placed, on the first attempt, in L3/4 interspinous space using a 17 G Tuohy needle with a plastic syringe to detect loss of resistance to air. No blood or cerebrospinal fluid (CSF) were seen. A 19 G epidural catheter was easily inserted, and a 3 ml test dose with 2% lidocaine with epinephrine was negative. Approximately 5 min later, you gave a bolus injection of 0.1% bupivacaine 5 ml with fentanyl 5 microgram/ml. Within 1 min of this later dose, the patient's leg became heavy and numb. Since you felt this was too quick, you ascertain by placing ice, on her skin, that the block is rapidly ascending to T5. She now experiences shortness of breath and labored breathing.

You suspect a spinal and confirm your diagnosis by quickly aspirating CSF from the epidural catheter.

You do the following:

- 1. Call for a crash cart.
- 2. Raise the head of the bed as much you can.
- 3. Give the patient 100% oxygen with a non-rebreather mask.

Anything else you would suggest?

Do a CSF lavage.

Discussion

In a previous case report [1], a CSF lavage was performed in the following manner. Cerebrospinal fluid (40 ml) was exchanged for an equal volume of normal saline. The patient's breathing difficulties and motor block resolved quickly. A new epidural catheter was placed, after removal of the spinal catheter. Pain control was effective, and the patient delivered a healthy baby.

Cerebrospinal lavage has been shown to be effective in reversing the effects of high/total spinal anesthesia [2]. High or total spinal anesthesia commonly results from accidental placement of an epidural catheter in the intrathecal space with subsequent injection of excessive volumes of local anesthetic but is rarely considered in obstetric cases [1].

In Case 32 in this book, saline epidural injection at the end of a surgical procedure can reverse the effect of epidural anesthesia [3–5]. Hence several studies have confirmed that both epidural and spinal anesthesia can be minimized by saline epidural injection.

Lesson

CSF lavage with normal saline can be used successfully to manage a high spinal in an obstetric patient.

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Case 83: Peroral Endoscopic Myotomy (**POEM**)



Today you are to anesthetize a 41-year-old female (5'4", 44 Kg) for a minimally invasive procedure called peroral endoscopic myotomy (POEM). This technique has been shown to be highly effective in treating achalasia. She is otherwise healthy (ASA 2). You see her in the preoperative area and find her normotensive with an oxygen saturation (SpO2) of 100% while breathing room air.

She is taken to the operating room and a routine uneventful general anesthetic is induced. The general anesthetic consisted of preoxygenation, a rapid sequence anesthetic induction with IV fentanyl, propofol, and succinylcholine, and her airway is secured with an endotracheal tube. General anesthesia consists of air/oxygen and 1-2% sevoflurane. Pressure-controlled mechanical ventilation is initiated, and you note that the patient is hemodynamically stable; her SpO2 is 100%, and her initial end-tidal carbon dioxide (ET-CO2) level is 29 mmHg.

Almost immediately following the start of esophageal CO2 insufflation, her ET-CO2 levels begin to rise, reaching 52 mmHg within the first 10 min. Over the next 10 min, her peak inspiratory pressure (PIP) also rises slowly to over a 30 mmHg, from 12–14 mmHg. The PIP reached over 40 mmHg 20 min later and the SpO2 drops to 90%. ET-CO2 levels continue to rise to 75 mmHg despite all your ventilator efforts.

What will you now do?

Examine the patient's abdomen. We had a case like this not long ago [1].

The abdomen was found to be very distended and tense. Subcutaneous emphysema was clinically present over the chest and neck. The surgeon was immediately informed of these findings. The procedure was temporarily stopped while the surgeon inserted a Veress needle into the patient's abdomen to decompress the pneumoperitoneum. A marked improvement in PIP occurred immediately, and ET-CO2 began to fall without changing the ventilator parameters. The patient's SpO2, ET-CO2, and PIP improved dramatically within minutes of the intervention. Fortunately, the patient made a complete recovery.

Discussion

POEM [2, 3] involves the insufflation of CO2 into the esophagus and stomach through a flexible gastroscope. A submucosal tunnel is made from the esophagus into the stomach. When circular fibers of the muscles around the LES are identified, a myotomy is performed by electrocautery. The length of the myotomy can vary between 7 cm and 19 cm.

Esophageal and gastric insufflation of CO2 under pressure can result in pneumoperitoneum, pneumomediastinum, pneumothorax, and subcutaneous emphysema [4]. Visible transmural openings into the mediastinum and into the peritoneal cavity have been associated with these changes [5, 6]. A previous review of the anesthetic management of POEM observed minor elevations of ET-CO2 in all patients (28/28), which were treated by increasing minute ventilation [7]. This study reported a small amount of subcutaneous emphysema in the neck in one patient, but no other adverse complications were seen. In another series, subcutaneous emphysema occurred in 6/16 patients, and needle decompression of a pneumoperitoneum was performed in 8/16 patients [5]. It is of interest that neither article mentioned hemodynamic or ventilator changes associated with gas accumulation in the chest or abdomen during POEM.

Adverse effects due to CO2 insufflation are usually minimal, although this will depend upon the length of the myotomy. We have observed significant ventilator changes requiring treatment in approximately one-third of the 90 POEM procedures performed at our hospital. PIP can rise to levels that may compromise ventilation and cause barotrauma. Marked hypercarbia and related acidosis are always present. Unfortunately, if the anesthesiologist is not aware of the cause or is not monitoring the patient closely, considerable time may be lost before the correct diagnosis is made and appropriate treatment commenced.

The most effective intervention for complications of CO2 insufflation is immediate decompression of the peritoneum. Usually a simple Veress needle decompression is sufficient, but in one of our patients, this was not adequate, and a 5 mm trocar needed to be inserted. Some surgeons preoperatively prep a region of the abdominal wall in anticipating the need for intraoperative decompression.

Lesson

It is important to realize that POEM is not the only surgery that can cause subcutaneous emphysema and pneumomediastinum. It can occur, for example, during dental extraction [8], laparoscopic surgery [9, 10], and during CPR in a patient with a duodenal ulcer [11]. Prompt recognition of this complication is imperative to prevent a disaster.

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Case 84: A Neonatal Emergency



A neonate with gastroschisis has been admitted. Besides the gastroschisis the child has an extremely low birth weight (ELBW) but does not have Down's syndrome or any cardiac anomalies. The child is brought to the operating room, and you provide a routine general anesthetic. The child is doing well. With your assistance, the anesthesia resident places a percutaneously central line. Through this line you hydrate the child with appropriate IV fluids.

The operation starts. About 15 min later, the child is exhibiting evidence of cardiorespiratory instability (bradycardia, hypotension, low saturation, and cyanosis). You diagnose cardiac tamponade, and without waiting for a transthoracic echocardiography, you instruct the surgeon to perform a subxiphoid pericardiocentesis. The vital signs improve as he aspirates fluid, but to everyone's surprise the aspirate is clear fluid and not blood.

What should be done? Stop aspirating? Anything else?

Keep aspirating and remove the central line.

Discussion

Cardiac tamponade must always be considered after a central line catheter has been placed. This is especially true in low birth weight neonates [1].

In this case, aspirating the fluid from the pericardial sac and removing the central line saved the day.

This case happened to a very good friend of mine, Professor Ray Fitzgerald, who at the time was a pediatric surgeon in a university affiliated hospital in Dublin, Ireland.

Pericardial effusion and cardiac tamponade are rare but life-threatening complications of percutaneously inserted central line (PICL) especially in extremely low birth weight (ELBW) neonates, with an incidence reported between 0.07% and 2% of PICLs placement [1]. Timely diagnosis and in this case pericardiocentesis.

Lesson

Cardiac tamponade should be considered in any newborn with a peripherally inserted central catheter who presents with cardiorespiratory instability (bradycardia, cyanosis, and metabolic acidosis), even when lines are believed to be placed correctly.

Reference

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Case 85: This Could Be Serious



You are an attending anesthesiologist in a university hospital. Today you are anesthetizing a 10-year-old girl for tonsillectomy and adenoidectomy. A medical student is with you, and after an uneventful anesthetic induction and endotracheal intubation by the medical student, the table is to be turned 90° . This is done to facilitate the surgery.

You disconnect the endotracheal tube at the wye, the medical student who is standing beside you grabs the head of the table. He starts to pull the operating room table.

Should you be concerned?

I have seen the head attachment of an operating room table come off when pulling on the headpiece. The result was that the patient's head dropped to an extremely extended position before I could catch the head.

Hence never rotate an operating table by holding the headpiece of the table. Always push the table around from the side. After it is in the 90° position, you can move the table toward you or away from you by pulling or pushing.

Discussion

Should this happen to you, then you must quickly get the head in its correct position to the body and then reposition the head on the headpiece. Make sure the fixation screws on the headpiece are tight. Cervical and upper thoracic spine radiography could be performed. After that the patient should be awaken to ascertain that all four limbs are seen to be moving. Then you should call for an emergency neurosurgical consult and speak to the family. In my case the patient was re-anesthetized after the neurologist had ruled out a serious injury. The surgery was performed without any more mishaps, and the postoperative course was uneventful. This problem has been discussed by Szmuk et al. [1].

It is interesting to note that most operating room table manufactures do not recommend moving the table while holding only the headpiece. Besides cervical spinal cord injury and head trauma, a potential extubation may occur.

Lesson

Never move an operating room table by holding on the headpiece.

Reference

 Szmuk P, Abramson D, Warters DR, Pereira K, Katz J. Accidental extreme neck extension during repositioning of an operating room table. Anesthesiology. 2001;94:940–1.

Case 86: A Case of Acoustic Neuroma



The removal of a left-sided acoustic neuroma in a 30° head up, left tilt, semi-prone position has just been concluded. The operation has lasted 8 h. There was no bleeding seen during the closure of the dura. The patient is a 55-year-old lady (200 lbs, 5.4 inches, ASA 2). She has a 3-year history of opiate use for osteoarthritis of her knees. A total of 20 mg of morphine IV has been given and the last dose of 2 mg was 90 min ago. The patient has had a total of 400 microgram of fentanyl which was given on induction. The isoflurane was turned off 20 min before the end of the surgery. The patient is allowed to wake up and the nitrous oxide and the remifentanil drip 0.5 microgram/kg /min are turned off. Unfortunately, there is no spontaneous breathing, and the patient is unresponsive with pinpoint pupils. She is still in the operating room intubated with assisted ventilation. Adding to your concern is the fact that the patient's blood pressure, over a 20 min period, is now trending upward to 190/120 mmHg from 140/80 mmHg.

Is it time for naloxone?

Do not give naloxone. Get an urgent computerized tomography scan.

Discussion

Postoperative unresponsiveness with pinpoint pupils can be caused by a hematoma (especially in the cerebellopontine region), pontine reticular formation compression, or intraoperative administration of opiates [1–4].

In a similar case report [4] as above, the authors obtained an urgent scan. The scan revealed a clot at the cerebellopontine angle. The clot was emergently evacuated under general anesthesia. Afterward the patient was left intubated and ventilated in the neurosurgical ICU. The patient recovered without major sequelae.

There would have been a temptation to give naloxone in this case. Although it could have expedited the diagnosis, naloxone could have resulted in further hypertension, tachycardia, and a sudden and uncontrolled awakening. This could have exacerbated the hematoma.

In similar cases where there is a slow awakening with pinpoint pupils, it is better to avoid naloxone even if the clinical signs suggest an opiate overdose. Instead you should opt for prolonged ventilation while you wait for the potential opiate overdose to dissipate. If a definite diagnose is required, then an urgent scan should be done.

Lesson

In cases like this, an urgent computed tomography scan may be less risky and more informative than giving naloxone.

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Case 87: Is the IV Infiltrated?



You, as an attending anesthesiologist in a small rural hospital, are called to the ICU at 5:30 a.m. Here you find a 4-year-old child (50 lbs, 4'4", ASA 2) on full ventilator support. She was an unrestricted passenger involved in a MVA. She was admitted with a Glasgow Coma Scale of 2. On arrival in the emergency room, she was immediately intubated and ventilated. A CT scan shows that she has had an intravascular bleed. The child is heavily sedated with morphine and paralyzed with vecuronium. Her prognosis is guarded.

The nurse is concerned that the only peripheral IV is not working as it is dripping slowly. She and others have made several attempts at IVs, but all have failed. The nurse wonders if this is not the time for a central line.

The IV is in the right antecubital fossa. The arm is fully extended with a wooden splint and bandaged. Hence you cannot see the IV site. You take the bandage off but find it dry. There is minimal swelling over the IV site. The nurse says she has attempted to flush and aspirate blood from the IV line but to no avail. You know that these two methods are not accurate as children have compliant tissues and you may not see extravasation of IV fluids. Also children have small vessels and in this case the child is most likely dehydrated.

You look for other possible IV sites but see none. Actually the child looks a bit like a pincushion. Your experience in placing central lines in small children is minimal, and you would prefer to call in one of your pediatric anesthesiologists to do it. The person you are thinking about is due to arrive at the hospital in 1 hour.

Are there any other tests you can do to verify that the present IV is in the right place?

Injection of 2.1% sodium bicarbonate has been shown to verify the correct placement of an IV line. This is done by observing an increase in end-tidal CO2 [1].

Discussion

Infiltration into the subcutaneous tissues by a mal placed IV catheter is one of the most common complication of IV vascular access. If fluid can extravasate, so can drugs. Both can cause injury. The incidence of peripheral IV infiltration has been reported to be 23-28% in neonates [2] and 11-28% in children [3, 4]. This is in contrast to percutaneous central venous catheters that have a much less infiltration rate of 1-16%. Infiltration of drugs can cause tissue necrosis with sloughing in up to 44% of infants when this occurs [5].

It is important to realize that repeat IV cannulation has been reported as the most distressing part of hospital ax mission and the cause of the most pain while in hospital [6]. Hence any test that can confirm the correct placement of an IV catheter will be an advantage for all concerned.

The injection of 2.1% sodium bicarbonate in the study above [1] did not have any clinically significant effects on blood pH, bicarbonate, or sedum concentration.

Indigo carmine has also been recommended to detect correctly placed IVs. The ability to see the flow of the IV dye intravascularly helped to confirm the correct placement in this case report [7]. The latter technique was stated to be quick, safe, and inexpensive.

Lesson

Using sodium bicarbonate or indigo carmine can help to confirm the correct placement of an IV catheter should there be any doubt.

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Case 88: Communication Is Essential



You are working as an anesthesiologist in a medium-size rural hospital. The hospital oxygen pipeline is to be closed down over the weekend. The reason is that the existing hospital pipeline is being joined up with the oxygen pipeline in the new hospital building which is adjacent to the original one.

It is now 54 h after the two pipelines have been attached and 200 oxygen outlets are declared safe and working by the bioengineering department. It is a Monday morning. Even though you have been told that oxygen pipeline is declared "safe," you are extra careful with your machine check. You find the oxygen pipeline pressure to be normal, and you ascertained that 100% oxygen is flowing from the oxygen rotameter. You breathe in the oxygen from the face mask to ascertain that it is N₂O. The inspired oxygen monitor says 100%. As you are a very good and careful anesthesiologist you make sure that you have two full oxygen tanks attached to the back of your anesthesia machine.

It is now 9 a.m. on a Monday morning, and you are concluding a routine general anesthetic for a gynecological procedure in a healthy female. Suddenly both the patient's saturation monitor and the inspired oxygen monitor fall precipitously. You activate your oxygen cylinders and the patient wakes up unharmed. At the same time, you inform the front desk, to warn your colleagues in the other six operating rooms that there may be a problem with the oxygen supply. As it turns out, the other operating rooms have no problem.

But just to be sure, all ongoing anesthetics are now on oxygen cylinders. The oxygen outlets in the operating suites are immediately checked by engineers. They find no faulty and declare the oxygen pipeline to be safe. All operating rooms use the piped oxygen and luckily no other adverse events occur that day.

The next morning, you find yourself in the same operating room and again a similar event occurs. With the same procedure as yesterday, everything goes well, and the patient wakes up unharmed.

You call on the engineers and tell them what happened, but this time you tell the engineers what to do.

They must do a quality control, not just pressure testing the system.

In a previous case similar to the one reported above [1], this is exactly what happened.

So when the engineer says the pipeline is safe, you have got to make sure you understand what is meant by "safe." What has been tested?

Discussion

In a previous report [1], the engineers were not asked to check the pipeline for quality and thought they were only asked to check the pressure.

In that case [1], the mostly likely cause was air entering the oxygen pipeline which could not have been flushed out properly with 100% nitrogen. When purging with nitrogen, it is important to use a particulate filter to test all outlets of the system. The filter should be sent to the lab to be analyzed for purity (personal communication Marcus B. Picou, Pneumatic Tube System Technician, Stanford Health Care, Stanford, CA 94305).

Lesson

Acute failure of oxygen delivery does occur [2, 3]. When it happens, it is imperative to know how to keep the patient safe and then inform the engineers what is to be tested. Communication is all important.

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Case 89: Watch Out



Today you are working with a very keen medical student. Your patient is a 6-yearold male scheduled for a knee arthroscopy (25 kg, 4'11", ASA 1). Your anesthetic plan is to use an LMA and have the patient breath spontaneously. He is anesthetized with propofol and is easy to ventilate without a Guedel-styled airway. You ask the medical student to attempt to ventilate the patient, but he is not able to do so satisfactorily. You take over and you have mores success. Thinking that the student will be able to ventilate better with a Guedel-styled airway, you ask him to open a number 2 Guedel airway from its plastic cover (Portex Ltd., Ashford, UK). He rips the cover off and you ask him to insert the airway. Unfortunately, despite the fact that the airway looks correctly placed, the student still cannot ventilate the child. You take over, but you cannot ventilate the child either. You remove the airway and find that it is now easy to ventilate the child.

What can the problem be?

On close examination of the Guedel airway, you find that part of the airway plastic cover is stretched tightly over the end and is thereby obstructing the airway lumen.

Discussion

This dilemma has been reported [1]. The plastic cover which obstructed the lumen of the Guedel was thought to be caused by the medical student, when he removed the airway from its plastic cover. Asked to do it again, he was seen to use the sharp end of the airway to break open the plastic. The result was that the plastic stretched until it became very fine, transparent, and almost invisible. This film therefore blocked the airflow (think of wrapping food in Glad Wrap). The author [1] recommends that the plastic which covers the airway should be opened with a scissors.

Lesson

Plastic covering of an airway should always be opened with a scissors.

References

1. McNicol LR. Unusual cause of obstructed airway in a child. Anaesthesia. 1986;41:668-9.

Case 90: A Simple Case but It Goes On and On



You are a senior anesthesiologist in a university hospital. Today you are working with a resident who has been with the program for 2 years. Of interest is that you have been asked by the Anesthesia Department's Clinical Competence Committee to work solely with him for a week and to give a report. You have been told by your colleagues that the resident is very much behind his peers in clinical skills, etc.

The first patient of the day is a 28-year-old ASA 1E (190 lbs and 6'1") scheduled for an open reduction internal fixation (ORIF) of his left humerus. He has no other health issues and is a triathlete of note. The surgeon requests no peripheral nerve block. You hear the surgeon tell the resident that this case should only take 1 h. You have worked with this surgeon before, so you are a bit suspicious, but say nothing.

You tell the resident that this week you will be observing him only; no help will be given unless he gets into trouble or asks for help. You also tell him that you will give him feedback at the end of each day. The resident accepts this modus operandi. The patient is brought to the operating room after an IV is started in his right forearm and 2 mg of midazolam is given IV. The oximeter is place on his right ring finger. Three-lead ECG is placed in the correct places. The blood pressure cuff is placed in the middle of his right calf. You note that he does not wrap any Webril which is an under cotton padding (Medline Industries Ltd., Chicago, IL) under the cuff. But since the case is scheduled to be under an hour, you say nothing.

The anesthetic management is acceptable. The patient is now asleep and turned into the right lateral position. An axillary role is placed in his right axilla. His right arm is placed on a board and the arm padded. The right elbow is flexed slightly. You note that a small pillow has been placed between his two legs. The noninvasive BP cuff is set to cycle every 2.5 min. The patient's eyes are taped and there is no pressure on them. The table is turned 90°, and the resident sits beside the patient's right arm and is facing the patient's face.

Anything wrong with this picture? If so what would you do?

If you think the operation could be long, then it will be long. You must prevent the possible development of a postoperatively compartment syndrome to his right leg [1-4].

Discussion

In fact, the above operation took over 4.5 h, and the patient developed compartment syndrome to his right leg (personal communication; Anna Harter, Meghana Yajnic, and Austin Schwab).

Here is a list of suggestions that could potentially have prevented this complication. Points in no special order:

- 1. Place a big pillow or pillows between the legs.
- 2. Attach a BP cuff on the upper leg, not the lower as in this case.
- 3. Attach a BP cuff on right arm and alternate BP measurements between arm and leg every hour.
- 4. Only check BP every 5 min on the leg, unless clinical indication should necessitate more measurements.
- 5. Consideration could have been given to an arterial line. This is especially true after 4 h and with no end in sight.
- 6. Place Webril on all BP sites if operation lasts over 5 h.

Webril is used by anesthesiologists to protect the skin from being pinched by the BP cuff. It is especially beneficial for obese and elderly patients. Also, it "rounds up" the shoulder profile, which makes application of BP cuff more secure, preventing it from slipping off to the antecubital fossa. Although debated, one of the proposed mechanisms for ulnar nerve injury (the commonest iatrogenic anesthesia-related complication) is a low BP cuff position, making it cycle against the ulnar groove. The claim that Webril can minimize the development of postoperative compartment syndrome is speculative at best.

Finally, since the cleaning procedures for BP cuffs are frequently subpar, putting Webril under the BP cuff is an obvious solution.

Lesson

If you think an operation will be long and there is a possibility of developing a compartment syndrome, then make every effort to prevent it.

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Case 91: Endotracheal Intubation in the ICU: Watch Out



You as the attending anesthesiologist are called to the ICU at 21:30 where a 66-yearold man with ankylosing spondylitis needs an emergent intubation.

He had been admitted to the ICU 2 days ago after he had undergone an emergency uncomplicated posterior decompression and fusion, including T9 to T11 laminectomy. The reason for the operation was that he fallen on ice and sustained an unstable thoracic fracture. There were no cervical fractures, subluxation, or any other acute findings identified on the initial cervical CT. You look at the notes and discover that there was no problem with the endotracheal intubation at the operation. The posterior part of the vocal cords was seen. Your airway assessment concurs with the previous anesthesiologist findings.

After the operation, the patient was extubated and taken to the ICU. On arrival in ICU, he was able to move all extremities. Unfortunately, on the second day, he developed a decreased mental status associated with decrease in oxygen saturation. It is at this point that you are now called to secure the airway.

With the above history, what is your plan to secure the airway? Will you use your favorite laryngoscope or do a fiber-optic intubation? If you elect to do the latter, why?

Do a fiber-optic intubation.

Discussion

In a previous similar case report, the anesthesiologist used his favorite laryngoscope [1]. It turned out to be a very difficult intubation despite manipulation of the head, etc. Fiber-optic intubation was then successfully performed. Unfortunately, the patient woke up with a C6 quadriplegia. Imagining showed a C6-7 fracture-dislocation associated with severe central canal stenosis.

In the case report [1], two similar cases were also described. Unfortunately, both developed quadriplegia after intubation in the ICU. Although exceedingly rare, catastrophic neurological decline may result from endotracheal intubation of patients with preexisting cervical spine disease [2, 3].

I am grateful to my friend Dr. Patrick Bolton, a previous chief resident at Stanford, for pointing this case out to me.

Lesson

In a case like this, always do a fiber-optic intubation with minimal movement of the neck. Although this intubation technique may not prevent paraplegia, you are seen to be aware of the problem and have attempted to prevent this devastating complication.

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Today you are to anesthetize a 4-year-old child for an elective orchidopexy. You meet the mother and the child in the preoperative area. The mother tells you that he is on a waiting list for an adenotonsillectomy (T/A).

In the interim, it is the urologist preference to have the orchidopexy done now. On examination the child is seen to have adenoidal facies. The tonsils are markedly enlarged and actually "kissing each other" in the midline. He has cervical lymphadenopathy and bilateral secretory otitis media. The rest of the exam is normal, especially the chest is clear, and he has no pyrexia. On direct questioning, the mother admits that the child snores while he is asleep, and she thinks he has periods of sleep apnea. The surgeon has listened to this interchange and realized that you are hesitant to give a general anesthetic to this patient. He knows you are skilled in pediatric caudal block and suggests minimal sedation with a caudal block for this procedure. The mother, who has taken off work to be with her child, feels that the operation is necessary. She is unhappy that her child is not receiving a general anesthetic.

With the above history what would you do? Cancel or proceed? If you proceed, what would you do?

I would cancel.

Discussion

In a similar case as described above [1], a child developed acute upper airway obstruction after a routine general anesthetic for orchidopexy. The child required an emergency adenotonsillectomy.

The authors [1] theorized several reasons for the sudden onset of airway obstruction. The drying of the oropharynx caused by preoperative starvation, the use of un-humidified anesthesia gases and the postoperative sedative effect of the premedication and general anesthetic drugs. Also the Guedel airway could have caused minor tonsillar trauma. The airway was used throughout the anesthetic with the patient breathing spontaneously on 50% nitrous oxide in oxygen with sevoflurane. No LMA or intubation with an ETT was performed.

Lesson

Enlarged tonsils and/or adenoids with possible obstructive sleep apnea should always be a concern for the anesthesiologist when evaluating a child for an elective operation.

References

1. Livesey JR, Solomons NB, Gillies EAD. Emergency adenotonsillectomy for acute postoperative upper airway obstruction. Anaesthesia. 1991;46:36–7.

Case 93: Postoperative Red Urine



A 10-year-old boy (40 lbs) has arrived in the recovery room. The time is 4 p.m. The anesthesiologist hands over the postoperative care to you and leaves. He tells you that the patient has had an uneventful general anesthetic for a strabismus surgery. A previous anesthetic at age 6, for his Achilles tendon, was also uneventful. Nitrous oxide 50% in oxygen with sevoflurane, with the patient breathing spontaneously via an LMA, was given on both occasions. A grandmother is at the bedside and tells you, the anesthesiologist, that the child is healthy. The child's vital signs are normal and he is sleeping peacefully. Since he is scheduled to go home on the same day, one of the criteria for discharge is that he voids before he leaves.

Two and half hours later you are called by the recovery room nurse, as the patient has just passed urine. But the urine is bright red. You check the vital signs and find them within normal limits (HR 100 bpm, BP 130/70 mmHg, temperature axilla 37.5 C).

A urine dipstick is positive for hemoglobin. An urgent urinary microscopic analysis is negative for red blood cells, thereby excluding hematuria.

What other test would you order in this case?

Blood test for creatine kinase (CK), lactate dehydrogenase, aspartate aminotransferase, and alanine aminotransferase.

Discussion

All the blood tests above came back with abnormally high levels. Hence the association of elevated creatine kinase, liver enzymes, and red urine led to a diagnosis of rhabdomyolysis with myoglobinuria. Hemoglobin, bilirubin, and serum potassium were all within normal limits. This case is akin to a previous case [1]. Postoperative work-up showed that the patient most likely had Becker muscular dystrophy (BMD).

Red urine postoperatively has several possible causes:

- 1. Hemoglobinuria (hemolysis)? From, for example, a blood transfusion reaction.
- 2. Hematuria (bleeding)?
- 3. Myoglobinuria (rhabdomyolysis)? Myoglobin appears in the urine when the serum concentration goes over 1.5 mg/dl and becomes visible when the urine concentration is greater than 100 mg/dl. Rhabdomyolysis can be caused by malignant hyperthermia. It could also reveal a metabolic or muscular disease [2–5] that could be complicated by hyperkalemic cardiac arrest [4].
- 4. Dye which is present in medication or food.
- 5. Porphyria. However, in that case the red color appears only when the urine is exposed to air.
- 6. Urological surgical trauma.

Urine can be red but arterial blood can be dark green [6]. This was seen in a patient admitted to a hospital with a compartment syndrome in both legs. He had blood creatine kinase concentration of 43,384 U/L, and the arterial blood was dark green.

Lesson

Unusual past surgical history in pediatrics (in this case the operation on an Achilles tendon) should always alert the anesthesiologist to the possibility of an underlying neuromuscular disorder.

An abnormal color of both urine and blood requires your urgent attention.

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Case 94: Patient's Toes Suddenly Become White During a Lower Limb Operation



A 30-year-old man (70 Kg, 5'11', ASA 1 E) is admitted to the hospital as an emergency at 8 p.m. You see the patient in the preoperative holding area. He is scheduled for excision of an infected sinus in his left lower tibia. He has not eaten for 8 h and his vital signs are within normal limits. He refuses a regional block and requests a general anesthetic. Previous anesthetic for an orthopedic procedure on his left tibia was uneventful.

A routine general anesthetic is given, and the patient is breathing spontaneously through a Mapleson A circuit with nitrous oxide 70% in oxygen and 1% sevoflurane. Meperidine is titrated to respiratory rate of 8–12 breaths per minute. A total of 80 mg is given. The operation is done under a tourniquet, 250 mmHg pressure. At the end of the procedure, the bony wound canal is irrigated with 20 ml of hydrogen peroxide, and a small drain is inserted. The tourniquet is released. While the surgeon dresses the wound, the drapes are removed. Within seconds of the removal of the tourniquet, the surgeon notices that the toes #1 and #2 go suddenly completely white. The surgeon also thinks he sees gas bubbles in the veins of the dorsum of the foot. Heart rate, BP, respiratory rate, and oxygen saturation remain unchanged. There is a decrease in end-tidal CO2 from 45 mmHg to 32 mmHg.

Are you concerned? If so, what if anything will you do?

Reapply the tourniquet; turn off the nitrous oxide; place the patient in left lateral position with the head down.

You could have auscultated the heart to listen for a "mill wheel" murmur. I personally have never been convinced that I have heard one.

Discussion

In a previous case report [1], a similar patient suffered from arterial and venous gas embolism caused by hydrogen peroxide. Luckily the patient made an uneventful recovery, mainly thanks to the quick action of the anesthesiologist. In the case report [1], the authors heard the classic murmur, caused by gas embolism, when auscultating the patient's heart.

Although rare, the anesthesiologist must be aware of this possible complication and institute rapid preventative measures as indicated above. In the case described by Neff et al. [1], after the above maneuvers, the mill wheel murmur disappeared within the period of 3 min. Within 10 min, the end-tidal CO2 increased to normal levels before the gas embolism. The venous embolism in the veins of the dorsum of the foot disappeared. At that time, the tourniquet was gradually released again. No change in end-tidal CO2 or no abnormal murmurs were heard. The color of the toes improved and the patient made an uneventful recovery. At no time did the EKG show any signs of right ventricular strain.

If the above patient had a persistent foramen ovale or an atrial or ventricular septal defect, then 0.5 ml of air can cause serious damage to the heart and brain [2]. Although in the absence of any cardiac defect, gas may still get access to the pulmonary artery and to the systemic circulation via pulmonary shunt vessels [3, 4].

Nitrous oxide increased the size of any gas bubble by two- or fourfold with 50% or 75% nitrous oxide, respectively. This increase could make a dangerous situation even more precarious.

Lesson

If you think you have a gas embolism, then you must quickly do all the preventative maneuvers that you can. Turn off nitrous oxide, place the patient in a left lateral position with the head down, and if applicable reapply the tourniquet.

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Case 95: A Percutaneous Tracheostomy



You, as the anesthesiologist on call, are called to the ICU where a 68-year-old woman (170 lbs 5'7'') is breathing spontaneously through a percutaneous dilatational tracheostomy [1]. The latter has been performed 3 days ago. Her Glasgow coma scale is 8. Her airway is considered normal and she has no dentition.

The nurse tells you that she is sucking blood via a suction catheter (14 F, CareFusion, Yorba Linda, CA 92887), both from the tracheostomy tube and from the oral cavity. The amount has increased in the past hour to 2–4 ml/h from the tracheostomy tube and 10–15 ml/h from the oral cavity. The patient is not on heparin or any other anticoagulants. The nurse has been instructed to compress the pretracheal tissues for 20 min, but no improvement has been seen.

The ICU attendant, who is an internal medicine specialist, requests that you remove the tracheostomy tube, perform an oral-tracheal intubation, and inspect the tracheal stoma. You confirm that the consent for this procedure is signed by the next of kin.

How, in detail, will you do this?

My suggestion would be to take the patient to the operating room (OR), with an ear-nose-throat (ENT) specialist available. In the OR you should adequately sedate and paralyze the patient. While ventilating the lungs via the tracheostomy, you should place the suction catheter (14F) through the glottic opening. This is to remove any blood or especially blood clots above the tracheostomy tube and cuff. Since the suction catheter is soft, place it in ice for a few minutes, and bend it like an ETT for easier insertion into the trachea. After that a flexible bronchoscope should then be inserted to see how successful your suction was. If no clots are seen, then the tracheostomy tube can then be removed. If the bleeding is minimal and localized, then the pressure of the endotracheal tube (ETT) cuff should be able to stop the bleeding. Otherwise you may want to refer to your ENT colleague.

Discussion

In a previous case report [2], the suctioning above the tracheostomy tube was not done. After the tracheostomy tube was removed and the ETT placed, it became impossible to ventilate the patient. A rigid bronchoscope saved the day by sucking out a large solid thrombus obstructing the right main bronchus and most of the left.

The authors [2] theorized that after the tracheal tube was removed, the thrombus above the tracheal tube got pushed down into the lower trachea by the ETT, causing an airway obstruction. They recommend that in cases like this one should use a flexible bronchoscopy to inspect the subglottic region for any possible thrombus formation prior to the removal of a tracheostomy tube.

The bleeding caused in this case is specifically thought to be related to percutaneous dilatational tracheostomy [2]. This is believed to be due to the extremely tight fit of the tracheostomy tube and the wound edges that adhere firmly to the cannula. Blood can then accumulate above the cuff and can form a clot.

Lesson

Changing a tracheostomy tube for an oral endotracheal tube can be fraught with danger. This is especially true if bleeding has occurred above the tracheostomy tube, potentially forming a blood clot that can obstruct the trachea when the ETT is inserted.

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Case 96: A Patient in the Prone Position – Watch Out



Today you are anesthetizing a 38-year-old woman (200 lb, 5'4", BMI 33, ASA 2) for stereotactic craniotomy in a prone position without neuro-monitoring. The patient has a class 1 airway.

A routine anesthetic is employed with N₂O 50% in oxygen with 1% isoflurane, remifentanil infusion 0.05 μ g/kg/min, and vecuronium. A 7.5 standard endotracheal tube (ETT) is inserted atraumatically into the patient's trachea and secured at 22 cm at the teeth using benzoin and tape. An esophageal temperature probe is inserted. The stereotactic head ring is applied to the patient's head using previously placed twist drill holes. Thereafter the patient is turned prone on a 3" foam mattress and supported transversely placed shoulder and hip rolls. The head ring is locked into position to the attachment at the end of the operating table. Before the patient is handed over to the surgical team, you ascertained that there are at least two fingerbreadths between the mentum and the sternum, bilateral air entry, a normal peak airway waveform with a pressure of 38 cm H₂O, and a normal end tidal CO₂ waveform at a peak of 28 mm Hg. The increase in the peak inspiratory pressure (PIP) is thought to be due the anterior compression in this obese patient.

Anything else you would do before handing over to the surgeons? If so what?

These patients must have a secure molar bite block (BiteGard, HudsonRCI, Teleflex, Research Triangle Park, NC 27708).

Discussion

In a previous case [1] like the one described above, the operation proceeded uneventfully for about 1 h when there was gradual increase in PIP from 50 to 60 cm H₂O. Manual ventilation proved very difficult. The end-tidal pCO₂ rose also from about 28 to 44 mm Hg, but the oxygen saturation remained stable at 99%. Train-offour stimulation of the ulnar nerve revealed one twitch. Inspection of the ETT at the mouth revealed that the tube was compressed between the patient's upper and lower teeth, resulting in a near occlusion. The mouth could not be opened. The surgeon was notified and found no slippage of the head pins. The head frame was then disengaged from the operating table with no relief of the airway obstruction. The wound was covered and the drapes removed before turning the patient supine. It was at this point one could see the cause of the dilemma. The patient's torso had slid cephalad on her panniculus in relation to the head ring table attachment. No fingers could be inserted between the mentum and the sternum which then led to the "biting down" effect. The table was put in a reverse Trendelenburg position and the patient repositioned and a BiteGard was inserted. The rest of the operation was concluded uneventfully.

It is imperative to have a bite block in prone patients. A bite block is preferred to a standard Guedel airway, which is not recommended in these cases, since the Guedel can damage the front teeth, compress the uvula and other soft tissues in the mouth, and may result in postoperative macroglossia with serious postoperative airway obstruction.

Some anesthesiologists use an armored ETT in these cases. However, the patient can bite down on an armored ETT [2, 3]. When that occurs the ETT can be completely occluded and the lungs cannot be ventilated. The solution is to manipulate the ETT into its original shape by compressing a hemostat at 90° to the occlusion. This simple method can rectify a potentially dangerous situation without having to resort to changing the ETT.

Obstruction of an ETT can have many causes, mucus, blood clots, kinking [4], flexing the head excessively on the chest [5], herniation of ETT cuff into the ETT lumen [5, 6], biting on the ETT [1, 7], kinking in the nasopharynx due to excessive flexion of the head, and an overinflated ETT cuff covering both the outlet of the ETT and the Murphy's eye.

Korn et al. [1] recommend, when lying prone, these obese patients should be placed in a slightly reverse Trendelenburg position. Cephalad movement may lead to main stem intubation and hypoxemia.

Lesson

A molar bite block like BiteGard should be used in all prone anesthetized patients. A slight to moderate reverse Trendelenburg is recommended in obese patients placed in the prone position.

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Case 97: A Patient with Obstructive Sleep Apnea



Today you are anesthetizing a 54-year-old man (130 lbs, 5'11", ASA 2) for a prostatectomy. He has obstructive sleep apnea (OSA) and uses a CPAP at home. You are with a medical student. The patient is taken to the operating room where routine monitors are placed. Induction of anesthesia is uneventful and the medical student successfully secures the airway with an endotracheal tube. Thereafter mechanical ventilation commences. You let the medical student draw up more fentanyl and tell him to give the patient 100 μ g. As he is about to inject the fentanyl, you notice that there are a few small air bubbles in the syringe.

Normally you would not worry, but in this case you tell him to stop the injection. Are you being unnecessarily cautious?

There is a recorded 72% prevalence of a right-to-left shunt (RLS) in OAS patients [1] compared to the general population, where the incidence is about 9% [2].

Discussion

The high prevalence of RLS in OAS necessitates scrupulous attention to IV management. Even a small volume of air entering the left side of the heart (paradoxical embolization) has the potential to obstruct portions of the cerebral or coronary circulation, with possible disastrous consequences [3, 4]. The inadvertent introduction of small amounts of air (<5 ml) into the venous system through an IV is fairly common, but is not considered clinically significant because of the small volumes and because of the filtering capacity of the lungs.

Paradoxical embolization is believed to occur most commonly through a patent foramen ovale (PFO) [5]. Autopsy studies have confirmed that up to 34% of the adult population has a probe-patent FO [5]. Coughing and/or Valsalva's maneuver are known to cause rapid changes in intrathoracic pressure associated with reversal of the interatrial pressure gradients [6]. The shunting of blood through a PFO can also result from intraoperative mechanical ventilation and/or the steady application of positive end-expiratory pressure (PEEP) [4].

Lesson

Patients with obstructive sleep apnea have a higher incidence of right-to-left shunts. It is essential that there are no air bubbles in IV fluid administration.

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Case 98: A Case of Wegener Granulomatosis



A 38-year-old female, BMI 50 kg/m², 4'11", ASA 2, was scheduled for a Rouxen-Y gastric bypass. Her past history was significant for Wegener granulomatosis and asthma. The former was treated 2 years prior with prednisone, azathioprine, and antibiotics. At that time her spirometry showed a mild flattening of the inspiratory limb of her flow-volume curve. Since her asthma was successfully treated with albuterol and mometasone furoate inhalers, no further pulmonary investigations were carried out. A chest x-ray done 1 week prior to the surgery was normal.

She was seen on the day of surgery. Mild wheezing was heard over both lung fields. She reported no exercise tolerance and complained of shortness of breath with even minimal physical exertion. This she attributed mainly to her weight. Her oxygen saturation on room air was 99%. Her routine laboratory results were normal.

A routine general anesthetic was induced with propofol and succinylcholine. She was easy to mask ventilate. A grade 2 view was seen, but a #7 ETT would not pass beyond the vocal cords. A gum elastic bougie was inserted into the trachea without any difficulty, but #7 ETT could not be advanced over it into the trachea. Throughout the intubation attempts the vital signs remain stable.

Now what will you do? Try a smaller ETT or what?

With the patient still anesthetized, the gum elastic bougie was removed and an LMA inserted. Through the LMA the lungs were easily ventilated. A fiber-optic bronchoscopy was performed through the LMA and revealed a subglottic stenosis, 6 mm below the vocal cords and extending 2 cm. An otolaryngologist was called.

Discussion

It is worth noting that tracheal stenosis is extremely common (25%) in Wegener granulomatosis [1-3].

A similar case as the one described above has been reported by O'Hear et al. [4]. The subglottic stenosis was considered so extensive by the otolaryngologist that a tracheostomy was performed in the operating room and the surgery cancelled. She was admitted to the hospital and, on the third postoperative day, she underwent a successful carbon dioxide laser excision of her subglottic stenosis. Four months later, she underwent a successful bariatric surgery.

Wheezing is common in morbidly obese patients and is usually attributed to a combination of asthma and reduced lung volumes [5]. Tracheal stenosis can be misdiagnosed as asthma [6].

Therefore, subglottic stenosis may not be easily recognized in a morbidly obese patient who avoids most physical activity and has no evidence of stridor. In the above case [4], the mild flattening of the inspiratory limb of her flow volume curve, noted 2 years previously, should have alerted the physicians to a possible upper airway involvement.

Lesson

In patients with Wegener's granulomatosis, the presence of a tracheal stenosis must always be considered. This is especially true if the patient has asthma and has limited physical activity.

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Case 99: What Can Possibly Go Wrong?



Today the ear-nose-throat (ENT) surgeon tells you that the last case of the day is to be done under local anesthesia with some sedation. You, the anesthesiologist, agree. The patient has the following comorbidity: coronary artery disease, obesity, peripheral vascular disease, chronic obstructive lung disease, and hypertension. Recently, she had undergone a triple coronary bypass grafting, atrioventricular node ablation, and repair of an abdominal aneurysm.

The patient is 65 years old (260 lbs, 5'4", BMI 45, ASA 3) and is scheduled for a repeat palatal biopsy. She is placed on the operating room table. Her vital signs are HR 86 SR, BP 155/96 mm Hg, and oxygen saturation 92% on room air. You tell the surgeon that you are somewhat concerned about her low oxygen saturation. He suggests that you use the Optiflow (THRIVE) (Fisher and Paykel Healthcare, Auckland, New Zealand). This he tells you will provide 100% oxygen in spontaneously breathing sedated obese patients [1]. The surgeon states he has been able to keep patients at 100% saturation when using Optiflow. He has brought it along to the operating room for this case. Since you have not seen or used the Optiflow, the surgeon attaches the equipment and starts the high flow of nasal oxygen. With this patient's history, you decide not to give her any sedation but reassure her by talking to her. The surgeon injects 2 ml of lidocaine 2% with 1:80,000 epinephrine in the roof of the patient's mouth.

Is there anything wrong with this picture?

Intraorbital fire can occur even with a needle diathermy [2].

Discussion

To cause a fire, three factors are needed. They are the oxidizer (oxygen), an ignition source (laser or diathermy, etc.), and a source of fuel. Airway fires during laser surgery are well documented, but fires caused by diathermy are much less common. But ASA Practice Advisory on operating room fires [3] states that electrosurgical or electrocautery devices can be an ignition source. In the report detailing an intraoral fire caused by the use of monopolar diathermy with THRIVE [3], the patient suffered no ill effects and was discharged as planned the same day. This was because only an arc was seen from the diathermy tip to one of the titanium dental implants causing a burn on the polytetrafluoroethylene (PTFE) on the diathermy shaft. The diathermy was immediately removed from the oral cavity. Further diathermy was used only when the THRIVE was turned off for a brief period while the surgeon used the diathermy. The authors [2] theorized that in this case the cause of the fire was the patient's false passage between the left maxilla and the oral cavity, the result of a previous surgery. This allowed high-flow nasal oxygen into the surgical field. It is possible that if the surgeon had used a bipolar diathermy, this may have posed a lower risk, but arcing is still possible [4].

We have used THRIVE with great success in morbidly obese patients, undergoing ECT. In ECT even a short apnea time can lead to severe desaturation [5].

Lesson

Diathermy, oxygen, and a source of fuel can cause fires. Be aware!

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Case 100: Severe Case of Hyperkalemia During Rapid Blood Transfusion



Today you are assigned to anesthetize an 80-year-old female for a uterine malignancy (88 lbs, 5'5'', ASA 2). She has hypertension but no history of ischemic heart disease. On exam she is marked cachectic with a hard mass palpated in her lower abdomen. Her HCT is 30%. The other laboratory values, ECG and chest x-ray, are normal. A routine general anesthetic is given and 20 G right radial line and a triple lumen CVP are inserted into the right jugular vein.

The operation reveals a large leiomyosarcoma and a radical hysterectomy with debulking is commenced. Unfortunately, a sudden blood loss of 1500 ml leads to hypotension (70/40 mm Hg), tachycardia (105 bpm), and a decrease in CVP to 0 cm H₂O. You start giving 2 units of packed cell with 500 ml of albumin 5% over a 5–10-min period using the patient's 14 G IV in the right forearm.

An increase in T waves and a widening of the QRS complex are seen on the ECG. You diagnose an acute hyperkalemia and send off a blood gas to ascertain if you are right. Without waiting for the potassium result, you give 10 ml of calcium chloride 10% and 20 mg of furosemide, both IV. You are contemplating giving insulin and glucose when the heart rate decreases precipitously from 90 to 45 bpm. The nurse tells you the serum potassium is 7.5 mmol/l. You give atropine 1.2 mg and stop the transfusion.

Is there anything you can give besides insulin and dextrose to bring the potassium levels down rapidly?

A low-dose epinephrine (2 mcg/min).

Discussion

Epinephrine is useful in treating acute hyperkalemia during a massive blood transfusion [1-3]. This is due to its beta-2 agonist effects which drive potassium intracellularly. Correction of acidosis and boluses of insulin and dextrose are important adjuncts in the treatment. But be aware that the response to insulin may be slow especially if the patient's temperature is low. I personally have observed that epinephrine corrects hyperkalemia quicker than insulin/dextrose.

In this patient, despite the small transfusion volume administered, several factors may have led to the hyperkalemia. Rapid infusion of blood products in this cachectic patient led to a dramatic change in the homeostasis. Added to this is the fact that the patient received 22-day-old blood. When blood is stored, alterations in its constituents occur; particularly the potassium levels tend to increase. The high potassium level is a result of the leakage of potassium ions from the red blood cells into the plasma. The increase is proportional to the duration of the storage of the blood. Potassium concentrations as high as 70 mmol/l on day 28 have been recorded [4]. If we consider that the blood given to the patient (a 1/4 of her blood volume) had a potassium concentration of 30 mmol/l, then this would have been a very large amount of potassium given to her over a few minutes. Potassium of 0.3 mmol.kg given rapidly to dogs has been shown to cause ventricular fibrillation [5]. In the above case, manipulation of the tumor may also have contributed to the potassium increase. The rapidity of the blood transfusion is important. Hyperkalemia may occur at transfusion rates in excess of 0.4 ml/kg/min [6]. It is important to remember that pressure bag infusion of red blood cells may traumatize the cells and lead to hyperkalemia [7]. However, in the case outlined above, it was necessary to increase the blood volume quickly.

Lesson

During rapid blood transfusion it is imperative to carefully monitor the ECG and check the serum potassium levels [8, 9].

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Case 101: A Monitor Is Just a Machine



Many years ago I anesthetized a frail 54-year-old man (68 kg, 5'4" ASA 4E) for an above-knee amputation for gangrene of his leg. He was a type 1 diabetic with coronary artery disease, poor exercise tolerance, hypertension, and ketoacidosis. The only monitor at the time was a Hewlett-Packard ECG machine. A routine rapid sequence anesthetic induction ensued and the airway is secured with an ETT. The operation started and the patient's vital signs were within normal limits. Twenty minutes into the case, excess blood loss occurred and the ECG went to a flat line. I called a code. There were no pulses and CPR was started immediately with the help of the surgeon. While I was busy with the resuscitation, an anesthetic colleague brought to my attention that the ECG was now working and showed a sinus rhythm (SR) 72 bpm. He congratulated me on a job well done. But unfortunately there was no pulse or BP to be obtained from the patient. CPR was continued, but the patient was declared dead 30 min later. The ECG machine was still attached to the patient and still reading 72 bpm SR.

What was happening?

The machine was a demonstration model. In this case, when there was suddenly no heart rate to be picked up, the machine started showing a normal ECG.

Discussion

I always teach that a monitor is just a machine. If something does not fit the clinical picture, you must always examine your patient. Failure to do that could be disastrous for a patient [1].

Lesson

A monitor is just a machine. Examine your patient.

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Case 102: A Case of Preoperative Sinus Tachycardia



Today you are to anesthetize a 42-year-old male (54 kg, 4'11", ASA 3) for a revision of his left total hip replacement. He has ankylosing spondylitis, Reiter's syndrome, hypertension, hyperlipidemia, and chronic pain. His past surgical history is significant for hip surgery and previous two spinal fusions. He reports no allergies or previous anesthesia problems. He takes lisinopril, simvastatin, and Vicodin. He denies taking herbal medication and/or illicit drugs. He smokes ten cigarettes a day and drinks two–four beers at the weekend.

On examination his heart rate is 110 pbm, BP 140/80, respiration 14, and oxygen saturation 98% on room air. Examination of his heart and lungs reveals nothing abnormal. He is afebrile, is resting calmly in the bed, and denies any pain or distress. No history of recent colds or illnesses is elicited. You ask him to stick out his hands and discover no evidence of hand tremors.

You call for a portable ECG to be brought to the preoperative area. With machine you confirm sinus tachycardia at 109 bpm. There are no ST or T wave changes. You look at the laboratory studies and all are within normal limits, including a TSH.

You wonder why he has such a high resting heart rate. You tell him your concerns, but he says he drinks five-six strong coffees a day and also a lot of energy drinks.

Since the ECG machine took some time to arrive, the surgeon has now come to see what the holdup is with his case.

What will you do? Proceed with surgery? Cancel surgery? More investigations?

Solution

The surgery was delayed temporarily and a urine test from the patient was sent off for a toxicology screen. The results came back as positive for narcotics, but also amphetamines >1000 ng/ml.

Discussion

We have previously discussed general anesthesia in patients with chronic amphetamine use (Case 56), but this is different. While chronic amphetamine users of Ritalin rarely have a blood level over 20 ng/ml, a patient with over 1000 ng/ml presents a totally different problem. Here, most likely, the depletion of the catecholamine receptor storage would be excessive, leading to diminished or nonexistent physiological and sympathetic response to hypotension.

The case above was cancelled. As an explanation for his high level of amphetamine, he stated: "I was at my friend's house last night and he was smoking something." He was told to come back for his surgery when he had 3 negative urine drug screens over a 3-month period.

Lesson

An unexplained physical finding always needs to be investigated. If you are concerned, act on your sixth sense.

Case 103: Bonus Question



You have anesthetized, in a routine manner, a 30-year-old man (ASA 2) who is scheduled for a cervical lymph node biopsy with a possible neck dissection. Since the surgeon requested no muscle relaxation during surgery, succinylcholine 70 mg was given to secure the airway.

Ten minutes later the surgeon, with his finger in the patient's mouth, asks you if the patient is paralyzed.

How can you best convince him that the patient is not paralyzed?

Solution/Discussion

Dr. Michael Keating and I were faced with this question by a surgeon [1].

It was quickly answered as Michael activated the nerve stimulator which was placed over the trigeminal nerve. Immediately the patient closed his mouth firmly. Thus, the surgeon was assured that the patient was no longer paralyzed, but he did complain of a sore finger.

Lesson

This sort of case is worth publishing and we did [1]. The title of our submission was "Another Use for the Nerve Stimulator?"

References

1. Keating M, Brock-Utne JG. Another use for the nerve stimulator? Anesth Analg. 1995;81:1312.

Index

A

Abbott Critical Care System, 105 Abdominal stab wound, 130 Absolute refusal, 96 Accu-Chek[®] active, 27, 28 ACE inhibitor, 225, 226 Acoustic neuroma, 281–282 Acute angle-closure glaucoma, 262 Acute bilateral vocal cord adduction, 168 Acute hyperkalemia, 326 Addition deficit hyperactivity disorder (ADHD), 196 Adenoidal facies, 297 Adenoids, 298 Adenotonsillectomy, 297, 298 Adequate bupivacaine, 214 Adequate surgical anesthesia, 16 Agitation, 167 Air embolism, 182, 183 Airway, 37, 38 complication, 75 obstruction, 164, 212, 290, 298.308 Alanine aminotransferase, 300 Albumin, 95, 96, 181, 182 Albuterol, 319 Alcoholism, 188 Alveolar ventilation, 80 Alveoloplasty, 211 Ambu bag, 3, 87, 133–135 American Society of Anesthesiologist (ASA), 41, 172 Ammonia encephalopathy, 220 Amphetamines, 195, 196, 332 Analgesia, 113

Anaphylaxis, 102 Anesthesia general and spinal, 217 nitrous oxide, 218 Anesthesia Department's Clinical Competence Committee, 291 Anesthesia machine, 41, 42, 44 Anesthesia Preoperative Assessment Clinic, 7.9 Anesthetic breathing circuit, 186 Anesthetic drugs, 205 Anesthetic technique, 248 Anesthetic tubing, 44 Aneurysm, 137 Angiocath, 110 Angioedema ED. 225 ETT, 225 Ankylosing spondylitis, 295 Antacid, 16 Antifungal prophylaxis (itraconazole), 269 Anxiety symptoms, 7 Aortic arch, 177, 178 Aortic stenosis, 19 Ardendorf, D., 2 Argatroban, 156 Argyle feeding tube, 61 Arndt endobronchial blocker, 244 Arrhythmia, 47, 50, 51 Arterial blood gas (ABG), 137 Arterial line, 257 Arterial pressure, 106, 107 Aspartate aminotransferase, 300 Asthma, 319, 320 Atrioventricular node ablation, 323

© Springer International Publishing AG 2017 J.G. Brock-Utne, *Clinical Anesthesia*, https://doi.org/10.1007/978-3-319-71467-7 Atropine, 16, 194 Automated implantable cardioverterdefibrillator (AICD), 105 Awake craniotomy ammonia encephalopathy, 220 children and young adults, 220 claustrophobia, 35 cold air, 36 ECG, 219 general anesthesia, 35 lack of air, 35, 36 MAC, 35 Polar Air Model 600, 36 propofol and remifentanil infusion, 36

B

Back brace, 224 Barotrauma, 274 Bayer diagnostics, 28 Becker muscular dystrophy (BMD), 300 Benzodiazepines, 16 Bilateral French nasal airways, 212 Bilateral pneumothorax, 126 Bilateral secretory otitis media, 297 Bilateral vocal cord adduction, 168 Biliary atresia, 207 Bite block, 3, 5 BiteGard, 163 Bivalirudin, 156 Bleeding, 138, 211, 212, 308 Blood loss, 181 Blood pressure cuff, 291, 292 Blood sugar, 27 Blood transfusions, 95, 96 BM diagnostics, 28 Bolton, P., 296 Bone flap, 229 Bradycardia, 196, 277, 278 Brain technique, 73 Braun epidural catheter, 148 Bromage scales, 113 Bronchoscopic port, 164 Bronchoscopic Swivel Elbow Adaptor, 38.164 Bronchospasm, 101, 102 BROVIAC® catheter, 19 Bucking/coughing, 248 Bupivacaine, 15, 215 Buprenorphine, 215, 216 Burns, 24

С

California Board of Medical Quality Assurance, 160 Cancel case, 160, 298 Capnography, 79-81 tubing, 80 waveform, 79, 80 Carbon dioxide laser, 67 Cardiac arrest, 270 Cardiac tamponade, 130, 277, 278 Cardiopulmonary bypass, 47, 177 Cardiovascular collapse, 102 C-arm, 58 Carotid endarterectomy (CEA), 167, 168 Carpal tunnel release, 235 Catecholamine, 196 Caudal block, 114, 297 Central line placement, 126, 245 Central nervous system (CNS), 196 Central venous catheter, 126 Central venous pressure (CVP), 125 Cephalad, 114, 312 Cerebellar tumor, 163 Cerebellopontine hematoma, 282 Cerebral angiography, 141 Cerebral swelling, 230 Cerebral thrombosis, 156 Cerebrospinal fluid (CSF), 121, 216, 271, 272 fluid-cutaneous fistulae, 122 and interstitial fluid, 122 lavage, 272 leak, 122 Cervical and upper thoracic spine radiography, 280 Cervical discectomy, 159 Cervical fracture/dislocation, 296 Cervical hematoma, 247 Cervical lymphadenopathy, 297 Cervical plexus block, 167, 168 Chemical Dependence Guidelines for Departments of Anesthesiology, 173 Chest drain, 125, 126 Chloroprocaine, 113 Cholecystectomy, 269-270 Chronic amphetamines, 196, 197, 332 Chronic kidney disease, 188 Claustrophobia, 36 CO2 waveform, 79 Coagulation, 137, 138 Cocaine 4%, 92 Code blue, 109 Coenzyme pyrroloquinoline, 28

Index

Combustion, 24 Compartment syndrome, 292, 300 Compound Benzoin Tincture USP, 147 Compressed gas source, 134, 135 Computerized tomography (CT), 282 Conditional refusal, 96 Contralateral recurrent laryngeal nerve, 168 Controlled ventilation, 135 Cook's exchange catheter, 164 Cordis catheter, 125 Corneal abrasion, 34 antiseptic solution, 262 autoimmune disease, 262 endotracheal intubation, 261 intraoperative ephedrine administration, 262 iridotomy, 262 meperidine, 261 midazolam, 261 retinal artery blood flow, 262 thyroidectomy, 261 Corneal laceration, 262 Coronary artery disease (CAD), 20, 155 Cranioplasty, 229, 230 Craniotomy, 171, 311 Creatine kinase (CK), 300 Cricoid pressure, 92, 145 Cricothyrotomy, 87, 88, 212 Crystalloids, 114, 125 Cyanosis, 277, 278 Cystoscopy, 213, 214

D

Damping, 107 Danaparoid sodium, 156 Datex capnograph aspirating port, 80 Datex Capnomac machine, 42 Datex Capnomac Ultima, 79 Decreased exercise tolerance, 319 Delay case, 160 Demonstration model, 330 Dexamethasone, 76 Dextroamphetamine, 195 Diabetes mellitus, 188 Dialysis fluid, 27, 28 Diameter index safety system (DISS), 238 Diathermy, 324 Difficult airway, 1, 15, 91, 119 Digoxin, 47, 50-52 Diphenhydramine, 76 Distended abdomen, 110 Do not intubate (DNI), 20

Do not resuscitate (DNR), 20, 21 Down's syndrome, 277 The Drager Vapor 2000, 41, 42 Draping of patients, 110 Draw-over vaporizer, 133, 135 Droperidol, 16, 215 Drug abuse, 172 DuraPrep surgical solution, 231

Е

Ear-nose-throat (ENT), 168, 308, 323 ECG, 68, 325, 326 abnormal, 8 asymptomatic, 9 atrial fibrillation, 8 computerized, 7-9 normal P-wave morphology, 9 patient. 9 pertinent clinical data, 9 vaso-vagal reaction, 8 Electrocautery devices, 232, 324 Electroconvulsive therapy (ECT), 151-153 Emergency department (ED), 225, 226 Emergency room (ER), 129 Enalapril, 151 Encephalopathy, 220 Endotracheal tubes (ETTs), 3–5, 33, 37–39. 57-59, 72, 75, 83, 101, 109, 119, 129, 163, 167, 168, 185, 186, 190, 191, 199, 201, 225, 243, 247, 248, 255, 257, 261, 295-296, 308, 311, 312 End-stage renal disease (ESRD), 27 End-tidal carbon dioxide (ET-CO₂), 41, 42, 109, 129, 134, 145, 273, 284 End-tidal pCO₂, 312 Enlarged tonsils, 297, 298 Enucleation, 203 Ephedrine, 196 Epidural, 113, 114, 122, 147, 148, 165 catheter, 121, 147-149, 215, 271, 272 infusion, 215, 216 Epinephrine, 129, 147, 167, 196, 323, 326 Etomidate, 145, 155, 199 Extremely low birth weight (ELBW), 277, 278 Eye patch, 203, 204 Eye vitrectomy, 218

F

Face masks, 34, 203 Facial deformities, 117 Fascia lata, 12 Femoral artery cannulation, 137 Femoral nerve block, 12 Femoral vein, 125 Fentanyl, 145, 203 Fiber-optic bronchoscopy (FOB), 168, 244 Fiber-optic endotracheal intubation, 190 Fiber-optic illumination system (FIS), 23-25 Fiber-optic intubation, 15, 91, 92, 225, 295, 296 Fiber-optic nasal intubation, 212 Fiber-optic scope, 15, 117 Fire, 24, 232, 324 Flail-segment rib fractures, 223, 224 Flexible bronchoscopy, 243, 308 Fluorescein dye, 34 Fluoroscopy, 142 Fluphenazine, 151 Fractured neck of the femur, 11, 12 Fresh frozen plasma (FFP), 137 Full stomach, 91 Functional endoscopic sinus surgery (FESS), 75 Furosemide, 68, 152

G

Gag reflex, 76 Gas embolism, 304 Gastric bypass, 145, 319 Gastric insufflation, 99 Gastroschisis, 277 Gauge intravenous catheter, 119 General anesthesia (GA), 1, 15, 28, 41, 68, 75, 83, 91, 101, 103, 105, 129, 145, 147, 159, 167, 177, 195, 196, 203, 214, 243, 252, 261, 262, 265, 273, 282, 291, 297-299.303 Glasgow Coma Scale, 283 Glucose monitors, 28 Glucose test strips, 28 Glycopyrrolate, 93, 152 Grant, M., 178 Guedel airway, 289, 290, 298 Guide wire, 245, 246 Gum elastic bougies (GEB), 117, 164, 319, 320 anesthesia, 38 bronchoscopic swivel adaptor, 38, 39 ETT, 37, 38 medical student, 37 ventilation, 38

H

Hallucination, 16 Halo, 189–191 Head attachment to operating room table, 280 Head down tilt, 304 Hematomas, 211 Hematuria, 299, 300 Hemodialysis, 109 Hemoglobinuria, 300 Hemothorax, 84, 125, 126, 223, 224 Heparin, 137, 138 Heparin-induced platelet aggregation assay (HIPAA), 157 Heparin-induced thrombocytopenia (HIT), 156, 157 Heparin-induced thrombocytopenia thrombosis syndrome (HITTS), 156 Heparin-PF4 antibodies, 157 Hepatic encephalopathy, 84 Hetastarch bag, 183 Hewlett-Packard ECG machine, 329 "Hidden" IV case complications, 32 CVP, 31 laparotomy, 31 operating room with 20G IV, 31 problem, 32 in vein. 32 Hip replacement, 213, 214 Hoarseness, 193 Homer, T., 212 Hotline® Fluid Warmer, 31 Hydrocortisone, 76 Hydro-dilation, 214 Hydrogen peroxide, 303, 304 Hyperammonemia, 220 Hypercarbia, 274 Hyperextension, 188 Hyperkalemia, 325, 326 Hyperlipidemia, 331 Hypertension, 155, 167, 331 Hypoglycemia, 27, 28, 171, 252 Hypoguard, 28 Hypotension, 101, 102, 277, 325 Hypotension pneumoperitoneum, 110 Hypovolemia, 95 Hypoxemia, 101

I

Icodextrin, 27, 28 Icodextrin dialysis fluid, 28 ICU, 283, 295, 307 Inadvertent trauma, 118 Indigo carmine (IC), 240, 284 Infiltration of IV solution, 32, 283–284 Inguinal hernia repair, 15, 27 Index

Inhalation induction, 1 In-line syringe technique, 182 INR, 125, 126 Inspiratory limb, 319 Insulin, 151 Insulin-dependent diabetes mellitus, 155 Insulin pump, 165 Interatrial pressure, 316 Internal jugular, 142 Interstitial fluid, 122, 123 Intra-arterial catheter, 208 Intracranial pressure, 230 Intramuscular injections, 205 Intrathoracic pressure, 316 Intravenous administration set, 182 Intubation, 1, 295, 296 Isoflurane, 145, 155, 281, 311

J

Jehovah's witness, 95 Jet ventilator, 100

K

Keating, M., 334 Keloid scar, 1 Ketamine, 11, 16, 206, 211 Ketamine infusion, 238 Knee arthroscopy, 113, 114, 289 Knife wound, 130

L

Lactate dehydrogenase, 300 Lateral medical bag, 206 Language disturbance, 251, 252 Language mapping, 35, 36 Laparoscopy, 53 Kasai, 207 surgery, 99 tubal ligation, 165 Laparotomy, 31, 121, 145 Laryngeal mask airway (LMA), 71-73, 168 Laryngoscopy, 109, 145, 176, 206, 295, 296 Laryngospasm, 151, 152 Laryngotracheal installation of topical anesthesia (LITA), 248 Latex allergy, 101-103 Leaking endotracheal tube, 175, 176 Left bundle branch block (LBBB), 245 LEFT-handed laryngoscope, 201 Leiomyosarcoma, 325 Lepirudin, 156

Leucomethylene blue, 240 Lidocaine, 15, 92, 147, 148, 323 Lidocaine infiltration, 113 LifeScan, 28 LMA, 72–74, 320 Local anesthesia, 117 Lost privileges, 159, 160 Lower esophageal sphincter (LES), 92 Low saturation, 277 Lung adenocarcinoma, 243 Lung compliance, 109 Lung isolation, 244

M

Macintosh laryngoscope, 190 Macintosh, R., 38 Magill forceps, 62 Mallampati, 71 Maltose, 28 Maltotetraose, 28 Maltotriose, 28 Manual ventilation, 45 Mask ventilation, 110, 203 Maxillofacial operation, 265-267 Median nerve injury, 187, 188 Mediastinal emphysema, 88 MediSense, 28 Meperidine, 211, 303 Metabolic acidosis, 278 Methylene blue, 239, 240 MicroMist nebulizer, 92 Midazolam, 175, 203, 211, 213, 291 Mobile Army Service Hospital (MASH), 182 Modus operandi, 291 Molar bite block, 312, 313 Mometasone furoate inhalers, 319 Monitored anesthesia care (MAC), 35, 159, 231 BROVIAC® catheter, 19 conflict and potential liability, 20 DNR/DNI, 19, 20 patient, 19 Morbidly obese patient, 145 Morphine, 11 Mucosal atomizer device (MAD), 92 MVA, 283 Myoglobinuria, 300

Ν

Naloxone, 281, 282 Nasal airways, 212 Nasal phenylephrine, 266 Nasogastric tube (NGT), 61, 62, 99, 100, 117-119, 256 Neck dissection, 333 Negative pressure pulmonary edema (NPPE), 152 Neostigmine, 269, 270 Neo-synephrine, 141, 142 Nerve conduction block, 187, 188 Nerve stimulator, 63, 334 Neuromuscular disorder, 300 Nifedipine, 151 Nitrogen flush, 288 Nitrous oxide, 218, 281, 303, 304 Noninvasive blood pressure, 105, 106 Non-rebreathing mask, 125 Nonrecurrent larvngeal nerve, 167 Non-woven paper surgical drapes, 24

0

Obesity, 155 Obstetrical patient, 271-272 Obstructive sleep apnea (OSA), 315, 316 Ohmeda Cyprane PAC, 133 Oligosaccharides, 28 Omar slave, 44, 45 Oozing, 137, 138 Open reduction internal fixation (ORIF), 291 Operating room (OR) table, 57, 58, 279, 280 Opiate overdose, 282 OpSite, 147 Optiflow, 323 Oral Bicitra, 16 Oral fiber-optic intubation, 1 Oral surgery, 211 Orchidopexy, 297, 298 Oropharynx suction catheter, 256 Orthopedic patients, 133 Orthopedic procedure, 303 Oxygen insufflation, 119 Oxygen pipeline, 287, 288 Oxygen saturation, 145, 187, 211, 212, 235, 239, 240, 273 Oxymetazoline, 266 Oxymethazoline, 266

Р

Pain, 15 Painful eye, 33, 34, 261–263 Paper clip, 118 Paradoxical embolization, 316 Paraplegia, 296 Park-bench position, 257, 258 Parotidectomy, 247 Paroxetine, 151 Patent foramen ovale (PFO), 316 Peak inspiratory pressure (PIP), 101, 273, 311 Peak pressure, 79, 145 Pediatric anesthesia, 179 Pediatrics, 300 Penlon Oxford ventilator (POV), 133-135 Percutaneously inserted central line (PICL), 278 Pericardial effusion, 278 Pericardial sack, 130 Pericardiocentesis, 277, 278 Peripheral intravenous, 142 Peripheral neuropathy, 188 Peroral endoscopic myotomy (POEM), 273-275 Phenobarbital coma, 238 Phenylephrine, 196 Phrenic nerve paralysis, 235, 236 Pilling retractor, 167 Pilot tubing, 3-5 Pinpoint pupils, 281, 282 Pipeline pressure, 41 Platelet aggregation, 156 Platelet count, 156 Pleural catheter, 126 Pleural cavity, 126 Pleural space, 126, 127 PMT[®] Corporation's, 189 Pneumomediastinum, 274, 275 Pneumoperitoneum, 110, 208, 274 Pneumothorax, 126, 223, 274 Polar Air Model 600, 36 Polytetrafluoroethylene (PTFE), 324 Polyvinyl chloride bags, 182 Porphyria, 300 Positive end-expiratory pressure (PEEP), 316 Post-anesthesia care unit (PACU), 215 posterior spinal instrumentation and fusion (PSIF), 223 Postoperative complications, 75-76 Preoxygenation, 145 Pressure-controlled mechanical ventilation, 273 Pressurizing IV glass bottle, 183 Pretracheal tissues, 307 Prone position, 164, 311, 313 Propofol, 142, 211, 319 Propofol infusion, 35, 36 Prostate cancer, 239, 240 Prostatectomy, 95, 315 Protamine, 138 Pulmonary edema, 67, 68 Pupillary dilation, 178 Pyloromyotomy, 61

Q

Quadriplegia, 296

R

Radial intra-arterial catheter, 187, 188 Radionuclide myelography, 121 Rapid blood transfusion, 326 Recovery room, 33, 113, 114 Rectal cancer, 185 Recurrent laryngeal nerve, 167, 168 Red urine, 299-300 Referred pain, 165 Refractory status epilepticus (RSE), 237 Reiter's syndrome, 331 Remifentanil, 35, 36 Reservoir bag, 43, 44 Respiratory failure, 110 Reversal of epidural anesthesia, 114 Rhabdomyolysis, 300 Right bundle branch block (RBBB), 246 Right endobronchial intubation, 83 RIGHT-handed laryngoscopy, 200, 201 Right-to-left shunt (RLS), 316 Robot-assisted surgery, 207, 208 Roche diagnostics, 27, 28 Rocuronium, 145, 146 Rotameters, 41 Rotational thromboelastometry (ROTEM) machine, 138 Ruben's valve, 134

S

Saline epidural injection technique, 114 Scalpel, 87-89 Scar tissue, 187 Seizure disorder, 35 Seldinger technique, 125 Semi-prone position, 281 Senning procedure, 47 Serum sodium levels, 68 Sevoflurane, 203, 298 Sevoflurane vaporizer, 41 Sheridan Jet Ventilation Catheter, 164 Shortness of breath, 319 Shoulder pain, 166 Sikh beard, 63–65 Sinus rhythm (SR), 8, 245, 329 Sinus surgery, 75 Sinus tachycardia, 145, 331 Sitting position, 92, 93 Sleep apnea, 297 Smoke, 23, 24

Sniff method, 42 Sodium bicarbonate, 284 Sphygmomanometer pump, 182 Spina bifida, 101, 102 Spinal anesthesia, 11, 12, 15, 23, 272 Spinal fracture **PSIF. 224** traumatic, 224 Stab abdomen, 31 Stab wound, 131 Stereotactic craniotomy, 311 Steroids, 76 Stridor, 167, 168 Stylet, 72-74 Subarachnoid catheter migration, 215 Subclavian artery, 87, 142 Subclavian triple lumen catheter, 187 Subclavian vein, 125 Subcutaneous edema, 121, 122 Subcutaneous emphysema, 274, 275 Subcutaneous tissue, 148, 149 Subglottic stenosis, 320 Subxiphoid catheterization, 110 Succinylcholine, 146, 151, 152, 185, 199, 319, 333 Suction catheter, 255, 256 Sufentanil, 155 Suicide, 1 Superior mediastinal syndrome, 87 Supraclavicular block, 235, 236 Supraglottic edema, 168 Surgeon, 159, 160 Syringes, 99 Systolic blood pressure, 142

Т

Tachyarrhythmia, 50 Tachycardia, 325 TEE, 130 Thiopental, 203 Throat pack, 176 Thromboelastography, 138 Thrombus, 308 Thyroidectomy, 167, 168 Thyroid-mental distance, 63 Thyroxine, 151 Tissue necrosis, 284 Tongue ring, 53, 54 Tongue sutures, 212 Tonsillectomy and adenoidectomy, 279 Total hip replacement, 331 Tourniquet, 101-103, 303, 304 Tracheal poly, 243 Tracheal tube exchanger, 164

Tracheostomy, 87, 168, 194, 225, 226, 307, 308, 320 Trachlight, 1 Transducer stopcock, 106, 107 Transducer system, 138 Transjugular intrahepatic porto-systemic shunt (TIPSS), 83 Transpac IV monitoring kit, 105 Transplanted heart, 269 Transthoracic echocardiography, 277 Transtracheal jet ventilation system, 119 Transurethral resection, 23 Trauma, 179 Trendelenburg position, 166, 185, 207, 208, 312 Trigeminal nerve, 334 Triple lumen venous catheter, 141, 142 Trismus, 63 Tuohy needle, 148

U

Ulcerative colitis, 147 Ulnar nerve injury, 292 Universal bite block, 3 Upper gastrointestinal bleeding, 109 Ureteral patency, 240 Uric acid, 28 Urinary microscopic analysis, 299 Urine, 239, 240 Urine dipstick, 299 Urological surgical trauma, 300 Uvular swelling, 76 Uvulitis, 76

V

Vaginal pack, 176 Valproic acid, 220 Vapor, 41, 42 Vapor concentration, 135 Vaporizer, 41, 42, 133 Variceal bleeding, 83 Vasectomy reversal, 7 Vasoconstrictors, 265-267 Vasospasm, 141 Vaso-vagal reaction, 8 VATS procedure, 244 Vecuronium, 155, 203, 311 Venous return, 208 Ventilation, 185, 186 Veress needle decompression, 274 Visual hallucinations, 16 Vocal cord mobility, 168 Vocal cord paralysis, 168 Vocal cord polyps, 193, 194

W

Warfarin, 157 Waste anesthetic gases (WAG), 237 Wegener granulomatosis, 319, 320 White clot syndrome, 156 White toes, 303 Wire-guided endobronchial blocker (WEB), 244

Х

X-ray technique, 83

Y

Yankauer suction, 75 Yodfat technique, 72, 74