Matthias Hübler
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Complications and Mishaps in Anesthesia

Cases
Analysis
Preventive Strategies

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Preface

Experience is like a lantern shining on our back.
It lightens the path lying behind us.
Confucius (551–479 BC)

Do we learn enough from our errors?

Probably yes, but we are not learning enough from the errors of others! Our knowledge increases during our professional life as we master more and more critical situations. This is called experience. Unfortunately, it takes time before we reach a certain level of experience. We just need too many events, and we need errors to happen: errors trigger emotions which trigger learning. Errors are very useful if we are ready to accept personal failure as part of our life.

Working in anesthesiology very often means working alone or working in small teams. The occasions when we can learn from errors of others are limited. Thus, we are learning enough from our errors but not enough from the errors which others experience. The first intention of this book is to reduce this gap by sharing with you our experiences with complications and mishaps in our daily anesthetic practice. The basis of the presented cases were real life events. They were modified such that recognition is impossible. Similarities with real persons are therefore purely incidental. Learn from our errors and do it better!

Doing it better is not easy… The second intention of this book is to assist you to reduce errors. The given tips or clues are not limited to medical facts but also include organizational and nontechnical factors and skills. Latter aspects are included in the case debriefings. In most cases more than one aspect could be discussed, but we focused the debriefings and limited the discussions per case, also to avoid redundancies.

Learning by error is important, but why? Because emotions are involved, negative or positive ones. These emotions influence our perception, our thinking, and our interpretation. Situations, circumstances, and facts immediately become part of our long-term memory. Accessing information, which was stored emotionally, is easy and fast. So the intention of this book is to stimulate you emotionally while presenting the facts – while you learn. The cases therefore tell you the story behind the scenes. These epic parts provide detailed information of the circumstances. Try to get emotionally involved and feel with the protagonists! Maybe you will later remember the facts because you still think about the story behind the scenes.

The second edition of the book was published in 2012 in German. The American edition was completely revised and new aspects of the rapid changing medical knowledge were included. Also, medicolegal and specific professional aspects were adapted.

We hope that you like this new concept of a different kind of textbook. We wish you interesting, stimulating, and maybe even entertaining reading – and learning. Eventually it helps to increase patient safety and improve patient care, because you may not have to experience some of the errors and mishaps yourself.

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Names and Their Meanings

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The names of the main protagonists in this book each have a deeper meaning. For the interested reader, here a list of additional information.

Alexander (Case 27)
Greek name derived from the words alexo (protect) and andros (man), therefore meaning the "protector of men."

Andrew (Case 23)
Greek origin, meaning courageous, manly, strong. Courage is a virtue that turns ordinary men into heroes. Courage is one of the four cardinal virtues, which are the foundation of all other virtues.

Aric (Case 28)
Aric is a variant of Eric, which is a composite of the Norse elements "ei" (ever) and "rikr" (ruler). Its meaning is "one ruler."

Armstrong (Case 2)
The meaning is obviously strong armed. He is a fighter and "the one who fights on his own."

Baldwin (Case 13)
Derived from the Germanic words "bald" (= brave) and "win" (= friend). In our case Baldwin stands for bravery. The brave takes risks that can be avoided and he is willing to carry out difficult and unpleasant tasks.

Benjamin (Case 19)
Hebrew: "son of right." Benjamin also has the meaning "lucky fellow."

Berenice (Case 30)
Berenice has the meaning "bearer of victory." The name has Greek origin incorporating the word nike (victory) and phero (bring).

Blue (Case 20)
Besides standing for a color, it means also "sad."

Buster (Case 16)
Buster is a slang word for a "person who breaks things." It means also "fellow."

Canute (Case 29)
Canute is the Anglicized form of the Old Norse named Knut. It derives from the word "chnuz" (daring).

Casey (Case 9)
The name is more often used for girls than for boys, as in our case. It is derived from the Celtic word "cathasaigh" with the meaning "vigilant, wakeful." As vigilance is the continuous concern for the safety of others, the concern for the safety of others is likewise a constant motivation for vigilance.

Cassidy (Case 27)
Cassidy is Irish derived from the Gaelic name Caiside meaning "clever" or "curly haired." Obviously, the named was not picked because the sharp-witted Dr. Cassidy has curls.

Cedric (Case 22)
Celtic origin with the meaning "friendly, likeable."

Charles (Case 2)
Charles is the English version of the old high German name Karl with the meaning "husband" as well as "free man." In our case Charles is a synonym for freedom. Freedom implies the chance to act independent of external influences.

Clare (Case 10)
Clare is the English form of Clara which is derived from the Latin name Clarus (bright, clear). In our case Clare stands for clarity. Clarity is a result of our attention. The German philosopher Christian Garve states:

"Nature keeps a constant balance between the dark and the bright side of our imagination. Once clarity increases in one part, other parts fall into deep darkness. Every rapprochement of the soul to an object will at the same time increase the distance to all others."

Conall (Case 24)
Conall is an old Gaelic name. The meaning is being strong and brave like a bear. The name is used in several Irish legends.

Conner (Case 25)
Conner is a Celtic name with the meaning "hound lover." It is also associated with the meaning "strong will."

Constantine (Case 3)
Latin: "constant one, not changing, steadfastness." Steadfastness is the resistance against obstacles. Resistance against legal obstacles might often be regarded as stubbornness.

Damel (Case 1)
Damel is of English-American origin and means "a strong-willed man." In our case Damel stands for "determinedness."

René Descartes defined four basic principles in his work "Discourse on the Method": "principle of prudence, determinedness, composure and leisure."

In times of changes from old to new knowledge, it is mandatory for Descartes to hold on to these principles to assure actual survival. The principle of determinedness means to hold on to the likely and persistently declare it as true, but not be paralyzed to act.

Dado (Case 30)
Dado derives from the Spanish verb "dar" (given).

By the use of our senses we become aware of things that are given. Our sensibility provides perceptions and with our understanding they can be thought. Only the unity of sensibility and understanding does result in cognition (Immanuel Kant).

Damian (Case 5)
The word Damianos is Greek and has the meaning "master, overcome, tame." According to Socrates, Daimonion is the inner voice of practical reason and of conscience. This keeps us from indecorous and unreasonable deeds.

Deborah (Case 17)
Deborah means "bee" in Hebrew. Deborah is as busy as a bee.

Diana (Case 9)
Diana is the name of the Roman goddess of the moon, the hunt, forests, and childbirth. She was known for her strength and was often a lone fighter.

Doxa (Case 32)
Australian female name. In our case the name is related to the Greek word "doxa," meaning opinion or belief from which the term orthodoxy derives. Greek human sciences differentiated between knowledge (Episteme) and opinion (Doxa). The science of knowledge (epistemology) is based on facts, truth, and objectivity and differs fundamentally from opinion, subjectivity, prejudice, and emotions.

Eldridge (Senior Physician, Present in Many Cases)
Eldrigde is of Germanic origin. The meaning is old counselor, ruler. The second origin of Eldridge is Old English. Here, the meaning is fearful and terrible. We would like to skip this meaning for our book, although for some senior physicians it might be true.

Elen (Case 26)
Elen is the Welsh form of Helen. In the chapter it refers to the Greek word "elenchos," which means asking and testing. Elenchos is a central technique of the Socratic method in which by debate, critical thinking and formation of new ideas are achieved.

Emerson (Case 8)
Emerson is the son of Emery. It has the meaning brave as well as "the powerful."

Emery (Case 7)
Derived from the Norman name Emmerich with the meaning "powerful ruler."

Ernest (Case 26)
Ernest is an English name of German origin. It is derived from the word "eornost" meaning earnest, seriousness, steadfastness, and battle to the death. Seriousness is often associated with the deliberateness, which is the ability to judge independent of current emotions. So action is guided by serious thoughts.

Faith (Neurosurgeon, Case 32)
The name Faith was adopted by the Puritans in the seventeenth century. In the chapter it stands for confidence and self-confidence. We have confidence because we expect a positive development. Loss of confidence results in insecurity.

Ferdinand (Case 21)
Derived from the Germanic words "frith" (protection) and "nanth" (courage), giving the name the meaning of "brave guardian."

Finn (Case 4)
Finn MacCumhail was a legendary Irish hero. As a surname Mark Twain used it in his novels. The meaning in the case is "young hero."

Greg (Case 15)
Derived from the Greek word gregoros (alert, vigilant). In our case it stands for alertness.

Hardy (Senior Physician, Case 14)
It is derived from the Middle English hardi (brave, hardy).

Harold (Case 16)
Derived from the Germanic words "chario" (army) and "waltan" (rule) and means "leader of the army."

Hugh (Case 18)
It is the English version of the name Hugo. It is derived from the Germanic word "hug" (mind), giving "Hugh" the meaning of a "minded spirit."

Imogen, Called Imo (Case 23)
The English name Imogen derives from Innogen, meaning "innocent." Innocence is defined either by the absence of guilt or as a state in which a person has no possibility to act immoral because he or she not aware of the presence of good and bad.

Justice (Case 3)
The meaning of the name is obvious. It implies fairness.

Leander (Case 18)
It is Greek in derivation and has the meaning "man of the people."

Leto (Case 12)
Greek for forgotten or hidden. In Greek mythology Leto was the mother of Apollo and Artemis.

Mallory (Case 17)
The name Mallory has French origin meaning "unfortunate" or "ill omened."

Marid (Case 31)
The origin of Marid is Arabic with the meaning rebellious. Here, we used it differently: in Arabic "ana marid" has the meaning "I am sick."

Martin (Case 17)
Mars is the Latin god of war. Martin derives from the Latin word "martinus" meaning "of war, warlike."

Maverick (Cases 6 and 7)
The name is English in origin. Maverick is used to describe somebody who is independent and nonconformist in his thoughts and action.

Unbranded cattle on a farm are also called Maverick.

Miriam (Case 27)
Miriam in Hebrew means "rebellious."

Niac (Case 17)
Niac is a variation of the African name Nyack. The name's meaning is "one who is persistent." Persistence is to endure and to overcome difficulties. In accordance to the German philosopher Friedrich Paulsen, persistence is a form of courage and is the strength of our own will to endure all kinds of complaints.

Pia (Case 14)
Female version of the Latin name Pius meaning "pious" and "dutiful."

Perk (Case 32)
Perk has its origins in the English-American language. The name is of the meaning "one who is cheerful and jaunty." Cheerful is close to humor and often the first step of laughing. Humor can be very useful to reduce interpersonal tensions and de-escalate various situations. But humor can become insulting in the form of "schadenfreude."

Pru (Case 11)
Pru is the short version of Prudence. Its origin is Latin (prudentia) and implies the meanings restrained and prudent.

Sage (Case 24)
The English word sage denotes either a spice or a "wise person."

Salvador (Case 19)
Salvador is derived from the Latin word "salvare" (to save). Salvador means saviour.

Sophie (Case 22)
Greek, "wisdom." According to Socrates, one is only wise if he knows the limitation of his human wisdom and is able to distinguish between things he knows and the things he doesn't know.

Spiegel (Case 33)
Spiegel is the German word for mirror. As an exception, a noun as a name is used in this case. Mirror refers to "mirror neurons" which were first described in the 1990s by the research group of Giacomo Rizzolatti. The group discovered that in the brains of primates the same neurons get activated by either performing an action or by solely observing it. Mirror neurons seem to be pivotal to feel empathy and compassion. We can only be empathic because we can mirror the feelings of others to our own neuronal system.

Sven (Case 16)
Derived from the Old Norse word "sveinn" which means "young man" or "young warrior."

Theresa (Case 2)
The name has Greek origin although its meaning is not exactly defined. Often translated as hunter in our case, it means observation (in Greek: parateresis). By observing things we direct our attention to objects and activities and discover their nature.

Tristan (Case 26)
Based on the Celtic name Trystan meaning "tumult," "riot," or "clash of arms."

Valentine (Case 26)
The Latin name Valens means "strong, healthy, vigourous."

Abbreviations

A	Arteria
ABG	Arterial blood gas
ACC	American College of Cardiology
ACCP	American College of Chest Physicians
ACE	Angiotensin-converting enzyme
ACGME	Accreditation Council for Graduate Medical Education
ACh	Acetylcholine
AChR	Acetylcholine receptor
ACLS	Advanced cardiac life support
ACT	Activated clotting time
ADH	Antidiuretic hormone
a-fib	Atrial fibrillation
AHA	American Heart Association
AIRS	Anesthesia Incident Reporting System
5-ALA	5-aminolevulinic acid
ALI	Acute lung injury
aPTT	Activated partial thromboplastin time
ARDS	Acute respiratory distress syndrome
ARDSNET	ARDS network
ASA	American Society of Anesthesiologists
ASAT	Aspartate aminotransferase
ASS	Acetylsalicylic acid
AT III	Antithrombin III
ATLS	Advanced Trauma Life Support
BE	Base excess
BIPAP	Biphasic positive airway pressure
BIS	Bispectral index
BMI	Body mass index
BNP	B-type natriuretic peptide
BP	Blood pressure
BW	Body weight
C	Compliance
$CaCl_2$	Calcium chloride
$CaCO_3$	Calcium carbonate
CaO_2	Arterial oxygen content
$Ca(OH)_2$	Calcium hydroxide
CAS	Central anticholinergic syndrome

CBC	Complete blood count
CBF	Cerebral blood flow
CCS	Canadian Cardiovascular Society
cCT	Cranial computer tomography
cGMP	Cyclic guanosine monophosphate
CHD	Chronic heart disease
CHE	Cholinesterase
CHF	Congestive heart failure
CIRS	Critical incident reporting system
CK	Creatine kinase
CK-MB	Creatine kinase, muscle-brain isoenzyme
CMT disease	Charcot–Marie–Tooth disease
CO	Cardiac output
CO_2	Carbon dioxide
$CO_{2\text{-ET}}$	End-tidal carbon dioxide
CO-Hb	Carboxyhemoglobin
COI	Critical oxygen index
COPD	Chronic obstructive pulmonary disease
CP	Cricoid pressure
CPAP	Continuous positive airway pressure
CPP	Cerebral perfusion pressure
CPR	Cardiopulmonary resuscitation
CRNA	Certified Registered Nurse Anesthetist
CRP	C-reactive protein
CSF	Cerebrospinal fluid
CT	Computer tomography
CTG	Cardiotocography
CVC	Central venous catheter
CVP	Central venous pressure
CQI	Continuous quality improvement
CVVH	Continuous venovenous hemofiltration
D&C	Dilatation and curettage
DDADP	Desmopressin
$\dot{D}O_2$	Oxygen delivery
DBS	Double burst stimulation
DGAI	German Society of Anesthesiology and Intensive Care
DIVI	German Interdisciplinary Society of Intensive Care and Emergency Medicine
DSA	Digital subtraction angiography
DVT	Deep venous thrombosis
DSG	German Sepsis Society
ECF	Extracellular fluid
EF	Ejection fraction
EMLA	Eutectic mixture of local anesthetics
EMT	Emergency medical team
ENT	Ear, nose, and throat
ER	Emergency room
EVD	Extraventricular drain

FEV_1	Forced expiratory volume in 1 s
F_iO_2	Inspiratory oxygen fraction
FORDEC	Facts–options–risks–decision–execution–check
FRC	Functional residual capacity
G6PD	Glucose-6-phosphate dehydrogenase
γGT	Gamma-glutamyltransferase
GCS	Glasgow Coma Scale
GOT	Glutamate oxaloacetate transaminase
GFR	Glomerular filtration rate
GI	Gastrointestinal
Hb	Hemoglobin
H_2CO_3	Carbonic acid
HCO_3	Hydrogen carbonate
HCT	Hematocrit
Heliox	Mixture of helium and oxygen
HES	Hydroxyethyl starch
HFNEF	Heart failure with normal left ventricular ejection fraction
HPV	Hypoxic pulmonary vasoconstriction or human papillomavirus
HR	Heart rate
HRV	Heart rate variability
HMSN	Hereditary motor and sensory neuropathy
I:E	Ratio inspiration to expiration
ICB	Intracerebral bleeding
ICP	Intracerebral pressure
ICU	Intensive care unit
ID	Inner diameter
Ig	Immune globulin
INR	International normalized ratio
iPEEP	Intrinsic positive end-expiratory pressure
IPPV	Intermittent positive pressure ventilation
IV	Intravenous
KOH	Potassium hydroxide
LA	Local anesthetic
Laser	Light amplification by stimulated emission of radiation
LDH	Lactate dehydrogenase
LED	Light-emitting diode
LMWH	Low molecular weight heparin
LOC	Level of consciousness
LOI	Limiting oxygen index
MAC	Minimal alveolar concentration or monitored anesthesia care
mADH	Mitochondrial aldehyde dehydrogenase
MAO	Monoamine oxidase
MAP	Mean arterial pressure
MEN	Multiple endocrine neoplasia
MET-Hb	Methemoglobin
MG	Myasthenia gravis
MH	Malignant hyperthermia
MI	Myocardial infarction

MMA	Methyl methacrylate
MMR	Vaccine immunization vaccine against measles, mumps, and rubella
MOCA	Maintenance of certification in anesthesiology
MRI	Magnetic resonance imaging
mV	Millivolt
N	Nerve
NA	Noradrenaline
nAChR	Nicotinic acetylcholine receptor
Na_2CO_3	Sodium carbonate
NADPH	Nicotinamide adenine dinucleotide phosphate hydrogenase
NaOH	Sodium hydroxide
Nd:YAG	Neodymium-doped yttrium aluminum garnet
Nd:YAG-KTP	Nd:YAG potassium titanyl phosphate
NG	Nasogastric
NIBP	Noninvasive blood pressure
NNP	Sodium nitroprusside
NNT	Number needed to treat
NPO	Nothing by mouth
NO	Nitric oxide
N_2O	Nitrous oxide
NPPE	Negative pressure pulmonary edema
NSAID	Nonsteroidal anti-inflammatory drugs
OB	Obstetrician
OCR	Oculocardiac reflex
OI	Oxygen index of flammability
OMS	Oral and maxillofacial surgery
OR	Operating room
ORIF	Open reduction internal fixation
PA	Posteroanterior
$PaCO_2$	Arterial partial pressure of CO_2
PAC	Pulmonary artery catheter
PACU	Postanesthesia care unit
P_AO_2	Alveolar partial pressure of O_2
PaO_2	Arterial partial pressure of O_2
Paw	Airway pressure
PCB	Psoas compartment block
PCC	Psoas compartment catheter
PEEP	Positive end-expiratory pressure
PGI_2	Prostacyclin (also called prostaglandin I_2)
PiCCO	Pulse contour continuous cardiac output
PMMA	Polymethyl methacrylate
PO	Per os (orally)
POD	Postoperative day
PONV	Postoperative nausea and vomiting
P_{Peak}	Peak pressure
PRAC	Pharmacovigilance Risk Assessment Committee
PRIND	Prolonged reversible ischemic neurologic deficit

PTC	Post-tetanic count
PTSD	Posttraumatic stress disorder
PVC	Premature ventricular contraction *or* polyvinyl chloride
Py	Pack year
$\dot{Q}$	Perfusion
R	Resistance
R	Ramus
RAE	Tube tracheal tube developed from W.H. Ring, J.C. Adair, and R.A. Elwyn
RBC	Packed red blood cells
RIFLE	Risk, injury, failure, loss, and end-stage kidney disease
RRP	Recurrent respiratory papillomatosis
RSI	Rapid sequence induction
SAH	Subarachnoid hemorrhage
SaO_2	Arterial oxygen saturation
SCCM	Society of Critical Care Medicine
SIRS	Severe inflammatory response syndrome
SNP	Sodium nitroprusside
SOP	Standing operating procedure
SpO_2	Partial oxygen saturation
SR	Sinus rhythm
ST	Sinus tachycardia
SVR	Systemic vascular resistance
T	Time constant
T_3	Triiodothyronine
T_4	Thyroxine
TEA	Thoracic epidural anesthesia
TEE	Transesophageal echocardiogram
TIA	Transitory ischemic attack
TIVA	Total intravenous anesthesia
TNF-α	Tumor necrosis factor-α
TOF	Train of four
TSH	Thyroid-stimulating hormone
TTE	Transthoracic echocardiogram
TUR, TURP	Transurethral resection (of the prostate)
UGI	Upper gastrointestinal
UTI	Urinary tract infection
$\dot{V}d / \dot{V}t$	Relative dead space ventilation
$\dot{V} / \dot{Q}$	Ventilation–perfusion ratio
$\dot{V}O_2$	Oxygen consumption
VT	Tidal volume
vWF	von Willebrand factor
vWJS	von Willebrand–Jürgens syndrome
WBC	White blood count
WFNS	World Federation of Neurological Surgeons

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Case 1: Retained Placenta

1

Matthias Hübler, Mike Hänsel,
and Karen B. Domino

Contents

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1.1 Case Introduction

>> Julia Mase had always been healthy, and didn't expect pregnancy or birth to change that. At 30 weeks, she was quite surprised to hear her obstetrician say that she should go to the hospital. The baby was fine, but contractions were beginning.

Lying in bed, Julia watched the cardiotocography (CTG) writing a long line of strong and regular contractions. She was told that she had amnionitis; she received antibiotics, magnesium sulfate, verapamil, and a drip to stop the contractions. The cramps in her pelvis felt better, but for the past 2 days, she had begun to feel increasingly ill; the first wave of chills began this morning.

During morning rounds, she was told the infection was worsening, and they would have to induce labor. Shortly thereafter, she was brought to the labor and delivery ward.

Six hours later her son was born, weighing 1,000 g. He was doing well; however, he needed a little extra support with his breathing, Julia was told. She saw him briefly before the neonatologist took him to the neonatal ICU. Although the delivery was much less difficult than expected, Julia was now very tired and sleepy. She vaguely overheard someone mention anesthesia, but she had no clear recollection of what happened next.

After 10 years working in this hospital, Dr. Damel, the on-call anesthesiologist, knew the origin of the numbers which most commonly appeared on his phone. "Labor and Delivery" he

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thought to himself and informed Corinne, the anesthesia technician. They set off for the labor floor, which was at the other end of the medical center, 5 min at a brisk pace.

"*We need to do an immediate D&C for a retained placenta*," *announced the obstetrician* (*OB*) *as Dr. Damel entered the room. The OB had been working in the hospital as long as he had, and Dr. Damel had learned to trust her judgment.*

As technician Corinne wheeled an anesthesia machine into the patient's labor/delivery room and set it up, Dr. Damel reviewed the patient's medical records. The OB was in a big hurry, as the patient was bleeding. There was no time for a complete preoperative evaluation and informed consent. The patient was 29 years old, 5' 6" tall, and weighed 80 kg. At first glance, the following lab results were abnormal:

- *Hb*: *9 g/dl* (*norm 11.9–17.2 g/dl*)
- *HCT*: *26 %* (*norm 37–47 %*)
- *Leukocytes*: *11.0/mcl* (*norm 3.8–9.8/mcl*)
- *Potassium*: *2.9 mEq/l* (*norm 3.8–5.2 mEq/l*)
- *TSH*: *6.2 mU/l* (*0.5–4.5 mU/l*)
- T_3: *2.0 pmol/l* (*2.8–7.1 pmol/l*)
- T_4: *4 µg/dl* (*norm 4.5–11.2 µg/dl*)

1.1.1 How Do You Interpret These Lab Values?

1.1.1.1 Anemia

During pregnancy, plasma volume increases dramatically, by about 45 %. The erythrocyte volume increases as well, but only by about 20 %, far less than the plasma. Therefore, anemia is commonly seen in pregnant women.

However, hemoglobin values under 10 g/dl (HCT <30 %) usually indicate iron deficiency anemia. In the presented case, at the time of the impending D&C, one must assume that the HCT was also low due to blood loss.

1.1.1.2 Leukocytosis

The leukocytosis was probably due to amnionitis.

1.1.1.3 Hypokalemia

Hypokalemia is often found in patients taking diuretics. In this case, hypokalemia is most likely a result of the β_2-agonist therapy. β_2 stimulation causes K^+ ions to move intracellular – an effect often seen in severely injured or trauma patients. The preoperative increased catecholamine levels stimulate the β-receptors and cause potassium influx into cells. However, total body potassium is unchanged.

1.1.1.4 Hypothyroidism

Mild hypothyroidism is often seen in pregnant women, usually due to iodine deficiency secondary to the elevated requirement for iodine during pregnancy.

Rescheduling an elective operation because of hypothyroidism is only done in cases with severe symptomatic deficiency. Rescheduling isn't an option in emergency operations.

1.1.2 Why Shouldn't Potassium Be Administered?

Preoperative potassium administration must be done with care, if at all, due to the dangers associated with giving potassium too quickly (such as cardiac arrest). It should only be considered if the total body potassium is reduced, such as when potassium is excreted, as with diuretics. Potassium administration is not indicated for Julia because the total body potassium is normal and the hypokalemia was due to the intracellular influx of potassium.

According to current practice, potassium administration is only indicated in severely ill patients (CHD, existing arrhythmia, digitalis therapy), or in high-risk operations (cardiac or thoracic surgery, major vessel procedures). Patients without such risk factors can tolerate a potassium level as low as 2.5 mEq/l. Furthermore, chronic hypokalemia is better tolerated than acute hypokalemia. In chronic hypokalemia, the resting potential tends to normalize over time, whereas in acute changes of extracellular potassium levels, there is no time for equilibration, causing an increased risk for cardiac arrhythmias.

>> The patient didn't appear to be much different from the many other postpartum patients Dr. Damel had seen. She was obviously exhausted

from the birth and was asleep. Her complexion hinted at her anemia. The obstetric nurse had already positioned her legs in the stirrups and was prepping for the operation. Anesthesia technician Corinne finished checking the anesthesia machine and hooked up the standard monitoring: *ECG*, *pulse oximeter*, *and a blood pressure cuff*.

1.1.3 Which Form of Anesthesia Would You Choose?

In anesthesia literature, there is consensus that pregnant and laboring patients are at increased risk of aspiration of gastric contents. For this reason, general anesthesia with "rapid sequence induction" (RSI) is generally performed. The goal of the RSI is to secure the airway as fast as possible (Sects. 8.1.2, 8.1.3, 8.1.4, and 8.1.5).

After preoxygenation with 100 % oxygen for 3 min, anesthesia is induced by a hypnotic agent followed immediately by a fast-acting muscle relaxant (succinylcholine or rocuronium).

The Sellick maneuver (cricoid pressure) is generally applied to prevent regurgitation of gastric contents. However, its use is somewhat controversial. Studies [4] have shown that:

- The applied pressure is usually not high enough to close the esophagus.
- Relaxation of the lower esophageal sphincter occurs as a reflex to the pressure.
- Forced vomiting could cause rupture of the esophagus.

It is important to remember that a RSI should only be used when an easy airway is expected. If a difficult intubation is expected, an awake fiberoptic intubation or a regional anesthetic should be considered.

>> *After the preparations were finished*, *Dr. Damel went to the patient to evaluate her and obtain informed consent. Julia Mase*, *however*, *was so exhausted that a discussion was not possible. He glanced at the monitor and saw*:

- *HR*: *130/min*
- *Blood pressure*: *140/80 mmHg*
- S_pO_2: *74 %*

1.1.4 What Is Your Most Important Action Now?

Due to the tachycardia and the poor oxygen saturation, the most important action is to improve the oxygenation. Follow the ABC-rule:

- A(irway): open airway (check tracheal tube).
- B(reath): give oxygen.
- C(heck): also, auscultation of the lungs is essential.

Simultaneously, check the location and function of the pulse oximeter to rule out artifact or malfunction.

>> *The* S_pO_2 *appeared correct*, *with a good wave form. With spontaneous breathing and 100 % oxygen administered by a tight-fitting mask*, *her* S_pO_2 *increased to 91 %. Dr. Damel delegated the mask holding to Corinne*, *the anesthesia technician*, *and auscultated the patient's lungs. The breath sounds were equal and symmetric with no bronchospasm. There were*, *however*, *discontinuous high crackles*. "*She's losing too much blood – we must remove the placenta immediately – put her to sleep now*!" *yelled the OB*.

Dr. Damel took the mask back and injected 120 mg propofol, *then 80 mg succinylcholine. The anesthesia technician Corinne applied cricoid pressure. After waiting a minute until the muscle fasciculations ceased*, *the* S_pO_2 *decreased to 85 %. Dr. Damel took the laryngoscope and began placing it into her mouth*. "*No*!" *he thought in panic when he realized her mouth only opened about 3 cm*, *and only the tip of the epiglottis was visible*.

1.1.5 What Do You Do Next?

The most common causes of difficult laryngoscopy are usually:

- Insufficient depth of anesthesia/muscle relaxation
- Suboptimal patient positioning

Both should be checked (and corrected). Improving positioning might be achieved by raising the head to the sniffing position. In this case, if visualization of the larynx is not improved, then the patient should be ventilated by mask ventilation with a peak pressure of <20 cm H_2O.

If not applied earlier, a second person should apply cricoid pressure to prevent insufflation of the stomach.

>> Position optimization with the help of a pillow did not improve the visualization of the larynx. With S_pO_2 of 80 %, Dr. Damel aborted the intubation attempt and began careful mask ventilation, while Corinne, the anesthesia technician, continued cricoid pressure. As the oxygen saturation slowly reached 90 %, he turned to Corinne and asked "Please get the difficult airway kit from the OR!" The OR nurse then applied cricoid pressure.

1.1.6 Which Airway Adjuncts Can Help You to Intubate in This Situation?

There are numerous airway and intubation assist devices on the market, such as a variety of types of laryngeal mask airways, Fast Track laryngeal mask airway, C-Track, various blades, videolaryngoscopes, fiberoptic bronchoscopes, and Glidescopes. A general recommendation cannot be given about which instrument to use. The decision is best left up to the individual anesthesia provider, as the optimal choice depends on individual experience and the particular patient and situation.

The highest priority in the presented case is securing the delivery of oxygen and simultaneously preventing aspiration. Since simple intubation is not possible, a laryngeal mask airway (ProSeal) may be chosen because these are easy to use, can be quickly inserted, and allow insertion of a gastric tube. The procedure is anticipated to be very brief.

With the countless possibilities available, one must also consider allowing the patient to wake up and then perform an awake fiberoptic intubation. The unexpected difficult airway is a rare, but dreaded, complication in anesthesia. It is important to thoroughly know the American Society of Anesthesiologist's (ASA) Difficult Airway Algorithm [1] and to have a difficult airway cart immediately available.

The use of various intubation assist devices should be regularly practiced in order to be proficient in their use in an emergency situation. In addition to the training, the proper equipment must be immediately available. The additional pressure of time constraints during emergencies increases mistakes, so procedures are better performed when standardized.

The ASA's Difficult Airway Algorithm is important to keep in mind in the event of an unexpected difficult intubation [1]. In addition to memorizing the various steps of this algorithm, it is also important to individualize the ASA's algorithm to fit your own practice based upon your skills and available equipment. An example of such an algorithm is shown in Fig. 1.1.

>> After a few minutes, anesthesia technician Corinne returned out of breath. In the meantime, Dr. Damel continued to mask ventilate the patient and gave 150 mg propofol in divided doses to maintain anesthesia.

He decided to use a ProSeal™ LMA, which proved to be difficult to insert with the restricted mouth opening; opting for a stylet after finger guidance didn't work.

The patient was easily ventilated using the ProSeal™ LMA. A gastric tube was inserted and drained 500 ml of gastric contents. The patient was given fentanyl 200 µg IV. The OB began the D&C, which took 15 min. Anesthesia was maintained with 1 MAC sevoflurane. Due to blood loss, which was very difficult to estimate, the patient received 500 ml of 5 % albumin.

1.1.7 What Should Be Done During Postoperative Care?

Due to the oxygenation difficulties, the patient should remain intubated and be admitted to the ICU for mechanical ventilation. The airway must be secured by an endotracheal tube, perhaps with the help of a fiberoptic bronchoscope.

The ICU team needs to know that extubation must be carried out with a difficult intubation cart handy, and with an anesthesiologist standing by.

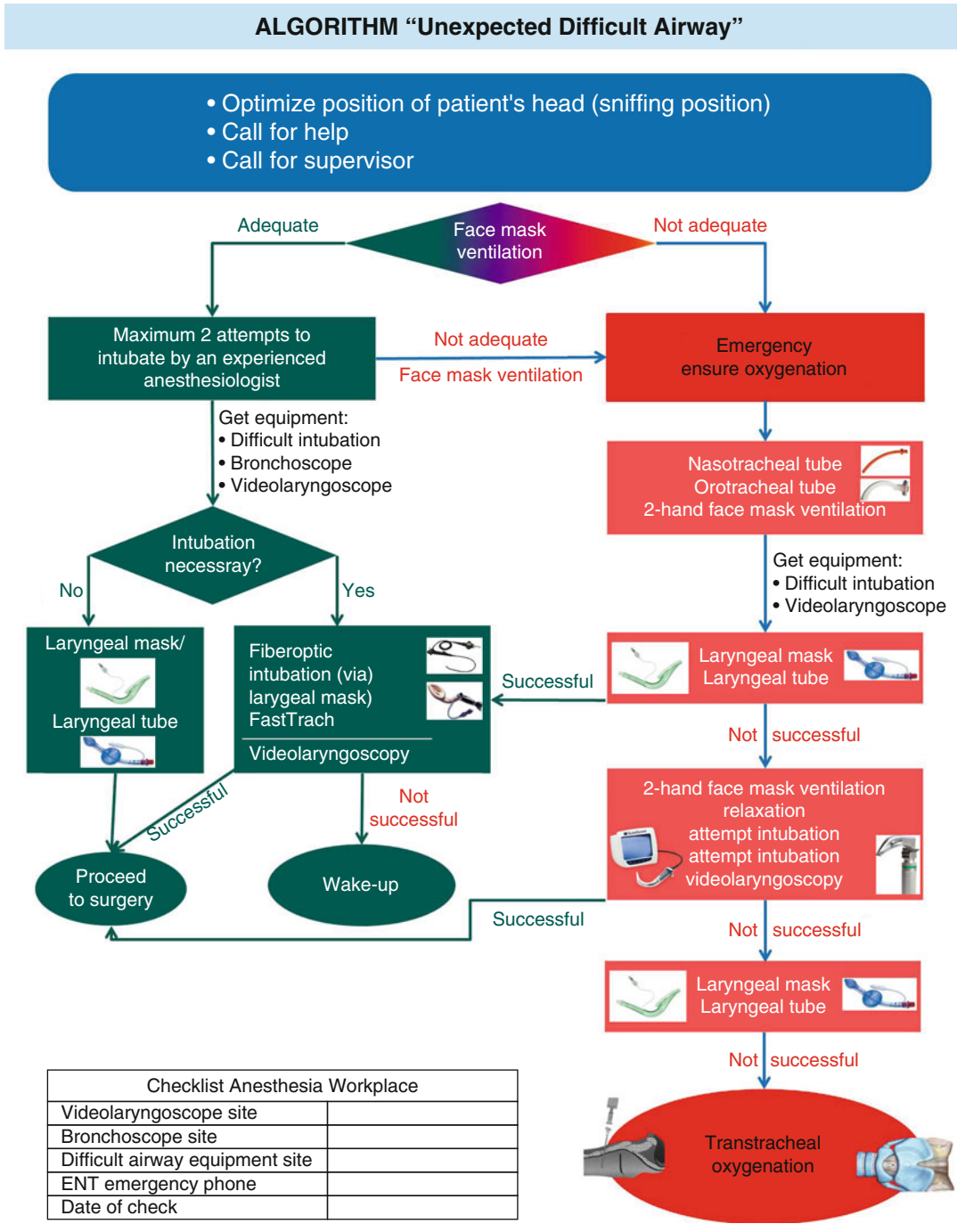

Fig. 1.1 Algorithm for the unexpected difficult airway of the Department of Anesthesia and Intensive Care of the University Hospital Dresden

Upon arrival in the ICU, further laboratory studies are required: CBC, blood gas analysis, and chest X-ray, and, finally, a specific treatment plan must be decided upon.

1.1.8 Interpret the Pulmonary Findings and the S_pO_2 Value in This Patient

Assuming that the oxygen transport function of the erythrocytes is normal, the hypoxemia was most likely caused by ventilation–perfusion mismatch. The low S_pO_2 could have been caused by:

- A perfusion disorder (e.g., by an embolus or pulmonary vasoconstriction)
- A diffusion disorder (e.g., pulmonary edema or pneumonia)
- A ventilation disorder (e.g., bronchospasm or hypoventilation)

The high fine crackles were caused by alveolar fluid accumulation; aspiration causes course crackles. Many different diagnoses must be considered with this patient, such as infection or pulmonary edema. Considering the sudden and intense occurrence, pulmonary edema is the most likely diagnosis. The patient had been treated with tocolytics (β-mimetics) for many days. The ensuing increase of antidiuretic hormone (ADH) secretion leads to fluid retention which, together with the reduced colloid pressure of pregnancy, leads to pulmonary edema [2].

Another possibility is amniotic fluid embolism, which typically presents in two phases: first with initial pulmonary hypertension and right-sided heart failure, followed by a decrease in cardiac output and lung edema, hypoxia, and hypertension.

1.1.9 Is the Aspiration Risk in Pregnant Women Actually Increased?

The answer to this question is not easy; one must probably first differentiate between an emergency procedure (as in this case) and elective procedures.

The concept of increased risk of aspiration in pregnant women was first described by Dr. Mendelson in 1946, and has since become well established in the heads and in the books of anesthesiologists worldwide. In the 1940s, women in labor were allowed to eat and drink freely. In the work of Dr. Mendelson, inexperienced young residents would carry out the anesthesia with ether masks [6] which are known to cause nausea and vomiting. The conclusions of these observations are well known – pregnant women have an increased risk of aspiration, being that intra-abdominal pressure is increased and the lower esophageal sphincter pressure is decreased.

More recently, some research has suggested that despite hormonal and abdominal pressure changes, gastric emptying is not delayed during pregnancy [7]. In addition, a large study of 1067 elective C-sections using laryngeal mask airways showed no increased aspiration risk [5]. Slowly, anesthesiologists are beginning to speak out against the theory of increased risk of aspiration in pregnant women, at least during elective C-sections [3]. In the presented case, however, the situation was different because pain, infection, and stress reduce gastric emptying, regardless of pregnancy.

The topic of delayed stomach emptying in pregnant women is an example of how difficult it is and how long it takes to change an established medical (and nonmedical) dogma. The influence of medical books is enormous, due to the fact that the authors do not take the time to critically examine dogmas.

1.2 Reflections on the Case/ Case Analysis

1.2.1 Which Medical Errors Do You See in the Presented Case?

1.2.1.1 The Patient's Preop Check, Performed by the Anesthesiologist

Due to the emergency situation, a complete medical history was no longer obtainable. However, an airway examination with a check of the mouth opening should always be performed. If the patient is obtunded and unable to cooperate with

the exam, the patient's mouth can be opened manually. The medical record also showed a low HCT. A stat HCT should have been drawn (done, e.g., by the OB nurse) and would have provided valuable information.

1.2.1.2 Patient Monitoring During Labor and Delivery

The patient was most likely already hypoxemic when the anesthesia team arrived. It is likely that the fetus was in danger of hypoxia during delivery.

A retained placenta is very dangerous and a postpartum D&C is challenging:

- This is primarily due to the blood loss. The danger was increased in this patient because of the previous days spent with tocolytic therapy and the early stage of pregnancy.
- Secondly, pulmonary edema after or during tocolysis is a well-known complication. Careful monitoring of vital signs should have been carried out regardless. The OB knew the seriousness of the procedure and potential for blood loss. Nevertheless, an HCT check (which can be done easily on most labor and delivery floors) did not occur.

1.2.1.3 Procedures in the Labor/Delivery Room

What occurred in the labor delivery room will be discussed below.

1.2.2 Which Systems Failures Can Be Found in the Presented Case?

1.2.2.1 Patient Monitoring by the Obstetric Team

This point was already discussed under medical errors (Sect. 1.2.1). However, systems failures often occur because they are not clarified by protocols and guidelines for care. Even though vital sign monitoring primarily affects the obstetric team, anesthesiologists should make sure the monitoring is carried out. In addition to increasing patient and fetal safety during birth, precious time can be saved during emergencies.

1.2.2.2 Manual Placental Removal in the Delivery Room

It is customary to carry out such "small procedures" in the labor/delivery room, stemming from the desire to optimize the organization of labor and delivery. However, the decision not to carry out these procedures in a proper operating room reduces patient safety. Due to the severity of the patient's condition (e.g., somnolence, hypovolemia, tachycardia, low S_pO_2), the patient should have been moved into an OR, either on the labor floor or, if none available, into the main OR area.

- Medically, there is:
  - Limited positioning possibilities for the patient in bed
  - No possibility to convert to a laparotomy
  - Inadequate intubation conditions
- Logistically there are:
  - Problems with instrumentation, device, and medication selection (i.e., ventilator, accessories, airway adjuncts, IV warmers, etc.)

1.2.2.3 Patient Informed Consent

A complete preanesthetic evaluation and informed consent was not possible in the emergency situation. However, the patient had been admitted several days earlier, with amnionitis and contractions. Thus, she had an increased likelihood of C-section delivery. Such patients need a preanesthetic consultation, so that a proper evaluation occurs before an emergency situation arises. Such care protocols are very important.

1.2.3 Evaluate the Nontechnical Abilities of the Personnel Involved

1.2.3.1 Communication

Communication between the anesthesia and OB team was minimal. Each department was concerned with their own specialty care, without seeing the importance of informing the other team members about patient condition, treatment status, or difficulties. Note that the anesthesiologist didn't tell the OB about his pulmonary findings or diagnosis.

1.2.3.2 Procedure

The involved specialists (OB and anesthesiologist) are on the same hierarchical level. The anesthesiologist in the presented case accepted the OB's leading role. As he took the role of a follower, however, he made hasty and intermittently poor decisions.

For example, he failed to perform a preoperative airway exam to check the mouth opening or obtain a recent HCT, as the OB demanded immediate induction of anesthesia.

1.2.3.3 Teamwork

As already discussed, the two teams did not work together; the labor and delivery team was exceptionally focused on the D&C and not the patient's overall medical condition.

1.2.3.4 Resource Management

Although the anesthesiologist used the resources available to him (asked the anesthesia technician to take over face mask ventilation), he should have asked for additional resources. Specifically, he should have enlisted the help of the obstetric nurse to hook up the monitors and draw an HCT or get the difficult airway cart.

1.2.3.5 Decision-Making

Both physicians made decisions quickly but separately, e.g., immediacy of surgery and management of the difficult airway. Time pressure and emotional stress often lead to suboptimal decision-making and failure to consider the best treatment options. Preoperative discussion by the physicians and communication during the procedure would have improved decision-making.

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Case 2: Elbow Dislocation with Fracture

2

Jana Kötteritzsch, Mike Hänsel,
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Contents

2.1 Case Introduction

>> At 95 kg and 173 cm, Frank Parker knew that he was a little overweight. He was 46 years old and on β-blockers to control his blood pressure. His family practitioner said his arterial hypertension comes from his job and from "a couple of extra pounds around the waistline" – at least, that's how Mr. Parker put it. As boss of his own construction company, deadlines were his bread and butter.

Today Mr. Parker's schedule was very busy again. He was up at 5:30 a.m. to get ready for the day. His wife and kids were still asleep as he finished breakfast. He felt fine as he parked his car

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at the construction site. He was just a little annoyed by his constant headache. The headache began suddenly 2 weeks ago. It had gotten better the past few days, but was still there.

Mr. Parker's construction supervisor greeted him warmly. Together they went to the scaffolding to get an overview of the construction work. Mr. Parker stepped up the first rung of the ladder, then groaned, reached for his head, and fell to the ground. The supervisor jumped to his side, shocked. Mr. Parker was unresponsive, and his left arm was positioned abnormally. The supervisor froze for a moment in confused panic; then he grabbed his cell phone and called 911.

2.1.1 What Is Your Diagnosis?

The symptoms presented are typical for a subarachnoid hemorrhage (SAH). In many patients SAHs occur repeatedly over time. The so-called warning bleeds are often accompanied by acute headaches which decrease in intensity over time. The warning bleeds are usually not recognized as an SAH, and often 2 weeks later the serious SAH occurs. This SAH usually presents with a sudden headache, often during physical exercise, and with vomiting or a decreased level of consciousness. Neck stiffness or other signs of meningeal irritation can be present. Depending on severity and location, various levels of neurological deficits can occur. The severity scale of SAH is shown in Table 2.1. It is based on the classifications of the WFNS (World Federation of Neurological Surgeons) [7] and of Hunt and Hess [4]. The latter is preferred since it is based on the sum score of the Glasgow Coma Scale and the presence of focal neurological signs. The patient's outcome is dependent on the severity of the SAH.

Table 2.1 Classification of SAH

WFNS classification [7]		Grade	Classification Hunt and Hess [4]
Clinical appearance	Glasgow Coma Scale		
	15	**0**	Asymptomatic recognition of an unruptured aneurysm
No motor deficit	15	**1**	Asymptomatic with minimal headache and nuchal rigidity
No motor deficit	13–14	**2**	Moderate to severe headache, nuchal rigidity, no neurological deficit other than cranial nerve palsy
Motor deficit	13–14	**3**	Drowsiness, confusion, or mild focal deficit
With or without motor deficit	7–12	**4**	Stupor, moderate to severe hemiparesis, possibly early decerebrate rigidity and vegetative disturbances
With or without motor deficit	<7	**5**	Deep coma, decerebrate rigidity

2.1.2 What Is the Most Important Differential Diagnosis, and What Are Other Possible Differential Diagnoses?

The most important differential diagnosis in this case is head injury following a fall.

Therefore, the paramedics arriving on scene must take spinal precautions. Other differential diagnoses are various causes of syncope (Overview).

2.1.2.1 Possible Causes of Syncope

- Cardiac syncope
  Arrhythmia disorders or low output syndrome.
- Circulatory syncope
  Possible causes: vasovagal, orthostatic, hypovolemia, postprandial, pressure related, carotid sinus syndrome, vena cava compression syndrome, medication side effect, autonomic neuropathy.
- Cerebral syncope
  Cerebral syncope is often seen in epilepsy, cerebrovascular insufficiency, stroke, and various intracranial bleeds or narcolepsy.
- Metabolic syncope
  Can be caused by hypoxia, severe anemia, hypoglycemia, or electrolyte disorders.
- Hypothermia

Table 2.2 Glasgow Coma Scale (<8 points = severe neurological dysfunction)

Neurological function	Patient's reaction	Points
Eye opening	Spontaneously	4
	In response to voice	3
	In response to painful stimuli	2
	Does not open eyes	1
Best verbal response	Oriented	5
	Confused	4
	Utters inappropriate words	3
	Incomprehensible sounds	2
	No response	1
Best motor response	Obeys commands	6
	Localizes painful stimuli	5
	Flexion/withdrawal to painful stimuli	4
	Abnormal flexion to painful stimuli	3
	Extension to painful stimuli	2
	No response	1
Total		3–15

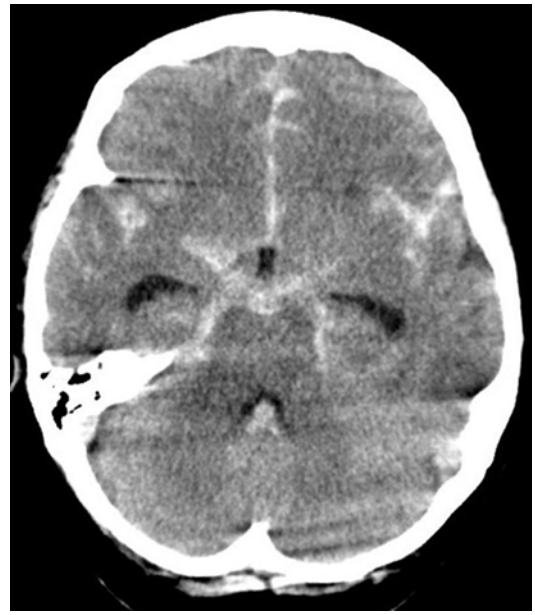

Fig. 2.1 For interpretation of the CT scan see 2.1.4

>> The medics who arrived 12 min later had to navigate through the construction workers who had gathered around. Paramedic Charles immediately checked vitals and placed Mr. Parker supine. He determined that Mr. Parker was somnolent, was tachypneic, but with adequate tidal volume. Mr. Parker moved in response to painful stimuli. Blood pressure was 180/95 mmHg, pulse was regular and strong with a frequency of 80/min. "Who saw what happened?" asked paramedic Charles. The agitated construction supervisor stepped forward, but his recollection was less than adequate. Even so, Charles knew what he had to do next.

2.1.3 What Needs to Be Done?

According to Table 2.1 Mr. Parker has the suspected diagnosis of a Grade 4 SAH. The medic does not have the necessary clues to come to the exact diagnosis, especially since it is not obvious if the loss of consciousness occurred before or after the fall. Table 2.2 shows the Glasgow Coma Scale (GCS).

On physical exam, Mr. Parker opened his eyes in response to pain and had no verbal response, but showed flexion/withdrawal to pain – thereby obtaining a GCS of 7 points.

2.1.3.1 Emergency Care of Trauma Patients

- **In the emergency care of trauma patients, there is one basic rule: "treat first what kills first."**
- **Securing the airway and providing oxygen has the highest priority. Experts recommend intubation after trauma if the GCS is 8 or below [3].**

>> The EMT started an IV and checked Mr. Parker's blood sugar level, which was normal. His trachea was intubated by the medic, and no signs of aspiration of gastric contents were found. Then the arm was splinted, and Mr. Parker was transported to the city's level 1 trauma center. After being handed over to the care of the trauma team, Mr. Parker was sent immediately for a head CT scan, spinal CT, and an X-ray of the left arm.

2.1.4 What Is Your Diagnosis of the Head CT?

The CT (Fig. 2.1) shows subarachnoid blood in the area of the circle of Willis, the basal cisterns, the anterior cerebral fissure, and the fourth ventricle. It is seen as a hyperdense (white) structure.

2.1.5 Which Cerebral and Systemic Complications May Arise Post-SAH?

The most important complication of SAH is recurrent hemorrhage. It usually occurs in the first few days following the event. The risk of recurrent hemorrhage from an untreated aneurysm is 35–40 % in the first 4 weeks, decreasing after the first month 1–2 % per day and finally stabilizing at 3 % per year [2]. Apart from the acute rebleed, patients are at risk of intracerebral and intraventricular hemorrhages.

Due to the altered cerebrospinal fluid reabsorption after SAH, occluding and malabsorption hydrocephalus often results. This leads to an increase in intracranial pressure, which acutely endangers the patient. The head CT shows the typical ventricular dilation, often together with CSF diapedesis, and additional signs of increased intracranial pressure such as flattening of the gyri. The therapy of choice is insertion of an external ventricular CSF drain (EVD).

Cardiac complications are not rare in SAH patients. ECG changes can be seen in ¾ of the patients; the varied symptoms include sinus brady-/tachycardia, QT prolongation, heart blocks, ST elevation and depression, T-wave changes, and pathological Q waves.

Cardiac markers can also be raised. ECHOs often show wall dysfunction and histopathological changes in the myocardium. These signs can mimic acute MI. Acute heart failure can lead to arterial hypotension, pulmonary edema, cardiac arrest, and sudden cardiac death. However, coronary angiography shows typically no evidence of coronary artery stenosis. Causes of the cardiac signs are excessive releases of epinephrine and norepinephrine, as well as imbalances in the parasympathetic nervous system.

Once the acute dangers of the SAH have passed, the patients are at risk for cerebral vasospasms. Vasospasm can cause cerebral ischemia, which further worsens the prognosis. Vasospasm usually occurs between the fourth and fourteenth day (maximum on the seventh day) and last approximately 3 weeks after the SAH. Permanent neurological deficits due to the recurrent hemorrhage/ischemia include paralysis, cognitive disorders, epilepsy, neuroendocrine dysfunction, and dysfunction of the sleep wake cycle.

>> The radiological tests showed an advanced SAH with beginning CSF accumulation and an elbow fracture with dislocation. Mr. Parker's pupils remained reactive. The anesthesia team inserted hemodynamic monitoring including an arterial line and a central venous catheter (CVC). Mr. Parker was then taken to the OR for the insertion of an EVD. The EVD delivered bloody CSF. ICP monitoring showed normal values. Digital subtraction angiography revealed a left anterior cerebral artery aneurysm. The neurosurgeon decided on immediate intervention, and the aneurysm was clipped the same day.

After surgery, Mr. Parker was transferred to the ICU intubated, ventilated, and sedated. Mrs. Parker had been waiting there for several hours already. She seemed lost in shock and had her two children with her.

2.1.6 What Would You Say to Mrs. Parker About Her Husband's Prognosis?

As outlined in Sect. 2.1.5, treatment of the bleeding source prevents rebleeding. The prognosis of SAH is not definitively known. A SAH is potentially life-threatening, and in an uncomplicated SAH, possible neurological deficits can appear as early as 14 days after the original bleed.

2.1.7 What Is the Basic Management of SAH Patients in the ICU?

The first goal is preventing rebleeding. It is therefore important to identify the source of the hemorrhage – usually an aneurysm. Early intervention, within 24–36 h, is recommended to reduce the risk of rebleeding. Cerebral aneurysms can be treated in either of the following two ways:

- Neurosurgically via a craniotomy and clipping
- Neuroradiologically via endovascular coiling

Lowering the blood pressure reduces the likelihood of rebleeding, especially in untreated aneurysms. But hypotension also increases the danger of cerebral ischemia, if the patient develops vasospasm. As a compromise to both risks, blood pressure in patients with untreated aneurysms should be maintained below 180 mmHg systolic and 100 mmHg diastolic.

If the aneurysm has been treated, the main goal is preventing cerebral ischemia due to vasospasm. Calcium channel blockers appear to have a beneficial effect and are begun early after the diagnosis of SAH. The standard dose is 60 mg nimodipine every 4 h PO or via gastric tube. IV administration is not recommended due to hypotension. Nimodipine therapy should be continued for 21 days.

The second mainstay of vasospasm therapy is the so-called triple-H therapy: hypervolemia, hypertension, and hemodilution. Triple-H therapy improves cerebral perfusion; however, it is unclear if cerebral infarction can be avoided. Side effects of the triple-H therapy are not uncommon and include cerebral edema, repeat bleeding, hyponatremia, and heart failure accompanied by pulmonary edema. Hence, aggressive triple-H therapy is not appropriate for all patients. Some neurointensivists prefer to avoid volume overload and instead restrict the therapy to arterial hypertension and hemodilution (double-H therapy). Scientific evidence supporting triple-H or double-H therapy is relatively weak.

Target values of the volume and blood pressure therapies are CVP of 8–12 cm H_2O, hematocrit of 30–35 %, and blood pressure of 20 % above the individual's normal value. Hypovolemia must be avoided. However, there is no evidence that hypervolemia is better or worse than normovolemia.

New therapeutic, largely experimental treatments to prevent cerebral vasospasm include intraventricular administration of thrombolytic agents; vasodilators; platelet aggregation inhibitors; anticoagulants; neuroprotection agents, such as tirilazad; and the administration of statins, magnesium, nitric oxide (NO) donors, endothelin antagonists, potassium channel activators, and erythropoietin. The quality of the scientific evidence for these treatments varies; in general, more research is necessary.

If cerebral vasospasm is detected, triple- or double-H therapy is begun. Further options to treat vasospasm include endovascular balloon angioplasty and intra-arterial administration of vasodilators [2].

>> Standard SAH therapy was initiated in the ICU. The trauma surgeons indicated the need for surgical repair of the elbow fracture. However, considering the acute cerebral trauma, they agreed to wait a few days. Mrs. Parker mentioned that her husband had been complaining of a bad headache lately. "*He thought his blood pressure was too high and he wanted to go to his doctor. If only he hadn't postponed the appointment*."

On POD 1, a follow-up head CT was done. It showed correct EVD placement without signs of cerebrospinal fluid accumulation. Blood was still visible in the subarachnoid space. Since the ICP was constantly between 20 and 23 mmHg, the sedation with sufentanil and midazolam was continued.

On POD 2, the cerebral pressure fell to an acceptable level. "*The fracture must be reduced*," *insisted the trauma surgeon during the morning ICU rounds. Dr. Theresa, the intensivist in charge, agreed.* "*I will prepare Mr. Parker for the OR*," *she said.*

2.1.8 Hmmm… What About Informed Consent in These Patients?

The problem of legal competency to give informed consent is extensively discussed in Case 24 (Sects. 24.1.6 and 24.2.1). The patient is not capable of giving consent, due to the acute SAH and sedation. The orthopedic surgeon stated that the patient required immediate elbow surgery. The next-of-kin, in this case, Mrs. Parker, was available to provide informed consent for the procedure. Two-physician consent may be used for an emergency procedure involving loss of life or limb, if there is no next-of-kin available.

>> Mrs. Parker was reached at about 2:30 p.m. The phone rang as she came in with her children.

She had just picked them up from preschool. As Dr. Theresa spoke, Mrs. Parker suddenly had to sit down. She felt as if the floor underneath her had fallen away. "When will he be operated on?" she asked tearfully, even though she actually felt relieved. She had feared worse news. "I would like to visit my husband, but I can't come too late because of the children," she said. "My mother has come to help out, but I need to be back at home in the evening."Dr. Theresa promised to check and get back to her – but when she called the trauma surgeon, he had already forgotten Mr. Parker due to all his other patients. He apologized and soon after requested an operation slot for Mr. Parker.

It was almost 4 p.m. and almost all operating rooms were still going. The anesthesia department supervisor, Dr. Eldridge, was not happy about the emergency case. Fortunately, he had an anesthesia resident available. Dr. Armstrong was only in his second year residency, but he had already had a trauma surgery rotation for several weeks. It was only an upper extremity procedure, and Dr. Eldridge was able to supervise it.

Mr. Parker was hooked up to the transport monitor. Dr. Theresa adjusted the ventilator. The medication infusion pumps were attached to the bed with a special fixation device. The EVD was closed for the transport. Dr. Armstrong was waiting to transfer the patient onto the OR stretcher. "Ready for ICU medicine, doc?" Dr. Theresa asked, in an uppity tone of voice. Dr. Armstrong didn't know what to say, so he busied himself with checking the settings on the mobile ventilator. The ICU docs were at the end of their residency and tended to let everybody else know it. "I'll be different when I near the end of my residency" he thought to himself.

In medical school, Dr. Armstrong had heard and read about SAH, not that he could recall all the details now. The patient was intubated, sedated, ventilated, and receiving 0.09 µg/kg/min norepinephrine to maintain normotension. Dr. Theresa sketched out the details of the case and added that it was just another SAH. "I've closed the EVD and placed it on the mattress. You should open it later, then check that the filter does not get wet. Mr. Parker is tolerating mechanical ventilation well. Due to the slightly increased ICP, we've kept him sedated. The systolic blood pressure is 160 mmHg on norepinephrine. The CPP has always been higher than 70 mmHg. We've got two bags of crossmatched blood available – call for it if you need it," finished Dr. Theresa.

2.1.9 What Is CPP?

CPP stands for cerebral perfusion pressure and is a measurement of cranial perfusion. In most tissues, perfusion pressure equals the difference between the arterial and venous pressure. A special situation exists in brain tissue. Due to the limited space, the skull adds an additional external pressure. In terms of physics, this is explained as a Starling Force. The CPP is calculated as follows:

$$\mathrm{CPP}[\mathrm{mmHg}] = \mathrm{MAP}[\mathrm{mmHg}] - \mathrm{ICP}[\mathrm{mmHg}]$$

where MAP is the mean arterial pressure and ICP is the intracranial pressure. If the pressure of the jugular vein/central vein pressure (CVP) is higher than the ICP, then CVP is used for the equation. Physiologically, the cerebral blood flow (CBF) is autoregulated to maintain a constant CPP between 50 and 70 mmHg over a wide range of MAP. An extended drop of the CPP below 50 mmHg leads to ischemic cerebral injury [9].

>> Dr. Armstrong hadn't cared for many critically ill patients yet, with so many lines ("spaghetti") to watch out for – but an EVD was only a minor procedure after all. Because he didn't really remember so much about SAH, he asked if there was anything else he needed to be alert for. "Yes!" answered Dr. Theresa. "To prevent the common side effect of cerebral vasospasm, stick with the Triple-H-Therapy with hemodilution and hypertension," she said. "Don't let the systolic blood pressure fall below 160 mmHg, and give sufficient fluid replacement," chimed in Dr. Theresa's less-experienced assisting physician. "Alright, I've got it!" thought Dr. Armstrong, before asking one more question: "Should I continue infusing midazolam and sufentanil during surgery?" "I can't believe you just asked me that," said Dr. Theresa. "You weren't paying

attention in medical school, were you? Volatile anesthetics can increase ICP!"

As they reached the operating room, the surgical nurse informed Dr. Armstrong that the patient would be positioned prone. "That should be okay," he thought. Working together with a CRNA, they taped the eyes, rolled the patient over, sorted the lines, assured proper head placement and cushioning, and eventually the patient was ready for surgery. Anesthesia was maintained with midazolam and sufentanil. Dr. Eldridge, the attending anesthesiologist, stopped in the OR and quickly checked the monitors and infusion pumps. The heart rate was 80/min, blood pressure 155/85 mmHg, oxygen saturation of 99 %, and temperature of 38.7 °C. The norepinephrine infusion was set to 0.2 µg/kg/min. Dr. Armstrong had deepened the sedation and therefore was not worried about the increased catecholamine requirement. Dr. Eldridge was then called urgently to another OR. Immediately before skin incision, Mr. Parker received 50 mg rocuronium IV. Dr. Armstrong, satisfied with his patient care thus far, sat down on his stool to fill out his anesthesia record.

2.1.10 Which of the Patient's Values Listed Above Is Worrisome and Requires Treatment?

A patient's increased body temperature of 38.7 °C is abnormal. Fever is defined as a central body temperature of >38.3 °C [5]. Fever occurs in about 70 % of critically ill patients in the ICU. According to Plaisance and Mackowiak, fever is "… a complex physiologic reaction to disease involving a cytokine-mediated rise in core temperature, generation of acute-phase reactants, and activation of numerous physiologic, endocrinologic, and immunologic systems" [8]. This reflects the complexity of the response of an organism to an inflammatory or infectious stimulus. Increased body temperature is very common in SAH patients.

The occurrence of fever in critical care patients is associated with an increase in mortality, morbidity, and days spent in ICU. One must differentiate between the cause of the fever: infectious or not. In fevers caused by bacteria or fungal infection, antibiotic therapy is essential. Fever, per se, does not always need to be treated. The positive effects of fever (bacterial, immune system stimulation, induction of the heat shock response) should be weighed carefully against the negative effects (increase of energy and oxygen requirement, increase in cardiac output, loss of fluids, increased effort of breathing, and changes in the CSF/blood barrier permeability). An extensive list of noninfectious causes of fever is presented in the overview.

2.1.10.1 Possible Noninfectious Causes of Increased Temperature

- Central nervous system causes:
  - Cerebral infarction
  - Intracerebral bleeding (ICB)
  - SAH
  - Traumatic brain injury
  - Alcohol and medication withdrawal syndrome
- Intrathoracic causes:
  - ARDS
  - Pneumonitis (post-aspiration)
  - Pulmonary embolism
  - Myocardial infarction
- Abdominal causes:
  - Intestinal ischemia
  - Gastrointestinal bleeding
  - Cholecystitis in the absence of cholelithiasis
  - Pancreatitis
  - Liver cirrhosis
  - Peritoneal carcinoma
- Endocrine causes:
  - Adrenal failure
  - Hyperuricemia
  - Hyperthyroidism
- Vascular causes:
  - Phlebitis/thrombophlebitis
  - Vasculitis
  - Deep vein thrombosis
- Nonspecific causes:
  - Posttransfusion fever
  - Postoperative fever (up to 48 h postoperative)
  - Medication fever

- Hematoma
- Neoplastic fever
- Transplantation rejection
- Contrast medium reaction
- Dehydration

Due to the diversity of the etiologies of fever, a general recommendation of when and how to treat fever cannot be given. However, in traumatic and nontraumatic brain injuries, a temperature <38.3 °C is desired. Hyperthermia increases cerebral metabolic demand, and may increase ICP, especially in the case of blood–brain barrier dysfunction, in which hyperthermia may exacerbate vasogenic cerebral edema. In SAH, hyperthermia worsens neurologic injury and patient prognosis. However, it is unclear if fever is the cause of further neuronal injury, or simply an expression of the extensive cerebral injury [1]. Also, it is not yet known if prevention of hyperthermia is associated with a reduction in mortality and morbidity. The European Brain Injury Consortium guidelines support antipyretic treatment at least for management of severe brain injury in adults [6].

>> The surgery required a tourniquet and proceeded very slowly. The blood loss was minimal. However, Dr. Armstrong had to continually increase the rate of the norepinephrine infusion. He wondered a little about that. After 45 min, 0.4 µl/kg/min norepinephrine was needed in order to keep the systolic pressure at 160 mmHg. The heart rate remained at 60/min. He also infused copious amounts of IV fluids as the words of Dr. Theresa's recommended triple-H therapy echoed in his head, "Be sure to give enough fluids…"

Dr. Armstrong noticed that 800 ml urine had been collected within the first hour. He thought that it was unusual that the norepinephrine infusion had to be increased to 0.5 µg/kg/min. The blood loss was minimal, calculating the input and the output, and Dr. Armstrong realized that the fluid balance was positive. Mr. Parker currently had a heart rate of 55/min, which was not indicative of hypovolemia. At least Dr. Armstrong knew that much. The CVP was not helpful due to the prone positioning.

Dr. Armstrong wasn't comfortable at all with the situation. He slipped around to the surgical side hoping to see that the operation would soon be over. He even crawled under the drape to make sure that the infusion line was connected securely to the CVC. Finally he asked the circulating nurse to get him a new norepinephrine infusion from the pharmacy, just in case, and thought that perhaps everyone makes mistakes… Nurse Almut gave him a very annoyed look, but followed orders, and the new syringe changed nothing at all. "Ugh," thought Dr. Armstrong, "that wasn't it either. What's the problem?" Just as he wanted to call his attending Dr. Eldridge, the trauma surgeons mentioned that they were finished. The surgery had taken 1 h, the blood loss had been about 300 ml, the urine output was about 1,000 ml, and the norepinephrine requirement was now 0.6 µg/kg/min. Dr. Armstrong reduced the analgesia and sedation. "Alright, now I will be able to reduce the norepinephrine pump rate for sure."

It took another 15 min before the dressing was on and the final X-rays had been taken. The trauma team certainly wasn't in a hurry today. Finally Mr. Parker was positioned supine again, and everything would certainly be okay again, thought Dr. Armstrong. He finished his protocol and wondered again about the increased catecholamine requirement. Nurse Almut was sorting out the lines. Just as Dr. Armstrong was planning to push Mr. Parker out of the OR, he opened the patient's eyelids out of habit and was shocked. "Were the pupils half dilated before?" he asked his nurse. She shrugged. Dr. Armstrong suddenly got a very bad feeling. Actually, he was sure that the pupils were not so dilated earlier. He called his attending, Dr. Eldridge.

2.1.11 What Could Cause the Pupillary Change?

Symmetrical pupil dilation can be caused by generalized increased cerebral pressure, due to an increase in volume of one of the three intracerebral compartments:

- Neuronal tissue (±88 %)
- Cerebrospinal fluid (9–10 %)
- Blood volume (2–3 %)

If only one pupil is dilated, then the cause is most likely ICB, infarction, local neuronal edema, or nerve injury.

>> *Dr. Eldridge came immediately, listened as the anesthesia and surgical reports were given. Then, after all details had been recounted, he asked about the one most important piece of information which hadn't been accounted for: "What is the ICP now?" Dr. Armstrong looked at him with astonishment: he had no idea how the ICP should be measured. At this moment, he realized that the monitor for ICP was not hooked up. Dr. Eldridge took the EVD line in his hand. "Was this closed the whole time?" he asked. Dr. Armstrong nodded. Dr. Eldridge opened the EVD and 14 ml of cerebrospinal fluid spurted out. The ICP measurement was then 20 mmHg. Dr. Armstrong was relieved that it was only slightly elevated. Dr. Eldridge was less satisfied: "How was the patient positioned during the operation?" When he heard "prone, with the head level to the body," he just shook his head.*

2.1.12 Which Actions Must Be Undertaken Now?

A head CT is now recommended. Since the EVD was closed for over 2 h, and no CSF could flow out, there is an acute danger of acute hydrocephalus with beginning cerebral swelling. This rise in ICP increases the possibility of cerebral infarction.

Dr. Armstrong was deeply affected by his mistake and hoped the CT would not show new pathological developments. After the CT was finished, he brought the patient to the ICU. Mrs. Parker was already there waiting for her husband.

2.2 Case Analysis/Debriefing

2.2.1 What Are the Possible Causes of the High Urine Production?

The pathological diuresis could be due to a central diabetes insipidus. Hypertonic dehydration occurs due to inactivation of hypothalamic osmoreceptors; destruction of the posterior pituitary following intracranial trauma, infarction, or hemorrhage; or due to pituitary removal or a tumor. Clinically, polyuria with >30 ml/kg body weight urine per day is seen. Lab tests show a low specific weight of urine, a low urine osmolality, or hypernatremia. Treatment with 0.4–4 µg desmopressin (DDADP) IV one to three times per day is recommended. Alternately, DDAVP may be administered intranasally or orally.

The differential diagnosis includes elevated blood pressure as the cause of elevated diuresis, sometimes referred to as "forced diuresis." In patients with normal/low blood pressures, the triple-H hypertension often causes increased urine output. Diagnosis depends on the serum electrolytes and the measurements of the specific weight of urine and serum/urine osmolality. In addition, a cerebral salt-wasting syndrome may occur after SAH.

2.2.2 Which Medical Errors Do You See in the Presented Case?

2.2.2.1 Body Temperature

As discussed in Sect. 2.1.10, the body temperature of the patient must be lowered.

2.2.2.2 Volume Therapy/Monitoring

Arterial blood gas analysis was not performed during the entire period. Even though there was an insignificant loss of blood, the increased norepinephrine requirement and the elevated diuresis warrant blood gas analysis. Central line readings are not valid in the prone position; however, regular measurements might have been useful because recording changes can be helpful.

2.2.2.3 Positioning

Positioning of patients with cerebral pathologies can be dangerous due to the increased intracranial pressure. Prophylaxis includes positioning with 30° elevation. Discussion between the anesthesia and OR team is recommended. Almost all limb operations can be carried out in the supine position, which, in addition to the semi-sitting positioning, also allows regular pupil monitoring.

2.2.2.4 Monitoring of the Cerebral Pressure

Continuous ICP and CPP monitoring was neglected. Such monitoring is essential in patients with increased ICP. An important part of the monitoring is the EVD and drainage of CSF as needed.

2.2.3 Which Systems Failures Can Be Found in the Presented Case?

2.2.3.1 Scheduling the Surgery

The surgery was scheduled at short notice late in the day. The inexperienced resident had no time to become acquainted with knowledge required to take care of the SAH patient. The attending was busy running the OR schedule, as well as supervising another OR.

2.2.3.2 Preoperative Evaluation

The preoperative evaluation was done by the ICU anesthesiologist. This makes sense from the aspect of time organization, but actually it would have been more helpful if the anesthesiologist carrying out the anesthesia had had a chance to see the patient on the ward.

2.2.3.3 Supervision

The resident didn't receive adequate supervision from the attending physician, even though the attending knew that a critically ill patient was under care of an inexperienced anesthesiologist. It would have been better to assign Mr. Parker to a more experienced resident or an attending anesthesiologist who could be present throughout the case.

2.2.3.4 Algorithm Care of Patients with Increased ICP in Non-neurological Procedures

In the care of patients with increased ICP – with and without an EVD – vigilance is essential. Hospitals should design their own algorithms to prevent mistakes like those that occurred in this case.

2.2.4 Stop! Don't Turn the Page! Here Is a Little Assignment for You!

Read the following terms out loud, and then turn the page:

- Ureteral calculus
- Necrosis
- Myoma
- Propofol infusion syndrome
- Vaccination reaction
- Cymbalta
- Vitiligo
- Sagittal
- Laser
- Psoriasis
- Hyperthyroidism
- Vasopressin

Now write down all the terms which you can recall.

_____
_____
_____
_____
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_____
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_____

How many words did you remember? According to the Miller's Law, you should have recalled about 5–9. According to Ebbinghaus' Forgetting Curve, you were most likely to remember the first and last term.

What does this exercise have to do with our case?

As Dr. Armstrong accepted the patient into his care, he received extensive information: ventilation management, sedation, triple-H therapy, EVD, etc. The amount and type of information was overwhelming for a resident physician in the second year of training – without ICU experience. His insecurities and doubts were expressed in his repeated questioning about the case management, despite the cutting remarks he received in reply. One of the biggest mistakes during this patient's handover was that Dr. Armstrong was not allowed to repeat the summary of information. So the ICU physician, Dr. Theresa, had no chance to check that all the details had been understood. What was holding Dr. Armstrong back from getting help? There can be many possible reasons: fear of being blamed, protecting his reputation as being competent, feelings of false safety, comforts, readiness to take on risks, etc.

The human capacity for understanding and storage of information is limited. When humans reach their maximum capacity for understanding, they are required to reduce the amount of information they take in. This is exactly what Dr. Armstrong did, and the detail about the CSF drain didn't make it into Dr. Armstrong's conscious recollection. Moments of **information overload** allow for recollection and realization of mistakes (more on this topic in the next chapters).

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Case 3: Pancreatic Surgery

3

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Contents

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3.1 Case Introduction

>> It was another Sunday on call for Dr. Constantine, who had just finished his cold pizza, when his attending anesthesiologist, Dr. Eldridge, informed him that a patient with pancreatic cancer would soon enter the OR. The surgical team planned to do an explorative laparotomy, but it could end up being more complicated. Dr. Eldridge wanted Dr. Constantine to do a preoperative evaluation and obtain the patient's informed consent, then prepare for and carry out the anesthesia. "*It is always the same on these weekend shifts*," *thought Dr. Constantine*, "*my attending surfs the Internet, while the residents do all the work*." *Dr. Constantine was in his last year of residency, and the weekend shifts had long since lost their excitement*. "*Pancreas*

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surgery," *he mumbled to himself*, "*sounds totally elective to me – not an emergency for a Sunday*."

On the ward, *Dr. Constantine met Mr. Anderson*, *65 years old*, *who complained of nausea*, *vomiting*, *and weight loss*. "*Because of the nausea*, *I haven't been able to eat for 2 days*," *reported Mr. Anderson*. "*Ever since I received this IV*, *I'm not as thirsty as I was*." *continued the patient*. "*I think I better revise my judgment – this is an emergency operation*!" *thought Dr. Constantine*.

Mr. Anderson's medical history included headaches and dizzy spells. "*My doctor told me 2 weeks ago that I had high blood pressure. I was scheduled for a 24-h urine measurement – but then all this with the nausea began 2 days ago*." *explained Mr. Anderson. He took no medications. Five years ago an uneventful inguinal hernia repair had been performed with spinal anesthesia. Dr. Constantine checked the medical record for blood pressure recordings*, *but couldn't find an entry*.

3.1.1 Which Form of Anesthesia and Monitoring Do You Suggest?

3.1.1.1 Anesthesia

Due to the symptoms of a bowel obstruction, a "rapid sequence induction" (RSI) is recommended (Sect. 1.1.3). For procedures in the upper abdomen, especially when the extent of the operation is unknown, general anesthesia combined with a thoracic epidural anesthesia (TEA) will be beneficial. It allows the patient to have excellent pain control with few systemic side effects, thereby allowing early mobilization and oral intake.

The TEA may also improve hepatosplanchnic perfusion, has cardioprotective properties if it extends far enough cranially, and assists intestinal peristalsis [13].

3.1.1.2 Monitoring

Besides standard monitoring (ECG, pulse oximetry), an arterial catheter is indicated in this patient. The arterial line allows simple blood sampling for evaluation of the pH, Hb, and glucose, and Mr. Anderson has a history of untreated hypertension and vertigo.

Placing a central line (CVC) in this case is justified for three reasons (Sect. 25.2.4): First, combined general/epidural anesthesia often requires constant administration of vasoactive substances, some of which may need to be administered centrally. Second, Mr. Anderson will probably receive IV fluids and perhaps parenteral nutrition for a few days after the operation. Finally, the CVC can help in determination of the patient's volume status. Other required monitors include a urinary catheter and an ECG monitor with ST segment analysis.

3.1.2 Which Lab Values Are You Interested in?

3.1.2.1 Electrolytes

Due to the nausea and vomiting (symptoms of bowel obstruction), electrolyte evaluation is necessary.

3.1.2.2 Hematocrit

The hematocrit gives clues to the intravascular hydration status – important information when a partial small bowel obstruction is in the differential diagnosis.

3.1.2.3 Coagulation Screen Including Platelet Count

Epidural catheter insertion is contraindicated in the presence of abnormal coagulation.

3.1.2.4 Blood Typing

In scheduled abdominal surgery procedures for which the extent of the surgery is unknown, one must anticipate extensive blood loss. In addition to the pre-surgical crossmatching, one should order at least two packs of RBCs to have in the OR.

3.1.2.5 Arterial Blood Gas Analysis

A preoperative arterial blood gas is not required; however, if performed, it may reveal a metabolic alkalosis if the vomiting were severe.

>> *Dr. Constantine explained to Mr. Anderson the risks of the TEA*, *the intra-arterial blood pressure monitoring*, *the CVC*, *and the transfusions. As he mentioned the rare risk of lower extremity paralysis with the TEA*, *Mr. Anderson*

asked "Do you really have to do that spinal catheter thing?" "No," replied Dr. Constantine, "however, patients have significantly less postoperative pain and get out of bed more quickly with the epidural, so I highly recommend it."

Mr. Anderson's electrolyte and coagulation values were within the normal range. His hematocrit was 53% (reference 37–47%), the slight increase indicating dehydration. Two packs of RBCs were already crossmatched. Dr. Constantine administered 1 mg midazolam IV to Mr. Anderson and informed the ward nurse that he could be taken immediately to the preoperative area. Dr. Constantine set off for the OR, pausing for a moment to gulp a cup of coffee and thinking to himself "You never know how long these operations can take; I might need this to stay awake."

In the lounge on the way to the OR, Dr. Constantine ran into Miriam, an anesthesiology technician. He told her about the planned surgery for Mr. Anderson. "Thanks a lot for the advance notice," complained Miriam. "I just cleaned and shut off everything in the last OR." With that, she stood up and left the lounge to set up for surgery. "That's my Miriam!" thought Dr. Constantine, "a little short with us docs, but always polite to the patients."

Dr. Constantine wanted to get a second cup of coffee, when the OR called to say it was ready. In the preoperative area, Miriam had Mr. Anderson hooked up to the monitor and had already sat him up. Dr. Constantine noticed increased blood pressure reading of 180/100 mmHg and administered 1 mg more of midazolam before beginning to place the epidural.

Dr. Constantine was already experienced in placing epidural catheters and successfully hit the eighth thoracic space on the first try. However, as the catheter over the needle entered the epidural space, it filled with blood. "It would have been too good to be true if it had actually worked," he thought.

3.1.3 How Would You Further Proceed with the Epidural?

If the catheter fills with blood when entering the epidural space, you have to assume that an epidural vein was perforated. First, pull the catheter out a bit, but never ever pull it out with the Tuohy needle in place, because this could cut the catheter and cause it to detach.

If, after pulling back a bit, blood can still be aspirated, the catheter must be completely removed. If, on the other hand, aspiration of blood is no longer possible, the catheter may remain in place. However, local anesthetics cannot be injected for 4 h. Highly concentrated local anesthetics which come into contact with a blood clot for a longer period of time inhibit the clot formation and cause fibrinolysis [4]. After about 4 h, the clot will have become thick enough that the local anesthetic exerts no anticoagulation effect.

>> Dr. Constantine called his attending, Dr. Eldridge. As instructed, Dr. Constantine pulled the catheter back, and aspiration of blood was no longer possible.

"Even so, it's annoying," said Dr. Eldridge, "but at least epidural anesthesia isn't absolutely necessary." Then Dr. Eldridge left again. After the dressing was securely in place, Dr. Constantine helped Mr. Anderson lie down. He moved Mr. Anderson to the OR. After hooking him to monitors, Dr. Eldridge returned and Mr. Anderson was induced using fentanyl 100 µg, lidocaine 60 mg, propofol 1.5 mg/kg, and rocuronium 1.2 mg/kg. Intubation was unremarkable. As usual on Sundays, everyone began to work simultaneously. The surgeons positioned Mr. Anderson and began to prep the site. Meanwhile, Drs. Constantine and Eldridge began to insert arterial and central venous lines. "This is the nice thing about call shifts," thought Dr. Constantine, "everyone wants to be done with their work as fast as possible."

As Dr. Constantine finished the lines and disposed of the last sharps, the door of the wash room opened and in walked chief resident Dr. Justice, with her hands slightly waving in the air. She was just about to give orders to the nurses when she spotted Dr. Constantine. Their eyes met, and both were relieved that they each wore face masks so no one noticed their fake smiles. They each thought of the time when Dr. Constantine was a first-year resident on her ward. They were fond of one another and had a

quick, but intense, love affair. Dr. Justice finally ended the fling in order to save her marriage. Other than her best friend, she never told anyone about it. "Never keep your honey where you earn your money" was her friend's advice.

Now Dr. Justice had come to understand; it was always a difficult situation when she and Dr. Constantine ran into one another. Anyway, Dr. Justice turned to the nurse to receive the sterile gown and gloves, and Dr. Constantine just pretended everything was normal.

Before the first skin incision, Dr. Constantine deepened anesthesia with 1 MAC desflurane without nitrous oxide. Even though Mr. Anderson also received another 150 µg of fentanyl IV, his blood pressure shot up to 172/89 mmHg at the first skin incision. Dr. Constantine gave another 50 µg of fentanyl and began to fill out his anesthetic record. The surgeons were making good progress, but Dr. Constantine began to wonder about the blood pressure of 170/100 mmHg. "Very annoying that I can't use the epidural," he thought. "Mr. Anderson seems to have a high tolerance for opioids."

3.1.4 How Do You Explain This?

3.1.4.1 Level of Hypnosis

The hypnotic depth may not have been sufficient for Mr. Anderson. MAC values are only an estimation of the required dose; MAC is not an absolute value.

The reduction or loss of consciousness can sometimes require significantly higher MAC values. One must keep in mind that elimination of the sense of hearing is nearly impossible (Sect. 8.1.10).

3.1.4.2 Depth of Analgesia

Just as with hypnosis, the required amount of analgesia also varies from person to person.

3.1.4.3 Hypertension

Poorly controlled and treated hypertension can become evident during anesthesia. However, usually these patients have low BPs due to the medication-induced sympathetic blockade.

3.1.4.4 Errors of Measurement

As a general rule, inaccurate measurement must be considered when a blood pressure is unusually high or low. In noninvasive blood pressure, the proper positioning and the proper size of the cuff must be checked. With invasive pressure measurement, the positioning of the electronic pressure transducer and the correct calibration must be checked. Also, over- or under-damping must be excluded.

3.1.4.5 Absence of an Effect of the Medication

One must always carefully consider the absence of a medication's effect when the medication being used has been diluted for better administration. Mathematical errors occur in 2–5 % of these solutions, and it has been proven that the physician error rate is much higher than the nurse error rate [10].

>> Dr. Constantine checked the arterial pressure transducer. "Looks OK," he thought. Just to be on the safe side, he recalibrated it, but the blood pressure still stayed significantly above normal. The door to the OR opened, and attending anesthesiologist Dr. Eldridge walked in. "As I see it, you are having trouble controlling the blood pressure without the use of an epidural," he said to Dr. Constantine. "What about a little more hypnosis and analgesia?"

Despite the face masks, the eyes of the surgeons clearly showed grins of amusement.

"Thanks a lot, boss," Dr. Constantine thought to himself, "So nice of you to walk in here and make me look like an idiot in front of everybody else. Why don't you just go back to surfing the Internet – I was doing fine without you." Only Dr. Justice gave him a consoling glance, and this time he was thankful that she was in the OR.

Dr. Eldridge didn't stay long in the OR. Dr. Constantine gave Mr. Anderson 100 µg fentanyl IV and turned up the desflurane vaporizer until the end-expiratory MAC was 2. Shortly thereafter, the systolic blood pressure was below 160 mmHg – however, within 10 min it began to rise again. Dr. Constantine was becoming quite perplexed; he was actually certain that the anesthesia was deep enough, so he decided to administer labetalol IV.

3.1.5 Why Is Labetalol Well Suited for Treatment of Intraoperative Hypertension?

Labetalol is a mixed α/β-adrenergic antagonist, commonly used to treat intraoperative hypertension [8]. Labetalol competitively blocks β_1- and β_2-receptors as well as α_1-receptors within vascular smooth muscle. Labetalol causes a decrease in systemic vascular resistance without a significant reduction in resting heart rate, cardiac output, or stroke volume. It is commonly used in the acute treatment of hypertension in pregnant patients during obstetric anesthesia.

>> *After administering 50 mg of labetalol IV in divided doses, Mr. Anderson's systolic blood pressure only briefly dropped below 160 mmHg. To be safe, Dr. Constantine gave Mr. Anderson another 100 μg of fentanyl IV. "That has really got to be enough now," he thought as he began to review all possibilities again. Perhaps he had overlooked something.*

As he concentrated, he remembered that Mr. Anderson had had some premature ventricular contractions (PVCs) on the ECG monitor. "Something is wrong here," he thought, and he decided to call his attending, Dr. Eldridge. However, his inner pride made him pause a moment before reluctantly dialing. "Yeah, anesthesia without epidurals can be challenging," announced Dr. Eldridge as he walked in. "Are you at least coming along alright?" he asked the surgeons. The chief resident surgeon Dr. Justice replied "Yes, the patient seems to be doing well on our side, very relaxed; no problems with the tumor excision; I think we might just need about another 90 min."

Dr. Constantine clearly noticed that Dr. Eldridge would have preferred a different type of response. He started studying the anesthesia record and finally looked up at the monitor. The blood pressure was 240/130 mmHg, the heart rate was 140/min, and two to three premature ventricular contractions across each screen were visible on the monitor. ST segments in the 5-lead ECG suggested myocardial ischemia in three leads. S_pO_2 was 92 %. Dr. Eldridge wrinkled his forehead and took a stethoscope to auscultate the chest. He heard bilateral crackles.

3.1.6 What Is Going on?

The symptoms Mr. Anderson displays include:

- Severe hypertension
- Premature ventricular contractions (PVC)
- ST segmental changes
- Beginning pulmonary edema

Clinically, the diagnosis is a hypertensive emergency with impending left ventricular failure. Signs of increased intracranial pressure (somnolence, paralysis, seizures) are not identifiable in anesthetized patients. A hypertensive crisis is defined by the acute increase in blood pressure to a systolic >220 mmHg or a diastolic >120 mmHg [3]. The deciding factor is not the absolute value, but the extent of the acute increase [12]. The risk of an MI during an acute hypertensive episode is about 4 %. The probable pathophysiological mechanism is an acute increase of the peripheral resistance.

3.1.7 What Is the First Goal of Therapy?

The primary therapeutic goal for a hypertensive crisis with pulmonary edema is to relieve stress on the heart by lowering the cardiac preload and afterload. Simultaneously, oxygenation must be increased.

3.1.7.1 Lowering Preload and Afterload

It is usually not possible to raise the head and chest of anesthetized patients during surgery. Nitroglycerin is predominantly a venous vasodilator, which works quickly, has a short duration of action, and is easily controlled [8]. The combination with a loop diuretic is useful in certain circumstances.

3.1.7.2 Improving the Oxygenation

In order to improve oxygenation, inspiratory oxygen concentration and the level of end-expiratory pressure (PEEP) should be

increased. Increasing PEEP also decreases in preload and afterload.

>> *Attending anesthesiologist Dr. Eldridge increased the inspiratory oxygen concentration to 100 % and the PEEP to 10 mmHg*. "*Please give me some nitroglycerin spray*," *he said to the anesthesiology technician Miriam. Two puffs of nitroglycerin were administered sublingually. The nitroglycerin brought a little relaxation*, *but no significant success*. "*Miriam*, *please go to the pharmacy to get a nitroglycerin infusion*." *Dr. Eldridge was sure he had chosen the best therapy*. "*And Dr. Constantine*, *could you please prepare to inject the epidural catheter with local anesthetic*? *Never mind that only 3 h have gone by – I've got everything under control*."

The chief resident surgeon noticed the hectic activity at the patient's head and wondered why the epidural catheter hadn't been used for surgery. She grinned under her face mask and couldn't help but say, "*Are you going to show your assistant how to use an epidural for anesthesia*?" *It was quite obvious that this comment annoyed Dr. Eldridge*, *but before he could respond*, *she continued*, "*The tumor is almost completely out. We will hurry up with the sutures so you can care for Mr. Anderson in peace*."

After injecting the epidural with 10 ml of bupivacaine 0.25 % and with the continuous infusion of nitroglycerin, *the systolic blood pressure decreased to 190 mmHg. The heart rate was 100 beats/min and the* S_pO_2 *had improved to 96 %. Even so*, *Dr. Eldridge was not satisfied*, *because the ST segment changes were still present*.

3.1.8 Which Drug Could Also Be Used Now?

The vasodilator with the highest efficacy is sodium nitroprusside (SNP) [8]. SNP is an arterial and venous vasodilator. SNP reacts with oxyhemoglobin to produce methemoglobin, cyanide, and nitric oxide (NO). NO activates guanylate cyclase of smooth vessel walls. The intracellular cGMP level increases and leads to inhibition of the inflow of calcium into the cell and increases calcium uptake in the endoplasmic reticulum. The result is vasodilation.

Production of NO after nitroglycerin administration appears to be the result of mitochondrial acetaldehyde dehydrogenase (mADH) [2]. mADH is inhibited by many drugs, such as sulfonylurea, urea, and metabolism by-product like nitrates, and is subject to significant gene polymorphism. The negative feedback from nitrates, which is produced in the metabolism of nitroglycerin, explains the reduced efficacy of nitroglycerin over time. As opposed to nitroglycerin, SNP is a direct NO donor, and tolerance does not develop with long-term use.

Due to the decrease in systemic vascular resistance, SNP is well suited for the therapy of acute heart failure. The myocardial oxygen consumption is dropped by the decrease in systolic and diastolic pressures. Provided hypovolemia isn't present, the cardiac output remains stable, or slightly increased due to the reduction of afterload, with a simultaneous increase in left ventricular preload obtained by administration of additional volume [1].

SNP must be protected from sunlight and is diluted in a 5 % glucose solution. BP control is easy because of the short duration of action of SNP. It is therefore often chosen for intraoperative blood pressure control when the blood pressure is severe or with need for moment-to-moment titration of blood pressure, as in cardiac and vascular surgery. The usual infusion rate is 2–10 μg/kg/min. In order to avoid cyanide toxicity (cyanide is produced by the breakdown of Na nitroprusside and depletion of the body's neutralizing sulfur donators), the SNP must be given with a sodium thiosulfate solution.

As with other vasodilators, SNP leads to increases of the pulmonary right–left shunt and $\dot{V}/\dot{Q}$ mismatch, with consecutive reduction of the arterial oxygen partial pressure and oxygen saturation.

A new short-acting calcium channel blocker, clevidipine, is also useful in the treatment of severe hypertension, such as in the perioperative management of pheochromocytoma [6].

>> *Dr. Eldridge*, *the attending anesthesiologist*, *asked assistant Miriam to obtain an SNP infusion from the pharmacy. In addition*, *he wanted*

esmolol and amiodarone in case the frequent PVCs didn't go away after lowering the arterial pressure. Just as Miriam came back with the drugs, Mr. Anderson's systolic pressure dropped below 100 mmHg. Dr. Eldridge stood up and glanced at the surgical field to see if there was major blood loss. The chief surgeon Dr. Justice glanced at him and said "We have the tumor out – it was not a pancreatic, but an adrenal tumor. Just a little longer to get the bleeding stopped, and then we will be done." Suddenly the cause of the problems for Dr. Eldridge and Dr. Constantine was crystal clear.

3.1.9 You Would Have Probably Recognized the Problem Immediately, Right?

Mr. Anderson reported his new hypertension diagnosis during his preanesthetic evaluation. Further symptoms included intermittent headache and nausea. The nausea was what convinced him to go to the hospital. Persistent hypertension during surgery and the location of the tumor suggest an adrenal neuroendocrine tumor. The histology results later did indeed confirm the diagnosis of a pheochromocytoma.

A pheochromocytoma is a catecholamine-producing tumor originating in the neuroectoderm, from the chromaffin cells of the sympathetic system [5]. Ninety percent of the tumors are benign and unilateral; however, sometimes they appear solitary or in groups along the sympathetic trunk and ganglia of the abdomen and pelvis. Sometimes the tumors are found in the thorax (2 %) and in the neck area (<0.1 %). In 10 % of these cases, there is a family predisposition. There is also an association with thyroid and parathyroid tumors (e.g., MEN syndrome) and with phakomatosis (e.g., neurofibromatosis, tuberous sclerosis).

Which catecholamine the tumors produce is dependent on the location and the type of tumor. If they originate from the adrenal cortex, they often secrete epinephrine and norepinephrine, but sometimes only epinephrine. If they arise from a sympathetic paraganglion, they often produce only norepinephrine. In malignancy, dopamine secretion can also be predominating.

3.1.10 Why Did the Arterial Blood Pressure Drastically Fall at the End?

A sudden reversal of blood pressure problems is typical for this stage of the operation. The catecholamine release due to manipulation by the surgeon ceased abruptly.

Usually the pheochromocytoma diagnosis is known before the surgery is begun. During surgical removal, the vein of the tumor is ligated before the tumor is manipulated. This phase requires good communication with the surgeon, so that the anesthesiologist can prepare for the new circulatory situation in advance.

By stopping the antihypertensive and epidural drugs, by administering volume and beginning catecholamine therapy early, a threatening hypotension can be avoided. The short half-life of the catecholamines is responsible for the blood pressure drop. Preoperatively, typically a downregulation of the α-receptors is seen due to increased catecholamine levels. Hypovolemia and intraoperative blood loss may further complicate the treatment of intraoperative hypotension.

>> *The surgical team relaxed a bit after the excised tissue was sent to the lab. The anesthesiology team, however, became more stressed with every successive decrease in Mr. Anderson's blood pressure. The systolic blood pressure barely held at 60 mmHg with large doses of phenylephrine and vasopressin. Finally, the norepinephrine infusion was ready to be hooked up, and with high doses of catecholamines and extra fluids, the patient finally stabilized. Dr. Constantine brought Mr. Anderson intubated and ventilated to the intensive care unit. On the way there, his phone rang. "That's probably Dr. Eldridge with the next assignment for me," he thought, "but I have to take a break first!" He was mistaken: it was the chief resident surgeon Dr. Justice. "Don't be too annoyed with your boss," she said. "Unfortunately, we often have bosses who are bad examples. I think you did a good job today." They wished each other a quiet on call and said goodbye.*

So at least there was a little happy ending to this story. Dr. Constantine was able to speak

freely again with Dr. Justice. Always trying to avoid her had been extremely stressful.

3.2 Case Analysis/Debriefing

3.2.1 Define Blood Pressure!

Physically, it is the potential difference, according to Ohm's law, defined as the product of the resistance and the current:

$$U = R \cdot I$$

In circulatory system terms, the blood pressure = systemic vascular resistance × cardiac output.

Many factors can influence vascular resistance and blood flow, such as vessel diameter and length, viscosity, elasticity, flow velocity, friction, contractility, etc. These examples explain why hypertension is often so hard to treat.

Why is it important to remember this definition? In our daily clinical routine, we often use blood pressure as a surrogate for blood flow. We shouldn't forget that there are many other complex factors involved. For example, an increase in blood pressure following vasopressor administration increases vascular resistance, but does not necessarily increase flow to the target organ.

3.2.2 Define Arterial Hypertension!

Adult arterial hypertension is defined as a systolic blood pressure above 140 mmHg or a diastolic blood pressure above 90 mmHg, measured on repeated days at different times. The cause is irrelevant.

3.2.3 How Many Forms of Arterial Hypertension Can You Name?

In 90 % of patients, no clear cause is found. For this so-called primary hypertension, there is no underlying medical cause. Secondary hypertension occurs in the remaining 10 % of patients. Those have a treatable underlying disease or condition. Table 3.1 shows an overview of the different causes of secondary hypertension.

Table 3.1 Sample causes of secondary hypertension

Causes	Conditions
Renal	Parenchymal (chronic glomerulo-/pyelonephritis)
	Renal vascular (e.g., renal artery stenosis, aneurysm) and general diseases with renal involvement (e.g., diabetes mellitus, gout, lupus)
Endocrine	Conn syndrome
	Cushing syndrome
	Pheochromocytoma
	Hyperthyroidism
	Acromegaly
Cardiovascular	Aortic stenosis
Neurogenic	Increased intracranial pressure
	Encephalitis/meningitis
	Tumors
	Porphyria
Pregnancy associated	Pregnancy-induced hypertension
	Preeclampsia, eclampsia
Medications	Corticosteroids
	Oral contraceptives
	Nonsteroidal anti-inflammatory drugs
	MAO inhibitor
	Cyclosporin A
Blood diseases	Polycythemia vera

3.2.4 How Are Patients Prepared for Elective Pheochromocytoma Surgery?

Massive catecholamine release due to surgical tumor manipulation can lead to a life- threatening hypertensive crisis and cardiac arrhythmias. Therefore, patients are premedicated with α-blockers and sometimes also with β-blockers [11]. Phenoxybenzamine, a nonselective α-antagonist, treats the catecholamine-induced vasoconstriction, normalizes blood pressure, and improves cardiac output.

Preparation is begun 14 days before the scheduled operation with 10–20 mg phenoxybenzamine PO and increased by 10–20 mg/day until the blood pressure is stabilized. The target pressure is 160/90 mmHg. The patients often

complain of orthostatic vertigo. This is a result of hypovolemia, which must be compensated for by drinking fluids until the hemoglobin drops. Sometimes undesired tachycardia occurs as a side effect of phenoxybenzamine, in which case a shorter working selective α_1-antagonist such as doxazosin is used, or a β-blocker is added.

Importantly with pheochromocytomas, β-blockade should not be started before the α-blockade has taken effect. Otherwise, removal of the β_2 vasodilator effects can lead to severe hypertension with left heart failure. When urgent surgery is required, phenoxybenzamine can be given IV in the ICU under careful monitoring. The sensation of a stuffed nose is a good indication for a functioning α-blockade.

Patients need good anxiolytics and should receive benzodiazepines the night before and the morning of the operation, considering the general contraindications, of course.

Note:

Since the operation is usually scheduled electively, unblocked patients should not be in the OR!

3.2.5 Which Medical Errors Do You See in the Presented Case?

3.2.5.1 Epidural Utilization

In Sect. 3.1.3 it is already described that utilization of an epidural after a bloody insertion has to be viewed critically. Whether such utilization is actually malpractice is unclear. Nevertheless, utilization of the epidural with a local anesthetic immediately before removal of a neuroendocrine tumor is medically questionable.

3.2.5.2 CVP Measurement

Continuous CVP measurements were not performed.

3.2.5.3 Blood Pressure Monitoring on the Ward/Preoperative Diagnostics

While checking the chart on the ward, Dr. Constantine couldn't find a blood pressure measurement for Mr. Anderson. Furthermore, one may assume that the high-quality radiographic images should have localized the tumor at the adrenal gland. However, further investigation into the localization of the tumor was not carried out. The initial diagnosis of an ileus was never questioned.

3.2.5.4 Monitoring of Laboratory Results

There was no intraoperative measurement of lab values. When the PVCs occurred, hematocrit and electrolytes should have been checked.

3.2.6 Which Systems Failures Can Be Found in the Presented Case?

3.2.6.1 Informed Consent from the Patient

Informed consent requires a discussion between the physician and the patient concerning the risks, benefits, and alternatives to treatments. It is an ethical obligation of the practice of medicine and a legal requirement in the U.S. in all 50 states [7]. The patient must have "competence" and "capacity" to provide informed consent. Competence refers to the patient's legal authority to make decisions. Adult patients, generally patients who are 18 or older, are presumed legally competent unless otherwise determined by a court. Capacity refers to a determination made by medical professionals that a patient has the ability to make a specific decision at a specific time. The informed consent discussion should focus on the indications for the proposed treatment, description of the procedure in terms a layperson can understand, and an explanation of viable alternatives. Disclosure of material risks of the recommended and alternate treatments is required. Material risks are risks that occurred frequently (e.g., backache, spinal headache, or failure after an epidural) as well as those that are rare but may result in serious morbidity or mortality (e.g., nerve injury/paralysis, seizures, coma, death). Some states also require disclosure of all persons anticipated to be involved in the patient's anesthetic care. Some

hospitals and some states require a separate written anesthesia consent form. In other places, a written surgical consent form suffices, with a verbal informed consent discussion about anesthesia risks. Specific risks described in the discussion, such as persistent numbness or paralysis from the epidural, should be charted on the preoperative evaluation or anesthetic record.

Mr. Anderson had been on the ward for 2 days. The anesthesiologist could have and should have been called earlier for a preoperative evaluation. This system mishap is therefore the fault of the surgical team. From an anesthesiology point of view, Dr. Constantine might not have obtained full consent for the epidural. The patient was not sure if he wanted to take the risk of paralysis; he had no time to think it over.

3.2.6.2 Preoperative Diagnosis

Even though it may seem at first to be a medical mistake, there is probably a systems failure to blame. Due to a shortage of time or manpower, Mr. Anderson's case was not carefully evaluated. Furthermore, due to the hypertension diagnosis, blood pressure should have been noted in the chart. There seems to have been no order issued to the nurses for such monitoring.

3.2.6.3 Communication Between Surgeons and Anesthesiologists

Even though this problem is discussed under nontechnical aspects, (Sect. 3.2.7), it is actually a systems failure. A pre-surgical checklist should have been performed, with discussion of possible differential diagnosis, potential surgical issues, and anesthetic concerns. During the procedure, the surgeons realized that the anesthesiologists were having a hard time. In such situations, an automatic knowledge exchange (review of facts, problems, and plans) would be most helpful. Ongoing intraoperative communication between the surgical and anesthetic teams could have led to earlier diagnosis and treatment of the pheochromocytoma.

3.2.7 What Psychological Technique Could Have Been Used by Dr. Constantine and Dr. Eldridge to Get to the Correct Diagnosis Earlier?

For Dr. Constantine and Dr. Eldridge, the diagnosis of the hypertension was simple: the anesthesia was not deep enough. This "simple model of the world" was not further explored. Dr. Constantine further expanded his "simple model" by viewing hypertension as a stand-alone disease which he treated with labetalol. The somewhat more "complex model of the world" was accepted by the supervising Dr. Eldridge, who was called in, and gave nitroglycerin. The actual cause of the hypertension, a catecholamine-producing tumor, was not considered until the blood pressure dropped after removal of the tumor.

The sources of error are the hurried analysis and tendency to accept oversimplified explanations. This error can be minimized by carefully considering all the possible causes of the disorder. For example, the decision-making model FORDEC, borrowed from the airline industry, can help in urgent decision-making. In this acronym every letter stands for a step in the decision-making process:

1. **F**: Facts. Collection of all the relevant facts
2. **O**: Options. Collection of alternative actions
3. **R**: Risks. Considering F and O, the chances of success for each action are considered.
4. **D**: Decision. The action most likely to succeed is chosen, with possible backup plans.
5. **E**: Execute. The chosen action is carried out.
6. **C**: Check. Compare the action and success with the expected result.

The **FORDEC** model works well as a mental checklist in time-sensitive situations.

It minimized ignoring and overlooking important decision-making steps. In the presented case, critical evaluation of the known preoperative facts would most certainly have led to an earlier correct diagnosis.

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Case 4: A Day in the PACU

4

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4.1 Case Introduction

>> Dr. Finn had worked a year and a half as an anesthesia resident. He had already performed anesthesia for major abdominal surgery, and with some supervision, he had handled significant intraoperative bleeding, including in ASA 3 patients. He was proud of himself because after 9 months of ENT and 5 months of ophthalmology, he could finally do "real anesthesia."

Due to staffing shortages during a summer vacation, Dr. Finn was assigned to the postanesthesia care unit (PACU). He hadn't yet completed this rotation, but the OR schedule was light, and there weren't any interesting cases for him.

Anesthesiologists have different opinions about the PACU. Some enjoy the fast pace and the constant surprises of the PACU; Dr. Finn was looking forward to the new challenge. Bridgette,

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a trusted and experienced PACU nurse, was the nursing supervisor and oriented him to the PACU.

Right from the beginning, Dr. Finn encountered unexpected tasks; he received requests from the ward for a placement of a central venous catheter (CVC) in a patient with poor vascular access and an epidural for a post-op patient who was in severe pain. Since the PACU was still empty, he decided to send for the patients from the ward. When the PACU charge nurse Bridgette heard of his plans she balked, "Oh no, that's not the way we do it. The total knee replacement patient will be coming out of the OR any minute. The additional procedures must be done in the afternoon, when it is quieter here." She promptly canceled the procedures and told the wards that they would have to wait. Actually, Dr. Finn didn't think it would be too difficult to care for a man's knee and to place a CVC and an epidural, but he kept his mouth shut so as not to make waves with the charge nurse.

The man's total knee replacement was performed under general anesthesia. The patient's anesthesiologist reported that the anesthesia had been unremarkable and the man would be quickly discharged to the ward. Dr. Finn was annoyed that he had given into the charge nurse's wishes, because he was sure that he could have already had the epidural in. Since he didn't have anything to do at the moment, he picked up a book and began to read "Complications and Mishaps in Anesthesia."

4.1.1 What Is the Purpose of a PACU?

The PACU is a place of temporary monitoring following an anesthetic [2]. The patients remain here until they recover, fully regain protective airway reflexes, and until no further circulatory/respiratory complications are likely. Usually, PACU stay is limited to less than a few hours. The PACU should deliver the immediate postoperative care for all patients, except care for ICU patients who will be mechanically ventilated overnight to several days (e.g., major trauma, liver transplantation, cardiac surgery patients). These patients go straight to the ICU. Perianesthesia nurses are trained to deal with acute postanesthetic problems, before transfer to the ward [4]. In addition, anesthesiology personnel are readily available to treat an emergency. The PACU care can be time-consuming and challenging. Proper PACU care can prevent the necessity of an expensive ICU stay; likewise, patients who require ICU treatment can be identified by the PACU team, which assesses criteria for transfer.

In addition, the PACU can be used as a safe monitoring location for the insertion of invasive lines, short procedures such as a cardioversion, electroconvulsant shock therapy, or relocation of a dislocated hip. Often, the PACU is used to place regional anesthesia catheters for the treatment of acute or chronic pain.

4.1.2 What Equipment and Staffing Qualifications Are Required in a PACU?

The PACU should be close to the OR. The necessity of quick entry for anesthesiologists and OR personnel requires close proximity to the OR, and, in case of an emergency, a direct and speedy route to the OR is essential.

Constant nursing presence is required in the PACU, with at least two nurses on duty. Table 4.1 shows an overview of the PACU equipment resuscitation equipment [2, 4].

Table 4.1 PACU equipment

ECG monitor	Required
Pulse oximetry	Required
12-lead ECG	Recommended
Noninvasive blood pressure	Required
Invasive blood pressure	Recommended
Oxygen	Required
Temperature measurement	Required
Suction	Required
Defibrillator	Required
Crash cart with medications/supplies	Required
Ambu bag and emergency airway equipment	Required
Ventilator	Recommended
Infusion pumps	Recommended
Emergency laboratory (nearby)	Recommended
Capnography	Recommended
Phones	Required

Each patient's space must be equipped with an ECG, pulse oximetry, temperature probe, and a noninvasive blood pressure cuff. In addition, oxygen and suction devices are required. More difficult cases require invasive blood pressure, CVP monitoring, and a 12-lead ECG. In case of emergency, a defibrillator, crash cart, Ambu bag, and airway equipment must be available. In addition, testing of lab values such as arterial blood gases, hemoglobin concentration, glucose, and serum electrolytes should be available nearby.

4.1.3 What Are the Most Commonly Encountered Postoperative Problems?

- Postoperative nausea and vomiting (PONV)
- Pain
- Cardiac and circulatory problems such as hypotension or arrhythmias
- Hypothermia (shivering)
- Prolonged action of anesthetic agents or medications, including opioids
- Postoperative residual neuromuscular blockade
- Hypoxemia and respiratory insufficiency
- Hemorrhage and coagulation disorders

4.1.4 Which Criteria Must Be Fulfilled for Transfer Out of the PACU?

The American Society of Anesthesiologists (ASA) Task Force on Postanesthetic Care defines the following guidelines for discharge [3]:

- Patients should be alert and oriented. Patients whose mental status was initially abnormal should have returned to their baseline.
- Vital signs should be stable and within acceptable limits. Discharge should occur after patients have met specified criteria. The Aldrete scoring system is used as a documentation of fitness for discharge. The system assesses activity, respiration, circulation, pulse rate, consciousness, and oxygen saturation.
- Outpatients should be discharged to phase 2 PACU, where they demonstrate ability to tolerate fluids, ambulate, and void. Afterward, they will be discharged to a responsible adult who will accompany them home and be able to report any postoperative complications.
- Outpatients should be provided with written instructions regarding postoperative diet, medications, activities, and a phone number to be called in case of emergency.

>> "Interesting book, but will it help my clinical practice?" thought Dr. Finn, as he put it aside to care for the newly arriving patients from the OR. One patient had a total knee replacement, another had laparoscopic cholecystectomy, and the third had a vaginal hysterectomy; all three procedures were unremarkable and carried out under general anesthesia. Dr. Finn began caring for the patients, treating problems such as pain, nausea, and shivering. In the meantime, the wards were calling again, so Dr. Finn gave in and accepted the patient needing the epidural and planned the patient requiring the CVC for half an hour later. Charge nurse Bridgette was very annoyed with this plan and left to take a coffee break. This left only the remaining PACU nurse and Dr. Finn to take care of all of the PACU patients.

While Bridgette was gone, two new patients arrived in the PACU, one patient who had a varicose vein surgery and one patient with a femur fracture repaired using an intramedullary rod. As Dr. Finn hooked them up to monitors, the patient S/P cholecystectomy requested help due to persistent nausea. Dr. Finn gave ondansetron 4 mg IV as the patient needing the epidural placement arrived.

Now the PACU had become immensely stressful; the nauseated patient received no relief from the ondansetron and began to vomit, and the patient S/P hysterectomy was still complaining of pain after 10 mg of morphine IV. Unfortunately, coagulation study results were still pending and needed to be done before the epidural could be performed.

The only thing that was going as it should at that moment was that the total knee replacement patient was ready for discharge. Dr. Finn first wanted to check the epidural patient's coagulation results and care for the patient with pain after the hysterectomy.

The hysterectomy patient was 68 years old, ASA 2, and 85 kg. Despite receiving metoprolol preoperatively for hypertension, her heart rate was 110/min. He again administered 5 mg morphine IV and promised her it would be better soon.

4.1.5 What Do You Know About PONV and What Affects PONV?

PONV is a common problem in the PACU, but can also occur many hours later, after discharge from the PACU. Patients are not only miserable, but PONV also affects the healing process, for example, by slowing the start of oral intake. Also, many patients use PONV to rate the quality of anesthesia; therefore, the importance of PONV is significant to anesthesiologists.

Many factors influence the occurrence of PONV, although some factors are controversial [1]:

Patient factors:

- Female, nonsmoker, history of motion sickness, young, ASA 1–2

Influence of anesthesia:

- Postoperative opioid administration, anesthesia duration, use of nitrous oxide or inhalation anesthetics, reversal of neuromuscular blockade with neostigmine

Influence of the surgical procedure:

- Strabismus surgery, ear surgery, laparoscopic surgery, upper abdominal procedures

In cases of prolonged PONV, a gastrointestinal mobility disorder must be considered. There is a large selection of medications available for therapy and prophylaxis of PONV, many of which can be administered synergistically. For example:

- Steroids (dexamethasone)
- 5-HT_3 antagonists (granisetron, dolasetron, tropisetron, ondansetron)
- Antihistamines (diphenhydramine)
- Butyrophenone (droperidol)
- Anticholinergics (scopolamine)
- Anesthetics (propofol)

Apart from pharmacological therapy and prophylaxis for PONV, acupuncture at the standard P6 (also known as Neiguan) point, located on the ventral side of the wrist, is also effective [6]. All therapeutic choices share the same NNT (number needed to treat) between five and ten. This means that in a NNT of 5, one out of five patients will benefit from the therapy. The second bit of good news is when a method fails, there is no information given about the effectiveness of other alternatives. The take-home message is that a combination of various medications or methods is useful and often clinically successful.

With PONV, it is important that patients with increased risk are identified preoperatively and prophylactic measures are begun before or during induction of anesthesia. The identification is made easier by an algorithm specific for your hospital. Figure 4.1 shows an example of a PONV algorithm. The algorithm is based on North American [5] guidelines. In addition, outpatient surgery was included as a risk factor, in order to reduce the occurrence of delayed discharge due to PONV.

>> The gallbladder patient vomited again. Dr. Finn didn't get an emesis basin to her quickly enough; the bedding now needed to be changed as soon as possible by the remaining PACU nurse. He wondered a little about charge nurse Bridgette; she seemed to be taking a very long time at her break. The telephone rang with the coagulation results. They were normal, so he began to obtain informed consent from the epidural patient.

As charge nurse Bridgette walked in and realized what was going on, she gave Dr. Finn a scolding look. She returned to the hysterectomy patient, noting that she was still in pain, so nurse Bridgette administered 5 mg morphine IV, and due to tachycardia, she increased the rate of the Lactated Ringer's solution. After that, she called the ward and requested pickup of the patient who had recovered from his arthroscopy, and she began to assist Dr. Finn in the epidural catheter placement. The monitor of the hysterectomy patient went off, due to an oxygen saturation of 89%. The patient appeared agitated. Dr. Finn was in sterile drapes, so charge nurse Bridgette turned up the hysterectomy patient's oxygen from 1 to 4 l/min. Thereafter, the saturation quickly rose to 95%.

4.1.6 Why Does It Make Sense to Increase the Oxygen Flow?

This patient received general anesthesia with muscle relaxation and mechanical ventilation. With general anesthesia, worse in the presence of mechanical ventilation, atelectasis occurs in the

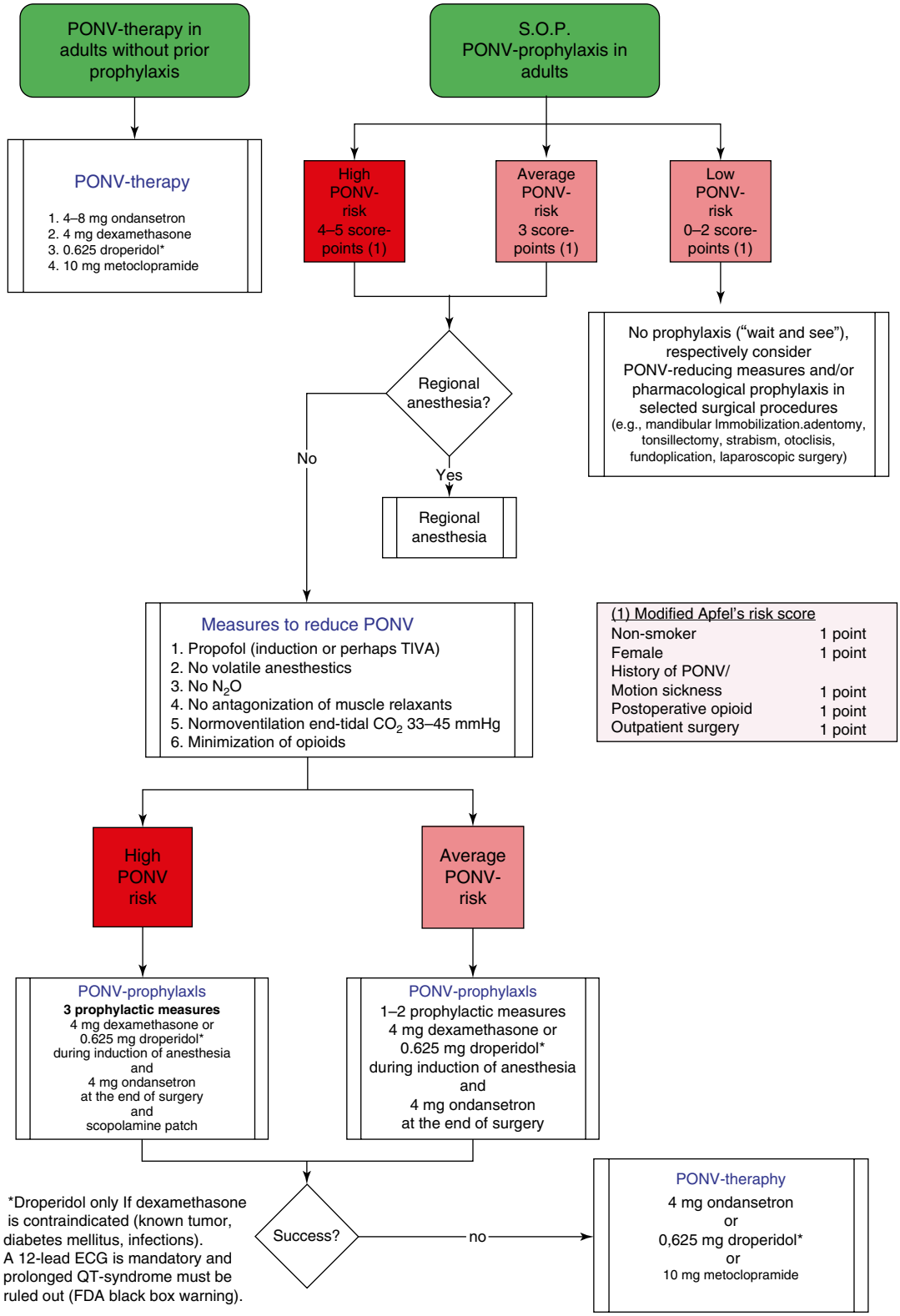

Fig. 4.1 Example of an algorithm for "postoperative nausea and vomiting (PONV) in adults"

dorsal caudal areas of the lungs. The measurable results are:

- Reduction in total lung capacity, residual volume, and functional residual capacity
- An increase in the closing capacity
- An increase in dead space ventilation
- An increase in intrapulmonary shunt

These changes are still in effect after general anesthesia has worn off and can last for an unpredictable period of time. Because of the ventilation–perfusion ($\dot{V}/\dot{Q}$) mismatch with increased dead space ventilation and increased intrapulmonary right to left shunt, PACU patients often have hypoxemia or hypercapnia. Other factors, such as reduced ventilation due to pain upon breathing or the respiratory depression caused by medications, can intensify the problem.

Oxygen is dissolved in blood plasma and chemically bound to hemoglobin. The amount of each form transported is dependent on the partial pressure of oxygen (P_aO_2). The oxygen saturation of blood (S_pO_2) shows the percent of hemoglobin which is saturated with oxygen. The relationship of S_pO_2 and P_aO_2 is demonstrated by the O_2 binding curve. An S_pO_2 value of 90 % correlates to a P_aO_2 value of 60 mmHg.

The P_aO_2 is directly proportional to the alveolar oxygen partial pressure (P_AO_2), so that an increase in the alveolar oxygen concentration is sufficient to increase the P_aO_2 (S_aO_2). One must note that the oxygen transport capacity of hemoglobin is many times higher than the physical solubility of oxygen in blood. In anemia, for example, despite good S_aO_2 values, the available oxygen can be low enough to result in anaerobic metabolism.

>> Just as Dr. Finn was finishing with the epidural placement, the patient needing the CVC insertion was brought in, and the nurses from the ward arrived to pick up the patient who had the total knee replacement. Charge nurse Bridgette wanted to know if the patient who had the cholecystectomy could be discharged to the ward, because her nausea had finally improved.

Dr. Finn came up with the following plan: first, discharge the patient who had recovered from the total knee replacement, then obtain informed consent for the CVC insertion, then see how the patient who had the hysterectomy was doing. The patient S/P cholecystectomy could wait, and nurse Bridgette and the other PACU nurse could look after all the others. Fifteen minutes later, as Dr. Finn approached the bedside of the hysterectomy patient, he noticed a heart rate of 130 beats/min and a blood pressure of 105/60 mmHg. The patient admitted being a little dizzy, tired, and short of breath. Dr. Finn knew something was not right, but what was the problem?

4.1.7 What Should Dr. Finn Do Now?

The symptoms are too vague to allow a definite course of action. The dizziness, exhaustion, and the dyspnea could all be caused by the morphine administration. However, the tachycardia in combination with the hypotension was worrisome. Dr. Finn must first obtain further information to differentiate between possible causes:

- Cardiac
- Pulmonary
- Pain
- PONV
- Hypovolemia or anemia
- Metabolic (e.g., hypoglycemia)
- Allergy
- Sepsis

First of all, get a more detailed history from the patient, and do a quick physical check. With the help of the anesthesia record, one can tally the intraoperative blood loss and check to see if similar episodes occurred during surgery. Also essential is an arterial blood gas analysis, with hemoglobin and hematocrit, and examination of the ECG for rhythm and ST segments.

>> Dr. Finn got a quick overview of the symptoms; the patient described the rapid heart rate as uncomfortable, and she had the feeling that she couldn't really breathe. Her chest had a squeezing feeling she said, and she had dull retrosternal pain. Dr. Finn's mouth went dry. He too felt a dull retrosternal pain and began to experience tachycardia himself.

4.1.8 What Is Your Suspected Diagnosis, and What Is the Most Important Therapeutic Step to Be Taken Now?

The symptoms presented by the patient could be due to myocardial ischemia or myocardial infarction. Unstable angina is potentially lethal and requires immediate therapy, even when the diagnosis is unconfirmed. The goals are to:

- Increase the coronary oxygen supply
- Decrease the myocardial oxygen consumption

In order to raise the available oxygen in the myocardium, the inspired oxygen concentration must be increased. If the blood pressure is high enough to tolerate it (systolic pressure >100 mmHg), nitroglycerin can be administered. Nitroglycerin can be repeated every 10 min, provided hypotension doesn't occur. Treating tachycardia also increases coronary oxygen supply.

Reduction of myocardial oxygen consumption is also accomplished by reducing heart rate. The simultaneous prolongation of diastole additionally increases coronary blood flow. Heart rate can be reduced by analgesics, sedatives, β-blockers, volume administration, or via α-mimetic substances. In the presented case – with the logistical possibilities of the PACU – further diagnostic tests should be carried out, before β-blockade is performed. Morphine reduces preload and can also decrease myocardial oxygen demand.

>> *Dr. Finn increased the oxygen flow to 8 l/min and gave the patient 2 puffs of nitroglycerin sublingually. Her retrosternal pressure subsided immediately, but did not completely disappear. Her blood pressure didn't change significantly; the next measurement showed 100/60 mmHg. He requested charge nurse Bridgette to do a 12-lead ECG and to draw blood for stat cardiac enzymes* (*see* Sect. 11.1.9). *Charge nurse Bridgette immediately realized the seriousness of the situation. The ECG showed significant ST segment depression in the chest leads* V_1–V_4.

Dr. Finn sensed that this was beyond his competence and called the anesthesiologist-in-charge, Dr. Eldridge, who appeared immediately. At the patient's bedside, Dr. Finn explained the situation, and in order to make his report complete, he pulled back the covers to do a quick physical exam. He couldn't believe his eyes. The whole bed was soaked in blood.

4.1.9 What Happened?

The causes of the symptoms are now clearly related to hypovolemia and acute anemia from blood loss. The increasing circulatory problems are all explained by the postoperative hemorrhage. The blood loss was initially compensated. The appearance of angina and hypotension showed beginning cardiac decompensation.

4.1.10 What Must Be Done Now?

The first step is to stabilize the cardiovascular system. IV fluids should be administered quickly. Elevation of the legs might bridge the gap before volume therapy takes effect. Sympathomimetics, such as phenylephrine, might be necessary, but are not a substitute for IV fluids. Acute anemia from hemorrhage is tolerated longer and better in a normovolemic state, because the reduction in viscosity more easily accommodates the increased cardiac output. (Do you remember the relationship between blood pressure and blood flow? No? Then reread Sect. 3.2.1.)

Hemoglobin/hematocrit determinations might let you estimate the extent of the blood loss and assist in calculating the amount of blood replacement needed. However, keep in mind that in acute hemorrhage without volume replacement, changes may not be seen.

In addition, the surgeon needs to be informed without delay so he/she can reevaluate the patient. In case of significant amount of blood loss, returning back to the OR is likely. The surgeon needs to be provided with all available information and so that he/she can decide if surgical intervention is indicated to stop the bleeding.

4.1.11 When Should I Transfuse Packed Red Blood Cells?

The oxygen delivery ($\dot{D}O_2$) of the body can be calculated from the product of the cardiac output (CO) and the arterial oxygen contents (C_aO_2):

$$\dot{D}O_2\,[\mathrm{ml/min}] = CO \cdot C_aO_2\,[\mathrm{ml/dl}] \quad (4.1)$$

C_aO_2 is the sum of the chemically bound and physically dissolved oxygen and can be calculated with the following equation:

$$C_aO_2\,[\mathrm{ml/dl}] = Hb\,[\mathrm{g/dl}] \cdot S_aO_2\,[\%] \cdot 1.34 + 0.003 \cdot P_aO_2\,[\mathrm{mmHg}] \quad (4.2)$$

where S_aO_2 is the arterial oxygen saturation.

When Eq. 4.2 is placed in Eq. 4.1, the influence of the individual parameters becomes obvious:

$$\dot{D}O_2 = CO \cdot [Hb \cdot S_aO_2 \cdot 1.34 + 0.003 \cdot P_aO_2]$$

A reduced $\dot{D}O_2$ can be caused by four pathophysiological mechanisms:

- **Ischemic hypoxia:**
  This hypoxia is caused by a low cardiac output. The decrease in cardiac output can be caused by various mechanisms, such as heart failure, coronary artery stenosis, sepsis, or the effects of general anesthesia.
- **Hypoxic hypoxia:**
  The reduction of the $\dot{D}O_2$ is caused by a low P_aO_2 and a low S_aO_2. A hypoxic hypoxia is seen in pulmonary disorders affecting the gas exchange or in comparatively low F_iO_2 (sees Sect. 1.1.8).
- **Anemic hypoxia:**
  The hemoglobin concentration drops below a critical level. The anemia leads to a reduction of the $\dot{D}O_2$ if compensatory mechanisms such as an increase of the cardiac output are no longer sufficient.
- **Hypoxemic hypoxia:**
  In hypoxemic hypoxia, the transport capacity of hemoglobin-bound oxygen is disrupted. It can be caused by poisoning with CO-Hb or MET-Hb binders.

Normally, $\dot{D}O_2$ is 800–1,200 ml/min and about four times higher than oxygen consumption ($\dot{V}O_2$) with 250–300 ml/min. An isolated reduction in hemoglobin therefore does not automatically cause inadequate $\dot{D}O_2$. Compensatory mechanism includes an initial increase in oxygen extraction by tissues. $\dot{V}O_2$ remains independent from $\dot{D}O_2$. The situation changes only after $\dot{D}O_2$ drops below a certain level and oxygen deficiency becomes evident. The value is called critical $\dot{D}O_2$ below which $\dot{V}O_2$ becomes linearly dependent on $\dot{D}O_2$. Hypoxia with anaerobic metabolism appears, evident by increasing lactate values and a decreasing mixed venous or central venous saturation.

Each individual has his/her own critical hemoglobin level, which is independent of compensatory mechanisms such as increasing cardiac output and/or the P_aO_2. An important requirement for adequate compensation is normovolemia. The individual critical hemoglobin value is not static, but changes dependent on many factors, such as sepsis-induced changes. Furthermore, one must notice that each individual organ or organ system has preexisting conditions which affect the tolerance of the organism to reduction in $\dot{D}O_2$.

A universal hemoglobin/hematocrit level at which one must transfuse blood does not exist [8]. Primarily, one must ensure normovolemia, in order to give the body a chance to exercise physiological compensatory mechanisms. Finally, it is important to consider other factors when deciding to transfuse [7]. Physiological transfusion triggers include:

4.1.11.1 Evidence of Tissue Hypoxia/ Increased Oxygen Extraction

Easily measured parameters include arterial blood gas analysis, lactate, and central venous saturation. A negative base excess or an increase in lactic acid represents important information to decide about transfusion.

4.1.11.2 ECG Monitoring

New ST segment changes in the ECG are indicative of a decreased myocardial $\dot{D}O_2$. Furthermore,

ECG monitoring can detect the appearance of rhythm disorders, which are often the result of myocardial ischemia and should influence the transfusion decision. Electrolytes must be checked when an arrhythmia is observed.

4.1.11.3 Cardiovascular Monitoring

Hypotension in normovolemic anemic patients is a physiological transfusion trigger.

In the post-hysterectomy patient in the PACU, the ST segment changes and the chest pain were indicative of the critical fall below a tolerated $\dot{D}O_2$ level. Causes of this fall include hypovolemia/anemia from blood loss and alveolar hypoventilation due to opioids.

>> *The patient's hemoglobin was 6.2 g/dl* (*reference 11.9–17.2 g/dl*), *and Dr. Finn ordered a transfusion of two bags of packed RBCs. The supervising anesthesiologist informed the surgeon. When the surgeon later came to the PACU*, *the patient was stable with a heart rate of 80 beats/min and a pressure of 125/80 mmHg. The signs of cardiac ischemia were no longer present. The bleeding from the wound had stopped*, *so the surgeon and anesthesiologist decided to proceed with conservative treatment*.

A coagulation screen needed to be done, *and the discharge should not be directly to the ward*, *but to an intermediate care unit for cardiac monitoring*.

Dr. Finn felt bad. How quickly an unforeseen crisis can occur. He was thankful for the offer from his supervising physician to take a break for a cup of coffee.

They were then able to talk about everything.

4.2 Case Analysis/Debriefing

4.2.1 Which Medical Errors Do You See in the Presented Case?

4.2.1.1 Opioid Administration Without Hesitation or Consideration

The repeated administration of morphine without considering the patient's condition can be dangerous. Dr. Finn and charge nurse Bridgette each gave opioids independent of one another, without asking the patient about the pain. Furthermore, they wrongly assumed over an extended period that the tachycardia was caused by pain. Other causes of the tachycardia were not considered.

4.2.1.2 Lack of Wound/Drain Checks and an Incomplete Physical Exam

Dr. Finn and charge nurse Bridgette had tunnel vision when dealing with the patient's post-hysterectomy bleeding. An important assignment of the PACU team is to recognize surgical problems and to quickly and respond adequately.

As the patient described her cardiac symptoms, Dr. Finn jumped to the conclusion that she had an acute coronary syndrome. A quick physical with a check of the surgical dressings would have only taken a moment and would have delivered important information about the cause of the symptoms.

4.2.2 Which Systems Failures Can Be Found in the Presented Case?

4.2.2.1 Lack of Introduction/Instruction/Training About Working in the PACU

Dr. Finn was assigned to the PACU for his first day. He had received no instruction about his assignment, and there was no supervision present, except for the anesthesiologist in charge of the OR schedule, Dr. Eldridge, who was only available in case of an emergency. The fact that it was during a staff shortage is no excuse.

4.2.2.2 Missing Guidelines

Dr. Finn pragmatically considered when would be the best time to call up the patients from the ward to perform the additional procedures, and he decided the quiet morning hours would be the best time. Due to a lack of guidelines about organization of such things in the PACU, charge nurse Bridgette was frustrated with Dr. Finn's decision and stated, "That is not how we do things here!"

The result was a morning phase with no work and an afternoon phase with too much to do.

4.2.3 Who Do You Identify Yourself with? Dr. Finn or Charge Nurse Bridgette?

Dr. Finn was new in the PACU. He was not familiar with the procedures and structure of the system; as discussed above, he received no instruction or training (see Sect. 4.2.2). How did he manage in this work atmosphere?

He scheduled his work, with the main goal of accomplishing as much as possible as quickly as possible. He promptly ran into a conflict with the charge nurse; the nurse did not approve of the patient coming for an epidural placement and subsequently left the PACU to take a break. As the workload increased, Dr. Finn was reluctant to call her back from her break and tried to accomplish as much of the workload as he could by himself and the one remaining PACU RN. However, even after charge nurse Bridgette returned, the workload was too much for three people. Dr. Finn's work schedule had weak points – what were they?

4.2.3.1 Time Management

By setting out a work schedule, Dr. Finn placed himself under tight time constraints. There was no allowance for possible complications in placement of an epidural or CVC, for example. Also, there was little anticipation or planning for the patients coming into the PACU from the OR.

4.2.3.2 Prioritizing

Dr. Finn was not able to schedule his work in the PACU setting.

As a result, he set the wrong priorities. Although he noticed that the patient who had a hysterectomy needed more in-depth medical care, he worked on less important tasks first, such as obtaining informed consent for the epidural.

In situations with a heavy workload, neglecting important care can have grave consequences, especially when unexpected emergencies arise. How can we compensate?

4.2.3.3 Resource Management

By recruiting resources, time can be saved. For example, in the case presented, Dr. Finn could have called in another co-worker for the epidural placement or CVC insertion or assigned the job of obtaining the missing lab results to the ward nurse.

4.2.3.4 Setting Goals Together ("Commitment")

Dr. Finn was new in the PACU and had his own vision of how the day should be organized. Charge nurse Bridgette's experiences had formed her opinions about how the day is best organized. Even though their ideas were different, they each had the same goal in mind at the end of the day. The conflict was "as soon as possible" vs. "evenly spaced throughout the day" and would have been solved by cooperative decision-making.

In good resource management and cooperative goal setting, the question "Do you identify more with Dr. Finn or with Nurse Bridgette?" is a purely rhetorical one.

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Case 5: TUR of the Prostate

5

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5.1 Case Introduction

>> The preparations for the third surgery of the morning were already complete, as the patient, Mr. Copper, was brought into the preoperative area. The attending urologist was pacing impatiently back and forth through the operating room.

Mr. Copper was a pleasant 76-year-old gentleman who was scheduled for a transurethral resection of his prostate (TURP). He had a few chronic medical problems: hypertension, hyperuricemia, and hypercholesterolemia. His medical history included past foot surgery under spinal anesthesia, 2 years ago without problems. His medications included triamterene, propranolol, allopurinol, and a statin. Yesterday evening he took 25 mg of clorazepate to sleep. The resident anesthesiologist Dr. Damian reviewed the previous anesthetic record. He was almost finished with his anesthesia residency and was assigned a urology OR today. The patient's labs were within normal limits; however, the ECG was missing. Dr. Damian asked the patient about his exercise

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tolerance. "*I live on the third floor – I walk up the steps all the time without a problem*," *said Mr. Copper. He also denied chest pain or pressure while going up to his apartment. After a quick consultation from the attending anesthesiologist*, *Dr. Eldridge*, *Dr. Damian decided to proceed with the planned spinal anesthesia*.

5.1.1 What Is the Value of a Preop ECG in Such a Patient?

In 2007, the American College of Cardiology (ACC) and the American Heart Association (AHA) issued guidelines for the perioperative, cardiovascular management in noncardiac procedures, [1] recommending a 12-lead ECG:

- In patients with unexplained chest pain or cardiac ischemia (Class I recommendation: procedure is effective).
- In asymptomatic patients with diabetes mellitus (Class IIa recommendation: evidence favors the effectiveness).
- In patients with a history of cardiac revascularization (Class IIb recommendation: effectiveness supported by a small amount of evidence).
- In patients with previous hospital stays due to cardiac problems (Class IIb recommendation).
- In asymptomatic men >45 years or asymptomatic women >55 years with ≥2 cardiac risk factors. Risk factors include diabetes mellitus, smoking, hypercholesterolemia, hypertension, obesity, and a positive family history (Class IIb recommendations). Mr. Copper, due to his age, gender, and presence of 2 cardiac risk factors, is at high risk for coronary artery disease. Even though he underwent spinal anesthesia 2 years ago without incident, a preoperative 12-lead ECG is necessary – and could even be done preoperatively with minimal hassle.

>> *In the preoperative area*, *the anesthesia technician Ron hooked Mr. Copper up to a 5-lead ECG and applied a blood pressure cuff and a finger* O_2 *saturation clip. The monitor showed the following values*:

- *Blood pressure*: *150/91 mmHg*
- *Heart rate*: *52 beats/min*
- *Pulse oximetry measured oxygen saturation* (S_pO_2) *on room air*: *97%*

Tech Ron inserted an 18 g. IV into a vein on the left arm and connected it to a lactated Ringer's solution. Dr. Damian gave Mr. Copper midazolam 1 mg IV and took Mr. Copper to the OR. Mr. Copper was helped to sit up, *and his lower back was prepped for the sterile drapes. The spinal anesthesia at the level of* L_4/L_5 *went quickly and without incident. The cerebrospinal fluid was clear*, *and Dr. Damian injected 2.5 ml of hyperbaric bupivacaine 0.5%*. *Dr. Damian was relieved that the insertion went so well*, *despite Mr. Copper's scoliosis*. "*I'm glad I got the spinal in quickly before the urologist complains that it took so long*!" *he said to tech Ron. Dr. Damian stepped outside the OR to wash his hands*, *when Ron called out loudly*, "*Dr. Damian*, *get in here*, *quick*!"

5.1.2 Are You in Agreement with the Course of Events?

5.1.2.1 Monitoring

Spinal anesthesia often leads to a drop in blood pressure, due to the sympathetic block. The extent and speed of the decrease are dependent on many factors and can be hard to predict. The largest fall in blood pressure is usually just after injection of the local anesthetic into the subarachnoid space. Therefore, careful monitoring is required during this phase in order to counteract hypotension with IV fluids and vasopressors. Unfortunately, tech Ron and Dr. Damian forgot to replace and reattach the monitoring cables when Mr. Copper entered the OR. These should have been in place during placement of the subarachnoid block.

5.1.2.2 Preoperative Fasting/ Volume Status

In this segment, nothing has been said about the preoperative fasting of Mr. Copper. According to recommendations from the ASA, adults should not eat for 6 h, and should not drink clear liquids for 2 h, prior to anesthesia [2]. By questioning the patient about his last oral intake, non-fasting patients are identified, and individual fasting vari-

ations are identified. These are often much longer than recommended by the ASA guidelines.

Before performing spinal anesthesia, a patient should be euvolemic. Mr. Copper was scheduled as the third patient of the day. One must assume that he arrived in the induction room with a volume deficiency. Therefore, at least 800–1,000 ml of IV fluids should have been administered before the spinal anesthesia was begun.

>> As Dr. Damian hurriedly ran back into the OR, he saw the following situation: Mr. Copper was turned to his side gagging. Technician Ron was supporting his head, and the OR nurse was holding an emesis basin. "Whoa, does he ever look pale," thought Dr. Damian. None of the cardiovascular monitoring was hooked up yet. Dr. Damian leaped over to the surgical table and grabbed a wrist to feel for a pulse. It was weak and fast.

5.1.3 What Is the Most Urgent Treatment?

The symptoms that Mr. Copper displays are consistent with the diagnosis of hypotension caused by spinal anesthesia. This tendency has already been discussed in Sect. 5.1.2. The nausea may be explained by the inadequate cerebral perfusion. The most important treatment to be performed immediately is to treat the sympathetic blockade.

>> Dr. Damian administered 200 μg of phenylephrine, assuming that the patient's problem was hypotension. The first blood pressure measured after that was 90/56 mmHg, and Mr. Copper showed clinical improvement. The IV fluid was flowing at maximum speed, and by testing with a cold vial, the sensory level of the spinal anesthesia was up to T_8. The urologist entered the room saying, "Can we finally get going in here?" He turned to Dr. Damian and continued, "We have a huge schedule to get done today."

"Warm hearted, as always," thought Dr. Damian, as he nodded to the surgeon to begin. In the meantime, anesthesia tech Ron had hung a second bag of lactated Ringer's. Mr. Copper's hemodynamics had stabilized:

- *Blood pressure: 124/76 mmHg*
- *Heart rate: 49 beats/min*
- *S_pO_2: 98% with 4 l O_2*

Mr. Copper received 3 mg midazolam IV in divided doses, and he dosed off peacefully.

Surgery took longer than expected. The prostate was small, but highly vascularized. The surgeon cursed quietly to himself. Mr. Copper woke up once and asked where he was. "Oh, right." he said after it was explained to him, and he fell asleep again. After about 70 min of operating time, the procedure was complete. The surgeon inserted a transurethral bladder irrigation catheter and removed the sterile drapes. Anesthesia tech Ron disconnected the monitoring cables, and Dr. Damian completed the anesthesia record. Together, the anesthesia and OR team pushed Mr. Copper out of the OR. Just outside the OR door, Mr. Copper suddenly began uncontrollably moving his arm. Then he began to gag exactly as he had gagged at the beginning of the operation. He looked cold and clammy. Then his eyes rolled back and he lost consciousness.

5.1.4 What Is Your Initial Course of Action?

There is no shortage of differential diagnoses for the acute deterioration of Mr. Copper's condition. At this critical moment, the various differentials don't really matter – the vital signs are of utmost importance.

- Level of consciousness
- Breathing
- Pulse

If the patient is unconscious and not breathing, testing for a pulse is optional [4–6], and CPR must be begun.

An early call for help is important to get qualified support so that defibrillation or pacing can be done if necessary.

If respiration is sufficient (perhaps with the chin lift or jaw thrust technique), then assess cardiovascular status. Mr. Copper should be hooked up to a monitor as soon as possible. The transport from the OR to the PACU is a critical time and usually takes place with minimal monitoring.

The equipment and personal requirements of a PACU were discussed in more detail in Case 4 (Sect. 4.1.2).

>> Mr. Copper didn't react to firm pressure to the sternum. After Dr. Damian lifted the jaw, Mr. Copper was clearly moving air. Dr. Damian thought he was feeling a slight carotid pulse. The cleaning crew was in the OR with their big cleaning cart. "Alright, let's move quickly to the PACU!" yelled Dr. Damian.

The PACU was very close to the OR. A patient who was very nearly ready for discharge anyway was quickly pushed into the hallway so that there was enough space to work in this 4-bed PACU. Anesthesia technician Ron hooked up the blood pressure – the first value was 58/35 mmHg. Dr. Damian administered phenylephrine IV, while tech Ron got the ECG and pulse oximeter hooked up. The heart rate was 44 beats/min, and the pulse oximeter showed 91% saturation. Mr. Copper moaned and opened his eyes a little. Thanks to the phenylephrine, the pressure was up to 90/52 mmHg.

"Could you please call attending anesthesiologist Dr. Eldridge?" Dr. Damian said, turning to tech Ron. The urologist blared out from the doorway. "Then, you tell him yourself that your spinal went too high and that we can't go forward on any surgery because you've got to take care of this!" Dr. Damian was annoyed with this comment, but it was possible that the spinal anesthesia had been too high. "But maybe there is another cause. After all, I used hyperbaric bupivacaine," Dr. Damian thought to himself.

5.1.5 Which Further Diagnosis Should Be Considered?

The loss of consciousness described in the case could have many causes. Basically, surgery and anesthesia-related causes as well as individual patient causes must be taken into consideration. The following possibilities – which may or may not be a complete list – should be considered.

- High spinal anesthesia
- Transurethral resection of the prostate (TURP) syndrome
- Absolute or relative hypovolemia
- Embolus/thromboembolism and also air embolism
- Cardiac causes, e.g., arrhythmia or MI
- Carotid stenosis, stroke
- Anaphylactic reaction

After stabilizing the vital signs, the various diagnoses in the differential should be validated or excluded by the fastest and simplest tests possible. Prioritize according to the principle: "When you hear hoof beats, don't think of zebras."

>> Attending anesthesiologist Dr. Eldridge entered the PACU and asked, "Is this the patient that didn't receive the ECG? How is he doing?" Dr. Damian responded, "I gave phenylephrine; now I'm giving a colloid solution with a pressure infuser. The BP is now 115/65 mmHg, the heart rate 70. Yeah, he's doing better." Immediately came the next question, "What do you think is the cause?" Dr. Damian held his breath. He hadn't yet thought about the possible causes; he was busy stabilizing the patient. "I considered a high spinal, but I haven't yet had time to test the level," he responded.

5.1.6 Which Symptoms Do You Expect in a High Spinal Anesthesia?

Spinal anesthesia, through the preganglionic sympathetic block, generally leads to a reduction in systemic vascular resistance. At a high level, hypotension is significantly more pronounced. Vasodilation leads to venous pooling with reduced filling of the right atrium and reflexive bradycardia. Responsible reflexes include the "reverse" Bainbridge and the Bezold–Jarisch reflex [3].

In a complete sympathetic block, the cardio-accelerator nerves are also blocked, which leads to reinforcement of the bradycardia. Trendelenburg positioning and volume administration may be ineffective. A total spinal would lead to respiratory insufficiency due to paralysis of the muscles of respiration, severe hypotension, respiratory arrest, dilated pupils, and loss of consciousness.

5.1.7 What Is the Treatment for a High Spinal?

Therapy consists of treating the symptoms:

- Administration of vasoconstrictors, e.g., catecholamines
- IV fluid administration
- Administration of an anticholinergic, such as atropine, to treat bradycardia
- Oxygen administration
- If unconscious or respiratory compromise, intubation and ventilation

Positioning the patient to increase venous return cannot be done when a hyperbaric local anesthetic has been used. In this circumstance, Trendelenburg positioning could lead to a total spinal.

>> *The physical exam showed that the level of the spinal was* T_4.

"*I wouldn't call that a high spinal*," *said Dr. Eldridge*, "*however, we now have an explanation for the relatively slow heart rate*." "*Mr. Copper takes a β-blocker for his hypertension*," *added Dr. Damian who was happy that he had thought of something to add*. "*Hypertension is not exactly his problem right now*," *replied Dr. Eldridge who gave Dr. Damian a condescending nod*. "*How do you feel, Mr. Copper? Do you have a headache?*" *he asked the patient*. "*I'm so tired; I just want to sleep. Breathing in is so hard. No, no headache*," *replied Mr. Copper, who then went back to sleep*.

The next check showed that the blood pressure had dropped back down to 80/55 mmHg. Heart auscultation was unremarkable. There were faint respiratory sounds, but Dr. Damian couldn't hear anything abnormal. Attending anesthesiologist Dr. Eldridge ordered more phenylephrine as a holdover until the norepinephrine infusion arrived. Mr. Copper received oxygen via a face mask at 4 l/min. "*Which tests would you like to perform next?*" *asked Dr. Eldridge as he turned to Dr. Damian.*

5.1.8 Which Tests Would You Do Next?

The first priority is a more complete physical exam. In the presented case, the pulmonary and cardiac exam was unremarkable. The neurological exam is only partially possible, due to the spinal anesthesia and pronounced fatigue. The decreased level of consciousness may be due to cerebral hypoperfusion or hypoxia.

When in the PACU, be sure to always remember to examine the surgical area (see Chap. 4)!

5.1.8.1 Assessment of the Surgical Area

Typical local complications of a TURP include prostate bleeding, bladder tamponade, and bladder perforation [7]. In order to prevent bladder tamponade, the bladder is usually postoperatively irrigated with a special catheter. The irrigation fluid must be collected and measured after draining out of the bladder; if not, the catheter could be clogged up or the bladder could have been perforated. Clinically, bladder perforation during bladder irrigation resembles an acute abdomen. The diagnosis is made more difficult by "sufficient spinal anesthesia," with a T_4 level.

An estimate of the postoperative blood loss cannot be made by observing the color of the fluid drained from the bladder. A hematocrit check is better, ideally with an arterial blood gas.

5.1.8.2 Arterial Blood Gas Analysis

A single arterial blood gas analysis delivers valuable information about ventilation, oxygenation, electrolytes, and pH.

5.1.8.3 12-Lead ECG

A further diagnostic step would be a 12-lead ECG, which delivers information about a possible myocardial ischemia/infarction or pulmonary embolism.

>> *The irrigation catheter drained a large amount of red-tinged fluid. Dr. Damian thought the color looked the same as usual. He examined the 12-lead ECG. There was nothing remarkable except an increased Sokolow index. He couldn't make out any arrhythmia or signs of cardiac ischemia or acute right heart failure. The arterial blood gas analysis showed*:

- *Hb*: *7 g/dl* (*reference 11.9–17.2 g/dl*)
- *HCT*: *22 %* (*reference 37–47 %*)
- Na^+: *125 mEq/l* (*reference 136–145 mEq/l*)
- K^+: *3.1 mEq/l* (*reference 3.8–5.2 mEq/l*)

- *P_aO_2: 80 mmHg (reference 70–100 mmHg in room air) with administration of 4 l/min O_2 via face mask*
- *PCO_2: 34 mmHg (reference 36–44 mmHg)*
- *pH: 7.30 (reference 7.35–7.45)*
- *BE −3.5 (reference ±2)*
- *Glucose: 145 g/dl (reference 70–120 g/dl)*

"*Just as I suspected*!" *said supervising physician Dr. Eldridge to Dr. Damian*. "*Now you know what you've got to do, right*?"

5.1.9 What Is Your Suspected Diagnosis?

The blood gas analysis shows anemia, hyponatremia, and hypoxia, with metabolic acidosis with partial respiratory compensation. Considering the situation and the clinical symptoms, the suspected diagnosis is TURP syndrome. In addition, there is a slight hyperglycemia.

The TURP syndrome is caused by intravascular absorption of large amounts of hypotonic irrigation solution [7]. The result is hypotonic hyperhydration with hyponatremia. The irrigation solution is free of electrolytes, so that a precise electrical cauterization can be carried out. Since the original use of water has been replaced with a standardized sorbitol mannitol solution (e.g., 27.0 g sorbitol, 5.4 g mannitol in 1 l, osmolarity = 178 mosmol/l), the incidence of serious cases of TURP syndrome has decreased.

5.1.9.1 Symptoms of TURP Syndrome

Central Nervous System Disorders

- Cerebral edema, caused by acute hyponatremia and hyposmolarity, presents clinically as restlessness, nausea, and decreased level of consciousness. Further progression can lead to hallucinations and seizures. In the past, when the solution contained glycine, visual disturbances were observed.

Cardiac Symptoms

- Usually, the patients develop systolic and diastolic hypertension due to hypervolemia. A primary tachycardia may progress into reflexive bradycardia and cardiac failure.

Intravascular Hypoosmotic Hyperhydration

- The increase in volume leads to an increase in central venous pressure (CVP), with danger of developing pulmonary edema with dyspnea and hypoxemia. In severe cases of decreased serum osmolality, an intravascular hemolysis can occur, with decreases in Hb/HCT and increases in potassium and LDH.

Coagulation Disorders

- Furthermore, coagulopathy due to the dilution may be seen, sometimes complicated by the influx of tissue thrombokinase from the prostate tissue, leading to activation of the coagulation cascade.

The seriousness of TURP syndrome is highly variable. It is dependent on volume and rate of absorption which is influenced by:

- The size of the adenoma
- Duration of resection (warning: resection durations of over >60 min)
- Surgical skills
- Intravascular pressure (volume status, patient positioning)
- Age of patient (worse hydration status in older patients)
- Intraprostatic pressure (optimal <15 cm H_2O)
- Height/hydrostatic pressure of the irrigation fluid (optimal <60 cm)

The simplest patient monitoring method is neurological monitoring; therefore, spinal anesthesia is the best anesthetic choice. Intermittent checks of the serum sodium concentration give information about the extent of intravascular absorption (Table 5.1).

Rough calculations of the absorbed volume can be done with (Eq. 5.1):

$$\textbf{Absorbed volume} = \left(\left(\frac{[\textbf{Sodium}]_{\text{preoperatively}}}{[\textbf{Sodium}]_{\text{postoperatively}}} \right) \times \textbf{ECF} \right) - \textbf{ECF} \tag{5.1}$$

(ECF = extracellular fluid ≈ 20 % of body weight in kg)

Table 5.1 Severity of the TURP syndrome

TURP syndrome	Na+ (mEq/l)
Low	135–125
Moderate	125–120
Moderately critical	110–120
Critical	<110

5.1.10 What Is the Therapy for TURP Syndrome?

The therapy consists of ending the surgery and reducing the fluid intake [7]. In symptomatic patients with low osmolarity ($Na^+ < 120$ mEq/l), loop diuretics and hypertonic saline administration may be indicated. Abrupt correction of the electrolyte imbalance may result in central pontine myelinolysis. How much Na^+ is needed can be estimated using Eq. 5.2. The substitution is done with a target increase in plasma Na^+ of 1.5–2.0 mEq/l/h, until the Na+concentration is >125 mEq/l.

Calculation of the Na^+ requirement in mEq (Eq. 5.2):

$$\mathbf{Na^+ requirement} = \left(\left[\mathbf{Na^+}\right]\mathbf{desired} - \left[\mathbf{Na^+}\right]\mathbf{current}\right) \times \mathbf{ECF} \quad (5.2)$$

(ECF = extracellular fluid, ≈20 % of the body weight in kg)

5.1.11 Does Every Transurethral Prostate Resection Have the Danger of a TURP Syndrome?

The question's phrasing already implies a No. As discussed in Sect. 5.1.9, TURP syndrome is the result of hypotonic hyperhydration. During the TURP, several bipolar prostate resection techniques can be used, and the use of an electrolyte-free irrigation fluid is not necessary. Absorption of isotonic saline solution rarely causes complications, even when large amounts enter the circulation.

An additional technique is laser resection of the prostate, which also uses isotonic saline solution. Laser resections are often time-consuming, but have the advantage that blood vessels during the resection are cauterized, thereby preventing inundation and bleeding. It is therefore the procedure of choice in large adenomas and patients with congestive heart failure who would not tolerate hypervolemia very well.

>> Dr. Damian was unsure about the therapy as attending anesthesiologist Dr. Eldridge gave the orders. "Ron, give Mr. Copper 20 mg furosemide IV and prepare a CPAP mask. Also, we need to change the norepinephrine infusion for dobutamine – his heart needs some help to clear the absorbed fluid. Why don't you go ahead and do that, Dr. Damian. I will place a CVC."

Mr. Copper didn't show any reaction as the procedures were performed; he merely moaned as the CPAP mask was fastened around his head. Placement of the CVC occurred without incident. After exchanging the vasopressor infusions, Mr. Copper's blood pressure dropped to 60/35 mmHg, and the heart rate was 50 beats/min. "Shouldn't the blood pressure be higher?" asked Dr. Damian, turning to his attending. "It will be in a second," replied Dr. Eldridge. "But you're right, we need to do something to improve his cardiac output – what would you suggest?"

5.1.12 What Could the Attending Anesthesiologist Dr. Eldridge Have Meant?

The arterial blood gas showed anemia with a hematocrit of 22 %. Every person has his own individual critical hemoglobin level. Circulatory depression in the presence of anemia and euvolemia is an indication for transfusion (Sect. 4.1.11).

>> Tech Ron prepared to transfuse two bags of packed RBCs, under the orders of attending anesthesiologist Dr. Eldridge. Meanwhile, the dobutamine infusion was running at 20 µg/kg/min; its only effect was that the pulse was now 110

beats/min. The blood pressure was 70/45 mmHg. "It will be better just as soon as he's got the blood," said Dr. Eldridge.

The bags were pressure transfused and brought the pressure up to 85/50 mmHg. However, shortly after the last bag was in, the blood pressure fell again. The heart rate increased to 120 beats/min.

5.1.13 What Would You Do Now?

Obviously, the therapies employed have not lead to the desired result. This could be because the therapies were not intensive enough, or because the working diagnosis, TURP syndrome, is wrong. An additional possibility is that the patient does indeed have TURP syndrome and it is complicated by additional undiagnosed diseases or other physiological disorders. In any case, the effect of the therapy must be critically analyzed. If there is doubt about the effectiveness of the therapy, one must follow the principle of "Restart from Zero!"

>> A new arterial blood gas showed that the hematocrit had increased to 30%. The Na^+ concentration was now 130 mEq/l (Reference 136–145 mEq/l). The other values were also almost in the normal range. The attending urologist came into the PACU to see why he couldn't proceed immediately with the rest of his surgical list. "Do you need help with your cognitive work? Why don't you let me help you figure out what the problem is?" he said with a sarcastic smile. He removed the blanket to have a look at the irrigation catheter. The anesthesiologists couldn't believe their eyes – Mr. Copper's torso was covered in urticaria. "This has got to be a latex allergy. We always use latex irrigation catheters. I better change that," said the urologist and disappeared into the OR to get the new supplies and an assistant.

Silently, the attending anesthesiologist Dr. Eldridge changed the dobutamine infusion back to norepinephrine. He asked tech Ron to quickly administer 500 ml of normal saline, then bring him prednisone and diphenhydramine. Before the urologist returned, Mr. Copper's circulatory status had improved, and he was fighting the CPAP mask.

5.2 Case Analysis/Debriefing

5.2.1 Which Medical Errors Do You See in the Presented Case?

5.2.1.1 ECG

The patient should have received a preoperative ECG. This omission was discussed in Sect. 5.1.1.

5.2.1.2 Monitoring After the Placement of the Spinal Anesthesia

Hypotensive episodes are most commonly seen within the first few minutes. In this case, the monitoring was insufficient (see Sect. 5.1.2).

5.2.1.3 Monitoring of the Amount of Intravascular Absorption

While neurological status was assessed, additional monitoring of intravascular absorption of irrigation fluid is needed during lengthy procedures. A reliable laboratory test is Na^+ concentration in serum (see Sect. 5.1.10). This was not performed.

5.2.1.4 Monitoring the Blood Loss

No tests were done to monitor blood loss, even though there was more bleeding than usual.

5.2.1.5 Monitoring of the Extent of the Spinal Anesthesia

The anesthesiologist did not test the level of the spinal anesthesia during the surgery; nor did he test it after the surgery, or before the patient's transfer to the bed. The same anesthesiologist later listed "high spinal" in the differential diagnosis list, because he couldn't rule it out as part of the differential diagnosis for his crashing patient.

5.2.1.6 Measurement of the Central Venous Pressure

After placement of the CVC in the PACU, no one measured the pressure! One would expect the measurement to be extremely helpful with the differential diagnosis: TURP syndrome, fluid

overload, and congestive heart failure. This diagnostic tool was not used. Therefore, the patient received suboptimal care.

5.2.2 Which Systems Failures Can Be Found in the Presented Case?

5.2.2.1 Standard Monitoring of the Amount of Absorbed Irrigation Fluid

With men undergoing TURP, the amount of absorbed irrigation fluid must be monitored. Standard operating guidelines or protocols must be available and understood by all involved.

5.2.2.2 Missing ECG

The ECG, which was ordered by the anesthesiologist doing the preoperative evaluation, was not done. The anesthesiologist assigned to the case should check to see that all required documentation (such as lab results, X-rays, consent paperwork) are present, and all additional orders (ECG, echo) have been performed and are documented in the medical record. A checklist might be helpful to prevent mistakes.

5.2.3 How Is the Cooperation Between the Two Specialists?

The two departments, anesthesiology and urology, don't have any shared goals. The urologist is interested in getting through his surgery schedule as fast as possible and views the anesthesiologist as a necessary service provider who should not slow things down. Accordingly, the atmosphere is tense. The anesthesiology team knows that the urologist views them as inferior, and is caught between a rock and a hard place. On the one hand, anesthesiology desires good teamwork with the urologist; on the other hand, this comes at the expense of the quality of patient care. The anesthesiologists attempt to avoid conflict with the urologists.

Because of this tense relationship among physicians, the anesthesiologist cut corners and compromised patient safety. The uncooperative working environment can be traced back to the lack of communication about the critical state of the patient. Tasks should have been explicitly mentioned and delegated. Dr. Damian thought he was working with an experienced team in which team members, during times of great responsibility, anticipate tasks, implicitly exchange information, and react accordingly. His preconceived expectation proved to be wrong.

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Case 6: Tonsillectomy: Hurray, a Child!

6

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6.1 Case Introduction

>> Dr. Maverick had two and a half years of experience in anesthesiology and had rotated through all the departments – except – pediatric anesthesia, an area in which he felt he knew nothing at all. Therefore, he was excited when his attending anesthesiologist in ENT, Dr. Eldridge, sent him to do the preoperative evaluation of a child. Actually, he was hoping to get to do the anesthesia the next day.

In preanesthesia clinic, a very impatient mother was waiting. Ms. Miller was accompanying her two-and-a-half-year-old son, Kevin, who was supposed to have a tonsillectomy the next day. She had already filled out the anesthesia preoperative form very carefully. She listed her son's weight as 20 kg and pretty much checked everything else "no." Dr. Maverick was relieved; he wouldn't have to inquire about much, and they would be finished quickly.

As he reviewed the patient's preanesthesia form, Ms. Miller spoke up: "I tried to answer

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everything as best I could, but I still have a few questions. Kevin had his second measles, mumps, and rubella vaccination 3 weeks ago. Is it OK for him to have surgery now?" Dr. Maverick was a bit unsure himself and wondered about the timing of general anesthesia after vaccination.

6.1.1 What Must Be Considered After Vaccinations Before General Anesthesia?

The optimal time of elective schedule after vaccination is controversial [1]. There is no evidence showing that vaccinations lead to postoperative complications after general anesthesia. Neither has it been shown that general anesthesia, despite a slight immune suppressive effect, weakens the effect of the vaccination.

However, in order to clearly differentiate between postoperative complications and vaccination side effects, a time period of 1–3 weeks between vaccination and surgery is recommended for live, attenuated vaccinations, and a time period of 2 days is recommended for inactivated vaccinations [10, 11]. In the measles, mumps, and rubella vaccination (MMR) there are living attenuated viruses. Kevin received the vaccination 3 weeks ago, so there is no need to postpone the surgery. Of major concern is that the risks of additional barriers to immunization far outweigh the risk of anesthesia and surgery in a recently immunized child [5].

>> Dr. Maverick thought that the vaccination 3 weeks ago was OK, but he checked his all-knowing anesthesia app, just to be sure. "That's a relief," said Ms. Miller. "I was at the pediatrician's office with Kevin last weekend and they weren't so sure. Actually, I only went because the ENT doctor told me that Kevin had to have a statement from his pediatrician saying he can undergo anesthesia. Lately, Kevin has been constantly sick. His cough is almost gone now, but his nose is always running. The pediatrician was concerned that the anesthesiologist would delay the procedure because of the runny nose. What's your experience with kids like this?"

Dr. Maverick hoped that Ms. Miller wouldn't notice how red his ears had become, because he had absolutely no experience with kids like this or kids at all.

6.1.2 What Should Dr. Maverick Say to the Mom?

6.1.2.1 Preoperative Medical Clearance

Some surgeons may refer patients to their primary care physician or cardiologist for preoperative medical clearance. Preoperative medical clearance was intended to prevent the anesthesiologist from canceling elective surgery at the last minute. However, preoperative medical clearance does not relieve the anesthesiologist of his/her duty to make his/her own informed decision; therefore, in most cases, preoperative clearance is not necessary and without value.

6.1.2.2 Upper Airway Infections in Children

In the past, elective surgery for all children who had an upper airway infection was postponed due to the association of respiratory complications. However, these complications are easily avoided or managed [12]. Nowadays, only clinically manifest upper airway infections with fever above 38.5 °C (101.3 °F), a general feeling of malaise, and infected looking sputum or nasal secretions are criteria to postpone an elective surgery. In such cases, surgery should be postponed until the symptoms have subsided.

In ENT, the purpose of the surgery is often to remove the focus of the infection, in order to prevent reoccurring infections. Whether or not a procedure will be postponed due to an infection is an individual decision. However, surgical treatment of a focal infection source should not be postponed.

>> Ms. Miller was relieved that the surgery didn't need to be postponed. Her husband had gotten a few vacation days in order to take care of their younger daughter. Now Ms. Miller had just one last question: "When will Kevin have to have his blood drawn?"

6.1.3 Which Blood Values Would You Check?

Kids hate having blood drawn; needles are the major cause of fear about anesthesiologist and doctor visits. A detailed history and a thorough physical checkup usually yield adequate information. The indication for lab tests should be critically evaluated. The most relevant question is: How will the test results impact the anesthetic management?

Also, in procedures with concern over postoperative bleeding, like tonsillectomy, coagulation studies may lend a sense of security. However, they have little predictive value for postoperative bleeding complications. A good bleeding history with questions targeting past bleeding/clotting abnormalities is much more relevant and useful [3]. Testing, including blood tests or chest X-ray, is not needed, unless there are no specific clinical indications.

>> Ms. Miller was so relieved to hear that Kevin wouldn't need to have blood drawn; she had been dreading that part most of all. Holding him still and trying to find a vein in the poor child had always been difficult. Dr. Maverick finished explaining about the procedure like the EMLA (lidocaine and prilocaine) patch and premedication.

"OK then, I have just one last question," said Ms. Miller. "Kevin often wakes up at night for something to drink. Until what time is he allowed to drink?" Now she had caught Dr. Maverick off guard for sure. He had thus far managed to impress Ms. Miller with his most basic of anesthesia knowledge; he really didn't want to blow his image now. "Err on the safe side," he thought to himself and chose 6 h as the preoperative fasting interval, for liquids as well as solids. Ms. Miller wrinkled her brow a bit, but his sovereign demeanor prevented her from questioning further.

6.1.4 What Is the Recommended Fasting Period for Children Before Anesthesia?

Pediatric patients don't tolerate long periods of fasting. Especially preemies, newborns and small children could quickly become hypovolemic and may develop metabolic disorders without food and water. The preoperative deficit is calculated from the fasting time in hours, multiplied by the hourly requirement of the child. In infancy, this is calculated as the "**4-2-1 Rule**," for example:

- Four ml/kg/h for the first 10 kg of body weight
- Two ml/kg/h for every kg above 10 kg body weight
- One ml/kg/h for every kg above 20 kg body weight

For elective procedures, the ASA recommends the fasting periods listed in Table 6.1 [2].

Table 6.1 Minimum fasting periods in elective procedures [2]

Fatty foods	Light non-fatty meal, nonhuman milk, infant formula	Breast milk	Clear liquids
8 h	6 h	4 h	2 h

The general stress reaction to accidents, injuries, intra-abdominal pain, or severe illness significantly slows stomach emptying, so that it is not useful to wait out the fasting period in such cases. Pediatric patients would not have an empty stomach by waiting under such circumstances; on the contrary, increasing stress and pain would increase gastric acid production. However, the time period between the last oral intake and the trauma gives a false impression of a fasting state (see Sect. 20.1.2).

>> The next morning, Dr. Maverick came in super motivated and ready to work. The past evening he had spent reading a pediatric anesthesia chapter with all the doses because his attending had promised to let him do the pediatric anesthesia case.

He was in a good mood walking into the OR where the anesthesia technician Robert had already set up everything. Tech Robert was experienced in pediatric anesthesia, and Dr. Maverick was thankful to have him at his side today. Kevin was brought from the preanesthesia area; he and his mom said goodbye, with tears pouring down each of their cheeks. Premedication with a 10 mg midazolam rectal suppository didn't seem to be working. Tech Robert didn't let this get to him; he carried Kevin and his stuffed animal into the OR, as the surgical nurses looked on with condescending looks.

6.1.5 How Should Pediatric Patients of This Age Be Premedicated?

Preoperative sedation is often desired in children 6 months of age and older, because separation from the parents leads to separation anxiety and therefore very uncooperative patients. Alternately, anesthesia may be induced with the parents present in order to avoid separation anxiety. It is common in many pediatric hospitals to have the parents accompany the child who is having elective surgery to the OR. Sedative premedication should be avoided in children with obstructive sleep apnea, intermittent obstruction, or very large tonsils.

Midazolam is the preferred benzodiazepine in pediatric anesthesia because it has a rapid onset of action and a short half-life.

6.1.5.1 Rectal Premedication

For rectal administration: 0.5–1 mg/kg (maximum 15 mg) begins to induce sedation after just 5 min. After 15 min, children should be sufficiently sedated. Sometimes, however, inadequate sedation is observed (see Sect. 28.1.3).

6.1.5.2 Oral Premedication

For oral premedication, a dose of 0.3–1 mg/kg (maximal 15 mg) is recommended, given either in tablet form (school-aged children may begin to swallow tablets) or as syrup.

6.1.5.3 Nasal Premedication

Nasal premedication with midazolam (0.2–0.4 mg/kg) works just as quickly as rectal administration. The fluid irritates the children's airways, making for very unhappy patients. If used, consider anesthetizing their nasal mucosa with a local anesthetic – for example, nasal spray or nose drops made with lidocaine 1 %.

Paradoxical excitation after midazolam is uncommon. It may occur due to a low dose or too early administration; the rapid onset of action and short duration demands careful timing between the preoperative area and the OR. Hence, midazolam is generally not given until the OR is ready to receive the patient.

Anticholinergics are no longer used as premedicants, now that succinylcholine and halothane are rarely used. In special circumstances, such as during strabismus surgery, anticholinergics can be given IV during or after induction of anesthesia (see Sects. 28.1.4 and 28.1.5). Barbiturates and neuroleptics are only given if there is a special indication. In general, one must use caution in pediatric premedication when increased intracranial pressure, cyanotic heart disorders, or obstructive sleep apnea is present.

>> Robert, the anesthesia tech, hooked Kevin up to the usual monitoring, and Dr. Maverick started working on an IV. "Skin looks mottled," he thought to himself as he removed the EMLA patch from the back of the hand. It made no difference; they had to get a line in. Tech Robert held Kevin's arm still; the dorsal hand showed only a hint of a tiny recognizable vein. Dr. Maverick declined to listen to tech Robert's suggestion to look for a vein on the patient's feet. "There wasn't an EMLA patch there," he thought, making up his mind to stick the back of the hand. His disappointment was enormous, as not one drop of blood was to be found, and Kevin began to cry. "I'll try just once more," thought Dr. Maverick. "Who knows when I'll have another chance?"

Sadly, his second attempt on the other hand was no more successful. Kevin now began to scream as if he was being burned alive. Dr. Maverick gave up. "You try," he said to tech Robert. Robert touched a nice vein in the elbow, but because Dr. Maverick didn't hold the arm tight enough, the child yanked away at just the critical moment, and the vein was shot. Dr. Maverick had underestimated the little guy's strength.

6.1.6 What Are the Possible Causes of the Difficult Venous Access?

Placement of an IV in conscious children should only be done after the skin has been anesthetized, such as with EMLA cream ("eutectic mixture of local anesthetics"). One gram EMLA cream

contains 25 mg of prilocaine and 25 mg of lidocaine. In order to achieve the full effect, the cream must be on for at least 60 min. If the EMLA patch is removed just before attempting IV placement, the vasoconstrictive effect of the prilocaine causes the IV insertion to be very difficult. Therefore, the patch should be removed at least 20 min before attempting an IV. EMLA is not approved for newborns <3 months of age due to the possible methemoglobin formation.

Fear and stress also lead to vasoconstriction. Waiting for a good pharmacological premedication can lead to success faster than repeatedly attempting IV placement, which increases stress (and not just the child's stress). The obesity epidemic has reached the pediatric population as well. Therefore, it is recommended to search for and mark the best vein while first seeing the patient; ordering an EMLA patch to be placed by the admitting nurse alone is inadequate.

>> *The attending anesthesiologist, Dr. Eldridge, heard the screaming coming from the OR and looked in. "What are you doing? There isn't any need for an IV before induction! Begin the induction with the mask!" he ordered. Dr. Maverick was glad he had reviewed the pediatric chapter the night before. He opened the sevoflurane vaporizer and pressed a face mask over Kevin's mouth and nose. Kevin subsequently squirmed and screamed even more, before finally slowly falling asleep. Even so, Dr. Maverick was actually very dissatisfied with himself and the events thus far. At least he could hold the child's mask well, and the child could ventilate well. As Kevin fell into a deep sleep, Dr. Maverick wanted to try once more to get an IV. Dr. Eldridge took over the mask, and Dr. Maverick chose a vein on the foot. With such deep anesthesia, he would have never expected the patient to react, but Kevin did and another vein was shot. Now Dr. Eldridge lost his patience. He handed over the mask to Dr. Maverick and felt the last visible foot vein on the other foot, and IV access was finally secured.*

"Just got lucky!" thought Dr. Maverick. Dr. Eldridge gave Kevin 4 mg/kg body weight propofol and 2 µg/kg of fentanyl, and he began to talk to the surgeon, who had just walked into the OR. Tech Robert gave Dr. Maverick a 2.5 laryngeal mask airway. Dr. Maverick was actually expecting an endotracheal tube. Much to his surprise, Dr. Maverick placed the laryngeal mask airway without incident, and it even seemed tightly sealed. He began to mechanically ventilate Kevin and declared the patient was ready for surgery. As the ENT surgeons started surgery, Dr. Maverick filled out his anesthesia record. Tech Robert went off to "prepare the next patient." Attending anesthesiologist Dr. Eldridge was paged to another OR and with a "See, it works!" comment, he was gone.

Suddenly the ventilator's alarm sounded: low respiratory minute volume was on the display. Yes indeed, instead of the programmed 200 ml of tidal volume, only 80 ml were going in. The oxygen saturation was good, and Dr. Maverick could not hear any sounds to indicate that the mask lost its seal. "Maybe the child is trying to breathe on his own," he thought, as he gave another 2 µg/kg of fentanyl. The heart rate decreased a little, but the respiratory situation did not improve. Dr. Maverick turned the ventilator to manual. With the bag, he was able to get 120 ml in very slowly. For a short moment he was relieved, then the tidal volume dropped to only 30 ml, and Kevin's O_2 saturation began to fall.

What is wrong now? Had the child aspirated – did he have a broncho- or laryngospasm? "This laryngeal mask airway is crap!" thought Dr. Maverick, and he began to get nervous. Because he felt he no longer had the situation under control, he called his attending, Dr. Eldridge, stat to the OR.

6.1.7 What Is Wrong? Is a Laryngeal Mask Airway Not Suitable for ENT Procedures?

Use of an laryngeal mask airway has increased in ENT surgery during the past few years. As opposed to orotracheal intubation, it does have some advantages [7]. Due to the decreased airway trauma, it causes less laryngospasm and bronchospasm during emergence from anesthesia and in the PACU. This is especially helpful in adenoidectomies and tonsillectomies, because pediatric patients are seldom free of upper airway infection.

The laryngeal mask airway prevents the aspiration of blood. The larynx is better protected from blood coming out of the pharynx with an laryngeal mask airway than the usual small-sized orotracheal tube, which, though often used in pediatric anesthesia, does not protect from aspiration of blood. A more peaceful awakening, reduction of sore throat pain, and reduced coughing with the laryngeal mask airway are more than just comfort measures, because these benefits combine to mean less bleeding in the immediate postoperative phase.

On the other hand, there are some disadvantages of laryngeal mask airways. During the placement of the mouth gag by the surgeon, it can become dislocated or obstructed. To prevent this, the laryngeal mask airway should not be secured in place when the surgical mouth gag is placed; the mask may even need to be disconnected from the breathing circuit. Loosening the mouth gag may solve the problem. The laryngeal mask airway is only successful if used by experienced surgeons and anesthesiologists who are committed to making this method work well through good communication with one another.

In this case, taping the laryngeal mask airway to the jaw let to its obstruction when the mouth gag was placed, a typical complication that occurs at the beginning of surgery.

>> *For the attending anesthesiologist, Dr. Eldridge, the problem was clear; he asked the surgeon to open the mouth gag, and reposition it. This appeared to be a completely normal procedure; the surgeon laid his instruments to the side and did it. The problem was solved, and Kevin could be effortlessly ventilated.*

Dr. Maverick stood there like an idiot and thought: "*They could have said that to me earlier…*" *Tech Robert entered and asked what was going on. Dr. Maverick explained the situation quickly and added that he had never anesthetized a pediatric patient before and certainly not for ENT surgery. To that tech Robert replied* "*You should have said something earlier, no one knew that. If I had known, I wouldn't have left*."

The surgery ended uneventfully. With the assistance of Dr. Eldridge, emergence from anesthesia went well. Now Dr. Maverick just needed to order postoperative pain medication.

6.1.8 Which Pain Medication Do You Suggest?

Tonsillectomy is a small but painful procedure. The sleepiness of the children in the PACU should not deter administration of pain relief. There are many non-opioid analgesics to choose from for pediatric patients [8, 9]. Following tonsillectomy, acetaminophen is most often prescribed.

6.1.8.1 Acetaminophen

Acetaminophen is an antipyretic and a pain reliever but lacks an anti-inflammatory component. In general, it is well tolerated; when given within the daily maximum dose and minding the contraindications, it hardly has any side effects. It is, however, not a harmless drug, since an overdose can cause severe liver damage and acute hepatic failure. Furthermore, if used during pregnancy or in the first year of life, it is suspected to increase the incidence of asthma in pediatric patients. However, due to overall convenience and lack of side effects in the acute setting, acetaminophen is commonly used to treat pain after tonsillectomy in children.

The recommended dosing interval is 6 h. In higher doses, the therapeutic period must also be determined. In pediatric patients under 3 months of age, the duration of therapy is limited to 48 h, otherwise 72 h.

Intravenous Administration

The dose is 15 mg/kg in pediatric patients >10 kg and 5 mg/kg in children <10 kg. The administration should be every 6 h, not to exceed the maximum dose of 60 mg/kg per day.

Oral/Rectal Administration

The oral dose of acetaminophen is 10–20 mg/kg every 6 h. The rectal dose is 20–40 m/kg. The

Table 6.2 Acetaminophen – age-dependent dosing

Age	Max. daily dose
Preschool and school-aged children	90 mg/kg body weight
Infants	75 mg/kg body weight
Term newborns	60 mg/kg body weight
Preterm neonate >34 gestational weeks	45 mg/kg body weight
Preterm neonate 30–34 gestational weeks	25 mg/kg body weight

maximum daily dose is dependent on age (Table 6.2).

6.1.8.2 Nonsteroidal Anti-inflammatory Drugs

For pediatric pain therapy, nonsteroidal anti-inflammatory drugs (NSAIDs) such as ketorolac or ibuprofen can be given for postoperative pain control. They have a higher analgesic potency than acetaminophen and are especially good for bony pain. However, there is concern that ketorolac may increase the risk of post-tonsillectomy bleeding due to inhibition of platelet function [8].

6.1.8.3 Opioids

Nowadays, opioids are seldom used for treatment of post-tonsillectomy pain. In the past, codeine was often prescribed. However, pain relief is similar with acetaminophen alone or alternated with ibuprofen compared to codeine plus acetaminophen [9]. In addition, the FDA recently issued a black box warning about a potentially fatal risk of codeine in children after tonsillectomy due to respiratory depression in some children [6]. Some children are ultrarapid metabolizers of codeine to its more potent form of morphine, which can lead to respiratory depression or arrest [4, 6]. The prevalence of ultrarapid metabolizers varies, mostly 1–4 % of children, but up to 29 % in the African/Ethiopian populations [6].

>> *Dr. Maverick carried sleeping Kevin into the PACU, where Kevin's mother was already anxiously awaiting his arrival. She thanked Dr. Maverick for the good care. She hadn't yet seen the various blue bruises from the missed IV attempts. Dr. Maverick thought*, "*if she only knew*."

6.2 Case Analysis/Debriefing

6.2.1 Which Medical Errors Do You See in the Presented Case?

6.2.1.1 Preoperative Evaluation

The preoperative evaluation was not thoroughly completed by Dr. Maverick; a bleeding history was not even taken prior to the tonsillectomy. The incorrect fasting order was already discussed in Sect. 6.1.4.

6.2.1.2 Venous Access

The venous access attempts should have been aborted sooner and delegated to the attending physician, Dr. Eldridge. In addition, IV access prior to induction of anesthesia was most likely unnecessary, and Dr. Maverick should have discussed the anesthesia plan in detail with Dr. Eldridge the night before surgery.

6.2.1.3 Laryngeal Mask Airway

Dr. Maverick was not familiar with laryngeal mask airways in pediatrics. He should have informed his attending, Dr. Eldridge, about his inexperience, and Dr. Eldridge should have paid more attention to the case and not left the OR at a critical point in the case.

6.2.1.4 Hand Ventilation After Ventilator Problems Arose

Many anesthesiologists are bagophiles. A commonly observed reflex after ventilation problems occur is to change to hand ventilation. Even though this can sometimes be helpful, it usually only brings higher ventilation pressures and higher tidal volumes. Modern ventilators can usually ventilate better than the human hand. Hand ventilation is also disadvantageous in that it takes mental and physical resources and

therefore may postpone problem solving. In the presented case, Dr. Maverick came to the differential diagnosis of bronchospasm. Auscultation would have been more helpful than bagging.

Hand ventilation is always indicated when ventilator or anesthesia machine malfunction is suspected; if the ventilator is functioning properly, the ventilator should be used.

6.2.2 Which Systems Failures Can Be Found in the Presented Case?

6.2.2.1 Supervision

Dr. Maverick had no supervision during the preoperative evaluation and he was inadequately supervised during surgery. Attending anesthesiologist, Dr. Eldridge, obviously, or perhaps hopefully, didn't know that Dr. Maverick was so inexperienced. The ACGME requires adequate supervision of residents for accreditation of residency programs. In addition, billing procedures require the presence of the attending anesthesiologist on induction, emergence, and all critical phases. Even more important is the ethical issue of leaving a patient's care in the hands of an inexperienced resident without adequate supervision. Do unto others as you would do unto yourself or your family!

6.2.2.2 Organizational Negligence

Attending anesthesiologist, Dr. Eldridge, assigned Dr. Maverick to the anesthesia (already a mistake!) and then neglected to provide supervision.

6.2.2.3 Negligence Due to Acceptance of Tasks

Dr. Maverick didn't disclose his lack of experience with his attending or anesthesia technician. He deliberately and intentionally committed negligence by accepting the task.

6.2.3 Why Didn't Dr. Maverick Disclose the Fact that He Had Lacked Experience in Pediatric Anesthesia?

Dr. Maverick wanted to look good in front of his team during his first pediatric anesthesia case, because he thought he would get more pediatric cases if he showed his flawless competence. He wanted to be a hero and portray himself as more qualified than he actually was. The excessive desire for acceptance added unnecessary pressure to perform and led to increased risk-taking behavior.

How should a team work with such a **perfectionist attitude**?

The solution strategy is easy; the implementation is difficult since numerous standards and values must be changed, some of which have been manifested through many years. The self-esteem of doctors and the reflected outside perceptions of the physician profession rely mainly on the expected perfect performance. Mistakes are not allowed. The solution therefore requires a strategy of establishing a culture of error. Working environments in which mistakes are openly discussed and the supervising physicians perform as a role model counteract a perfectionist attitude. Mistakes should not be sanctioned, but constructively discussed. Every team member should know the departmental expectations together with the norms and values which are bound to those expectations.

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Case 7: The Rebleed: Uh-Oh, a Child!

7

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7.1 Case Introduction: Continued from Case 6 (Chap. 6)

>> The remaining part of Dr. Maverick's day in the OR was uneventful. As the last elective surgeries were completed, attending anesthesiologist Dr. Eldridge excused himself to attend a meeting. Dr. Maverick left the OR, feeling bummed out about the sub-par anesthetic of the child earlier in the day. He had to preop several inpatients for ENT/head neck surgery for the next day. Thank goodness there were no children to see; he had had enough pediatrics for a while.

As he reviewed patient medical records in the nursing station, he heard a frantic scream: "We need a doctor in Room 10!" The nurse sitting across from him looked up at him with alarm – and together they ran to the room. "The tonsillectomy child who had surgery today is in Room 10," the nurse said, right before they reached the door. Upon entering, Dr. Maverick's worst fear came true. Kevin was in the bed: the pillow, the blanket, and everything covered in blood.

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The mother was talking to Kevin through her tears; he was unresponsive, but weakly spit blood, a mouthful of which landed on Dr. Maverick's knee, and Dr. Maverick's legs turned to jelly. "Why did I get up this morning?" he asked himself. The nurse positioned Kevin on his side with his head down, and started soaking up the blood around his mouth with gauze compresses. "Thank goodness you are here! Doctor, please help him!" begged Mrs. Miller, looking up at Dr. Maverick with faith and hope in her eyes.

7.1.1 What Should Dr. Maverick Do Now?

Obviously, Kevin has a severe post-tonsillectomy hemorrhage. Postoperative bleeding appears frequently after tonsillectomy, most often on day 1 and then about a week later after the scab comes off. Vital signs should be checked first, and then airway reflexes, to assess for the danger of blood aspiration. The child needs to be taken back to the OR as soon as possible for control of bleeding. Dr. Maverick can't get control of the situation alone – he needs to organize help. He must call:

- His attending physician
- The OR head nurse
- The ENT surgeons

Dr. Maverick should personally accompany Kevin into the OR and delegate the telephoning tasks to the nursing team.

>> Dr. Maverick tried to appear professional in front of Kevin's mom, but inside he was a nervous wreck. He had never been in such a situation. Would this day never end? The ENT nurse returned from telephoning and informed him that all parties had been notified. "Your attending Dr. Emery said to tell you to bring the patient directly into the OR; he is waiting there. And he said to hang a bag of IV fluid immediately."

7.1.2 Exactly What IV "Bag" Should Dr. Maverick Hang?

In pediatrics, as with adults, blood loss up to the maximum loss tolerable can be replaced with either crystalloid or colloid solutions (see Sect. 4.1.11). Healthy pediatric patients can tolerate a low hematocrit rather well, so a return to normal volume status is first priority. Evidence concerning use of crystalloid vs. colloid solutions is controversial [8]. While crystalloid solutions leave the intravascular space fairly quickly, they are generally used to initially replace blood loss in the emergency setting. Electrolyte solutions with sodium content equal to that of plasma should be given in boluses of 10 ml/kg titrated to improvement in hemodynamics. Lactated Ringer's solution is also acceptable, although it has lower sodium content than plasma. Large quantities are contraindicated in traumatic brain injury with potential for increased ICP. Note that glucose solutions with lower sodium concentration than in plasma (e.g., D5/W, D5/0.25 NS, D5/0.5NS) are converted to hypotonic solutions, after the glucose has been metabolized (hyperglycemia danger), and may cause dangerous hyponatremia, which can lead to cerebral edema. These types of solutions are contraindicated in treatment of hypovolemia.

Alternately, hydroxyethyl starch (Hespan) can be given if the hemorrhage isn't severe and there is no concern over coagulopathy or increased membrane permeability. Five percent albumin can also be used to replace intravascular volume. The advantage of a colloid solution is that a smaller volume is needed to restore intravascular volume; there is a prolonged increase in plasma volume and less peripheral edema. Concern is greater cost (especially with albumin), coagulopathy with hydroxyethyl starch at higher volumes, and pulmonary edema in capillary leak states, such as with sepsis, trauma, or shock. In the presented case, either crystalloid or colloid would be fine, but first choice is crystalloids (see also Sect. 21.1.3).

Hypertonic solutions are generally not given to restore intravascular volume after hemorrhage. An exception to this rule is the specialized case of fluid resuscitation in patients with or at risk for increased intracranial pressure [5].

>> As far as Dr. Maverick could tell, Kevin was spitting out all his blood. The little guy was coughing, so his airway reflexes seemed to be intact. Dr. Maverick asked the ENT nurse to prepare a

PlasmaLyte infusion. To the mom he said, "Kevin has bleeding after his surgery. We need to get him into the OR as soon as possible." "Everything will be OK, won't it?" pleaded Kevin's mom, "Please do the anesthesia yourself!"

Dr. Maverick wanted to give 20 ml/kg of PlasmaLyte, which was 400 ml for Kevin's 20 kg. He started the drip and set off with Kevin for the OR. Anesthesia technician Bruce was ready and waiting, but attending anesthesiologist Dr. Emery was not yet there. Dr. Maverick changed into scrubs quickly. As he entered the OR, Bruce already had Kevin hooked up to the monitors. Kevin's heart rate was 160 beats/min, blood pressure 95/64 mmHg, and he was tachypneic. "We've got to get a new IV line in," said Tech Bruce, "the last one got pulled out during the transfer." "Fantastic," thought Dr. Maverick, "this is going to be fun. At least Kevin's blood pressure is still OK." About 300 ml PlasmaLyte was already infused.

7.1.3 Can You Be Sure That the Patient Is Stable?

In this case, Kevin's condition has only been assessed clinically.

His blood pressure is still holding up well, but his heart rate is significantly above normal (Table 7.1).

Tachypnea can be a sign of compensation for early shock. The assumption that a satisfactory blood pressure alone equals cardiovascular stability can be a fatal assumption in children, and this delays the diagnosis of pediatric shock. Due to increases of systemic vascular resistance (SVR) and heart rate, blood loss can be well compensated for a long time in pediatric patients. Then, hemodynamic decompensation occurs suddenly. Small children can maintain their cardiac output almost completely from an increase in heart rate, because stroke volume and contractility are limited due to the small amount of contracting components. Blood pressure may be sustained in children until hypovolemia is extremely severe.

Table 7.1 Hemodynamic values in pediatrics

Age	Systolic blood pressure [mmHg]	Diastolic blood pressure [mmHg]	Heart rate [min^{-1}]
Newborns	65–80	40–50	130 ± 20
1 month	85	60	120 ± 20
1 year	95	65	120 ± 20
2 years	95	65	110 ± 20
4 years	95	65	100 ± 20
6 years	100	60	100 ± 20

>> Dr. Maverick looked for a vein. Apart from the bruises from the many missed attempts that morning, there were no veins to be found. Kevin's heart rate increased to 180 beats/min, the saturation hovered at about 90 %, and the blood pressure was down to 72/45 mmHg. Kevin was uneasy, fearful, and pale. Tech Bruce held an oxygen mask over Kevin's face, and he seemed to calm down a bit.

7.1.4 What Is the Cause of the Uneasiness? How Much Blood Do You Think He Has Lost?

The patient's uneasiness can be a result of beginning cerebral hypoxia, caused by hypotension and anemia. The increase of the inspiratory oxygen concentration via the face mask increased the dissolved oxygen (see Sect. 4.1.6) and might have improved cerebral hypoxia. Of course, pain, fear, or the taste of blood could also be a cause of the uneasiness.

Children of Kevin's age have a blood volume of 80 ml/kg body weight. Therefore, the total blood volume is 1,600 ml. A blood pressure decrease of >25 % with symptoms of shock indicates a blood loss of >20 % of the blood volume. Therefore, the blood loss is >300 ml.

>> Dr. Maverick was totally stressed out. He could only find one vein on the back of Kevin's foot, but he didn't trust himself to puncture the last possible option. Where was his attending? Why didn't someone come? Why couldn't he just have one functioning IV?

At that moment he heard the voice of the attending anesthesiologist, Dr. Emery, as he conversed with the ENT surgeon and approached

the OR. Dr. Emery recognized the severity of the situation at first glance. "Why didn't you call me sooner? I had no idea the child was in critical condition!" Dr. Emery was quite upset and cried out, "He doesn't even have decent venous access!" Sheepishly, Dr. Maverick showed his attending the last vein. Dr. Emery complained about the many ruined veins which had been blown that morning. He was given his requested IV catheter, but even his attempt to puncture the vein on the back of the foot failed. The next blood pressure reading was 60/30 mmHg; the heart rate remained at 180 beats/min. They needed venous access stat.

7.1.5 What Options Do You Have for IV Access?

Obviously, Kevin now has a life-threatening hypovolemia, and immediate volume replacement is necessary. Since peripheral access is no longer possible, there are two possibilities:

- Insert a central venous catheter.
- Obtain intraosseous access.

Placement of a central venous catheter in a child is not easy, is especially challenging for the inexperienced, and is a time-consuming process. Intraosseous access is the best option in this case.

7.1.5.1 Intraosseous Access

- All necessary infusions, medications, and blood products can be administered via an intraosseous line.
- The rate of flow is equivalent to that of intravenous access.
- It is even possible to draw a blood sample.

In the current guidelines of the Pediatric Advanced Life Support, intraosseous line placement is indicated after cardiac arrest if venous access is not immediately obtained [1, 4].

A recent article promoted the use of intraosseous access in the event of difficult IV access in pediatric anesthesia [9]. Following these guidelines, three indications for use are outlined (Table 7.2).

Intraosseous puncture locations include the proximal tibia, a few centimeters distal to the tuberosity, the proximal femur, the humerus, the tibia's medial malleolus, or the anterior superior iliac spine on the iliac crest of the pelvis, with lower extremity locations most popular in children. The puncture location must be spaced safely away from the epiphyseal plate, and the direction of puncture must also point away from the growth plate.

Various systems are available, which are more or less equivalent [3]. In pediatric anesthesia, manual insertion systems are usually used. Apart from the manual insertion systems, there are intraosseous needles (Cook/Jamshidi), mechanical devices which use a spring-loaded gun (F.A.S.T. 1/B.I.G.), and a drill device (EZ-IO).

>> *The attending anesthesiologist, Dr. Emery, requested an intraosseous needle and promptly placed it right into the proximal tibia. Kevin*

Table 7.2 Indications for an intraosseous access in pediatric anesthesia [9]

	Definition	Signs
Emergent indications	Early or primary	Respiratory or circulatory arrest Critical hemodynamic instability Shock Severe hypovolemia
Urgent indications	Without delay, following unsuccessful venous puncture attempts	Urgent anesthesia induction in a non-fasting child Urgent anesthesia induction in a hemodynamically unstable child or a child with severe cardiovascular insufficiency
Semi-elective indications	After unsuccessful venous puncture attempts and after careful use–risk consideration	After anesthesia induction with face mask (if access is necessary) Required intravenous induction (e.g., disposition to malignant hyperthermia)

briefly moaned. Dr. Maverick was so relieved to see blood flow in the needle. "I didn't even know that we had such a thing in the OR," thought Dr. Maverick. Attending anesthesiologist, Dr. Emery, took a sample of blood and gave it to Dr. Maverick, ordering a hematocrit. Then he started a crystalloid infusion on the needle and began the anesthesia induction.

7.1.6 How Would You Induce Anesthesia?

Kevin has swallowed a lot of blood. Therefore, to prevent aspiration, rapid sequence induction with cricoid pressure and insertion of an endotracheal tube is indicated. Apart from that, Magill pliers and large lumen OP suction should be prepared in order to remove coagulated and fresh blood that may block the view of the larynx. Propofol/barbiturates and opioids can cause severe hypotension in this hypovolemic child; therefore, ketamine or etomidate should be given for induction.

>> Attending anesthesiologist Dr. Emery induced and intubated Kevin quickly and without incident. A small decrease in blood pressure (from the induction of anesthesia) was treated with phenylephrine, and the IV infusion stabilized the blood pressure. The surgeons began to stop the bleeding. Dr. Emery could now peacefully insert an IV into a vein on the elbow. Obviously pleased with himself, he asked Dr. Maverick, "All right, what do you think the hemoglobin will be?"

7.1.7 What Value Do You Expect?

Blood loss from a post-tonsillectomy hemorrhage is very difficult to estimate because the blood is not suctioned into a labeled container, but is swallowed and lost onto sheets, pillows, and towels. The initial lab value for hemoglobin/hematocrit could be almost normal, and lend no information whatsoever to the actual blood loss. This is because plasma and red blood cells are proportionately lost together.

>> The analysis of the blood sample showed hemoglobin of 10 g/dl and a hematocrit of 30 %. "All right, what do we do now?" asked the attending Dr. Emery. Dr. Maverick wasn't sure. Was a transfusion indicated with this finding?

7.1.8 Do These Values Warrant a Blood Transfusion?

The normal value for hemoglobin in children is between 11 and 13 g/dl. Therefore, Kevin is just below the normal level. However, the hemoglobin concentration will decrease after volume replacement therapy brings Kevin back to euvolemia. Since a tonsillectomy wound covers only a small area, and bleeding can be quickly halted by surgery, the blood transfusion can be postponed as long as hemodynamic stability is maintained. Just in case, a type and crossmatch for packed RBCs should be ordered and intravascular volume status must be carefully monitored! In other surgeries, such as extensive scoliosis corrections, these hemodynamic values would already warrant transfusion, if further intraoperative blood loss or rebleeding is expected.

>> After consulting with his attending Dr. Emery, Dr. Maverick called the lab to order some blood. In addition, Dr. Emery asked Dr. Maverick to order "some coagulation studies." As Kevin stabilized, Dr. Emery left the OR with these words: "You'll be able to finish this all right, won't you? I have to start another case." The surgeons completed the procedure with these words, "Well, the wound isn't completely dry. Under no circumstances should the child cough!" With the good IV access that Kevin now had, the lab work shouldn't be a problem, but emergence from anesthesia without coughing?

7.1.9 What Is the Best Plan for Emergence from Anesthesia?

Even though the surgeons may desire a peaceful emergence, prevention of aspiration takes first

priority. Therefore, extubation will occur after return of protective reflexes.

Suctioning of the throat is not advised. The gurney can be positioned with the head down, and the child can be leaned to the side. Antiemetic intraoperative prophylaxis with dexamethasone 150 µg/kg and ondansetron 0.15 mg/kg is advised. Lidocaine 0.5–2 mg/kg IV may be given to reduce coughing prior to emergence.

>> Dr. Maverick was satisfied with himself. Extubation and anesthesia recovery went well. Kevin was extubated with only a very small cough, and Kevin was breathing spontaneously and had visible swallowing motions. Kevin breathed with a rasping sound, but tech Bruce reassured Dr. Maverick: "That's just because of bloody secretions in the pharynx." Kevin was transferred to the PACU.

Dr. Maverick finished the anesthetic record and PACU orders. Then he went to the bed to check on Kevin. The rasping was audible from 10 ft away; the S_pO_2 was a shocking 92 % with oxygen administration.

7.1.10 What Is Kevin's Problem?

A common postoperative complication is laryngeal and pharyngeal edema. After all, the problem arises from irritation of the pharynx and larynx by repeated suctioning and manipulation. Clinically, stridor and respiratory exhaustion are seen due to the increased mechanical work of breathing. In order to prevent reintubation, nebulized racemic epinephrine should be administered immediately. In addition, in severe cases, prednisolone at 3 mg/kg can be given IV or as a suppository.

>> As the anesthesia nurse in the PACU prepared the nebulizer, the lab called with the coagulation studies:

- *PT: 10.1 s (reference 9.5–13.5 s)*
- *aPTT: 45 s (reference 20–40 s; laboratory dependent)*
- *Platelets: 180,000/mcl; (reference 150–400,000 /mcl)*
- *Hemoglobin: 8.7 g/dl; (reference 12–14 g/dl)*
- *Hematocrit: 28 %; (reference 37–47 %)*

Dr. Maverick wondered about the prolonged aPTT. The blood loss hadn't been that extensive, and the other coagulation values were within the normal range. He informed the ENT surgeon of the results, who was just in the process of rechecking the wound for bleeding. "Well, you better do something about it, there's still diffuse bleeding going on," responded the ENT surgeon.

7.1.11 What Should Dr. Maverick Do with These Values?

The mystery is the isolated prolongation of the aPTT together with the otherwise normal coagulation values. Clinically, Kevin began to bleed diffusely a few hours after the first surgery. Even after the second surgery, the bleeding has not yet ceased. The combination of the lab results and clinical presentation leads to the suspicion of a genetic coagulation disorder, 95 % of which are due to a deficiency of factor VIII (hemophilia A), factor IX (hemophilia B), and von Willebrand disease. Hemophilia A, with an incidence of 1:10,000, is 10 times more common in boys than hemophilia B. Both are X-linked recessive inherited traits. Von Willebrand disease (vWD) is either autosomal dominant (type 1) or recessive (type 2 and type 3), whereas type 1 is the most common, accounting for 60–80 %. The vWD does not have complete penetrance and is the most common genetic coagulopathy disorder, with an incidence of with 0.5–1 %. It is only overtly symptomatic in about 10 % of those afflicted.

Since in-depth coagulopathy tests cannot be carried out in an acute situation, the likelihood of the various disorders must be taken into consideration. In addition, the parents need to be carefully questioned about everyday bleeding in the patient and problematic bleeding in family members [2]. A positive family history is not always present, however, since about 30 % of hemophilia A appears as a new mutation. Less serious forms

with a remaining factor activity of 5–20 % can exist without being detected clinically: normal as a newborn or child with uneventful vaccination and blood draws. The first hints of the disease are seen after large trauma or after smaller surgeries, such as tooth extraction or tonsillectomy.

>> Before Dr. Maverick informed his attending about the coagulation results, he questioned the mother once more. She had sat down on the bed and was holding Kevin's hand when she replied, "I can't think of any excessive bleeding he has had. Oh, one time he fell from the shopping cart and got a big blue bruise, but it was a big fall. I don't know of anyone in my family or my husband's family who has had problems with bleeding. Before Dr. Maverick knew it, the mom had taken hold of his hand and said, "I am forever grateful to you for saving my son's life!" Taken aback, Dr. Maverick mumbled, "It's all part of my job, Ma'am."

Then he called his attending Dr. Emery to discuss possible treatments. Dr. Emery thought about it a moment and asked, "And he's still bleeding?" Then he recommended administration of 0.3 µg/kg of desmopressin IV over 20 min.

7.1.12 What Was His Recommended Therapy?

Von Willebrand disease (vWD) is the most common disorder in a group of hemorrhagic diathesis. In vWD, there is a qualitative or quantitative defect of the glycoprotein called von Willebrand factor (vWF). The vWF is produced in the endothelial cells of the vessels. After vessel damage, the vWF binds to exposed collagen and activates platelets of a damaged vessel to build a clot.

Administration of desmopressin releases vWF from endothelium cells, increasing the concentration up to fivefold [7]. In plasma, vWF associates with factor VIII and protects it from enzymatic degradation. This molecule complex plays an important role in the platelet activation in primary hemostasis and as a cofactor for the activation of factor X to factor Xa in the plasma coagulation.

With postoperative diffuse bleeding, treatment should be directed toward the most common coagulation disorder, vWD type 1, which has decreased levels of vWF [6]. Even in mild forms of hemophilia A (with <10 % active clotting factor remaining), desmopressin can enhance hemostasis. It can be administrated in 8-h intervals, for as long as the tendency to bleed remains; however, it loses its effectiveness after a few days.

>> Just as his attending, Dr. Emery, recommended, Dr. Maverick administered 0.3 µg/kg body weight of desmopressin IV over 20 min. The desired effect was achieved, and the bleeding stopped. Kevin finally fell asleep, without stridor, on his fresh clean pillow. His mother's relief was felt by all. Dr. Maverick was relieved as well and explained to her that he suspected a coagulopathy. "We'll ask a hematologist to further evaluate Kevin." Dr. Maverick ordered the transfer to an intermediate care station and then left the hospital, exhausted but happy. He wouldn't soon forget the lessons he had learned today.

7.2 Case Analysis/Debriefing

7.2.1 Which Medical Errors Do You See in the Presented Case?

Medical errors associated with the first anesthetic (see Case 6; Chap. 6) had serious consequences for the resulting chain of events. First to be noted were the many unsuccessful IV attempts.

7.2.1.1 Patient History: Bleeding

The patient/family history for bleeding disorders was not obtained before the first anesthetic. At the latest, this history should have been taken when the postoperative hemorrhage occurred.

7.2.1.2 Venous Access

The loss of the venous access while moving to the operating room table is an unacceptable disaster.

7.2.2 Which Systems Failures Can Be Found in the Presented Case?

7.2.2.1 Algorithm for Emergency Venous Access

Dr. Maverick didn't know that an intraosseous kit was in the OR for use in emergencies.

Securing venous access quickly can be a life or death matter. It is essential to maintain the necessary equipment. Also, all team members must be made aware of the accessibility of the equipment.

Just as in an emergency cricothyrotomy, one must overcome a psychological threshold of resistance, before carrying out the insertion of the intraosseous system. Therefore, a specific guideline is important for such situations, and all team members must be made aware of it and better yet be trained in it via simulation.

7.2.3 Step Back! You Are a Doctor!

Kevin's ever-worsening vital signs and failed venous access attempts made Dr. Maverick totally stressed out. He focused his concentration on Kevin's life-threatening situation. He was unable to think of alternative venous access possibilities – his limited attention was exceeded by the demands. What can be done during such instances of "**attention fixation**"?

In a time-sensitive, complicated emergency situation, imagine yourself outside the situation (a mental "step back") and calmly review the situation, just taking a moment to reanalyze. What can you do to quickly secure venous access in this emergency situation?

First of all, you need to practice a relaxing ritual or behavior so that it can be employed under stress. This ritual or behavior should be simple and inconspicuous, so that no one present notices it. Examples include breathe deeply three times, clear your throat, take one step back, or make a fist. Once you find a ritual/behavior, practice it regularly in your daily work. Then it will be easy to use under stressful and emergency situations.

7.2.3.1 Practice a Mental "Step Back"

Think of an inconspicuous ritual or behavior (e.g., breathe deeply three times, clear your throat, take one step back, or make a fist). During your next routine situation, for example, face mask ventilation, imagine that there is an emergency or a complication, such as the patient vomits or the intubation is difficult. Use your ritual/behavior to take a mental "step back" and then decide upon a strategy to handle the situation. If you then repeat this technique often enough, you will discover that the "step back" is also reproducible in a real crisis. That is a small step for the anesthesiologist and a giant leap for the patient!

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Case 8: Bowel Obstruction

8

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8.1 Case Introduction

>> Before I discuss my personal experience, let me tell you a little about myself. My name is Sharon Collins; I am 45 years old and 5′3″ tall and weigh 56 kg. For the past 10 years, I've worked as an anesthesiologist at a small suburban hospital. We do about 6,500 anesthetics for procedures per year. I am happy with my job; I enjoy it very much. But that's enough about my job. Here's my story:

Five months ago, I developed severe abdominal cramping which came over me in waves. I was lying at home on the sofa with a hot water bottle on my tummy and I didn't want to move. Ibuprofen had helped in the past, but this time the pain was so bad that I couldn't go to work. After about a day and a half, I couldn't even drink a cup of tea because of nausea and vomiting. I didn't have a fever. When my husband came home from work that evening, he

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was notably shocked at the way I looked. I knew it was time to go to an emergency room (ER). Somehow he got me in the car and drove me to a bigger hospital, which was a little further away. I had, understandably, declined the offer to be taken to my own hospital. I was worried about our son John, but my husband said I should just focus on getting myself well; the two of them would be fine.

In the ER, I was seen quickly by the attending physician, Dr. Emerson. She greeted me with a handshake "Hello, so nice of you to have chosen our hospital; I understand you have a fine one of your own. What's going on?" I described my symptoms and mentioned my acute appendicitis 5 years ago. I had to have an open appendectomy; it was a severe infection with local peritonitis. Dr. Emerson examined me – my abdomen was tense and hard, every touch extremely painful. Even an anesthesiologist could make the diagnosis: small bowel obstruction. She ordered some blood tests.

The surgeon ordered a CT of the abdomen, just to be sure. "Classic presentation," commented the radiologist to Dr. Emerson, as I was caught in the middle of a new wave of nausea, vomiting, and debilitating cramps. "We need to operate now," said Dr. Emerson, turning to me. "I'd say you've got a complete small bowel obstruction." She then called Dr. Eldridge, the attending, "Listen, I've got a colleague of yours here with a complete bowel obstruction. We need to operate as soon as possible." Then she turned to me to say, "Looks like we can't get you in for another 2 h. We will give you a room on the ward and wait for the results of the blood tests." The surgeon obtained my informed consent for the procedure – I couldn't have cared less at that moment.

Meanwhile, the attending anesthesiologist, Dr. Eldridge, came to see me in the ER. I told him about my previous surgery and that I have asthma, treated with Advair (a combination preparation of fluticasone and salmeterol), and that I take 2 puffs mornings and evenings. I never skip a dose; if I do, I quickly become dyspneic. As far as my family history is concerned, it was important for him to know that my 14-year-old son John has Duchenne's muscular dystrophy. He deteriorates year after year; he requires constant care. Nevertheless, we had our daily routine worked out well. Dr. Eldridge promised to personally administer my anesthesia. "Can I give you something now for pain?" he asked. I didn't want anything. Despite pain, I wanted to be able to observe the procedure from a patient's point of view.

I waited another 2 h on the ward until the OR was ready for me. I can't remember how I spent those 2 h; all I can remember is the pain.

I was brought to the OR at 8 PM. The attending anesthesiologist Dr. Eldridge was there, as promised. Looking at me, he furrowed his brows. I really didn't look good; I felt weak, dizzy, apathetic, and I was constantly nauseated. I had never felt like this in my life. After CRNA Ruth hooked up the ECG, blood pressure, and pulse oximetry, Dr. Eldridge placed the IV line. My blood pressure was 115/75 mmHg, my pulse was 110 beats/min, and S_PO_2 was 91 %. Dr. Eldridge reviewed my lab results before induction. Kindly CRNA Ruth held my hand.

Dr. Eldridge reviewed the following values:

- *Hb: 17 g/dl (reference 11.9–17.2 g/dl)*
- *HCT: 48 % (reference 37–47 %)*
- *WBC: 14,000 cells/mcl (reference 3,600–9,800 cells/mcl)*
- *Platelets: 350,000 (reference 150,000–400,000)*
- *PT: 10.3 s (reference 8.0–14.7 s)*
- *PTT: 38 s (reference 20–41 s)*
- *Fibrinogen: 5 g/l (reference 1.5–4.5 g/l)*
- *Na^+: 141 mEq/l (reference 136–145 mEq/l)*
- *K^+: 2.7 mEq/l (reference 3.8–5.2 mEq/l)*
- *ALAT (GPT): 10 IU/l (reference <17 IU/l)*
- *ASAT (GOT): 8 IU/l (reference <15 IU/l)*
- *Bilirubin (total): 0.8 mg/dl (reference <1.0 mg/dl)*
- *Creatinine: 0.78 mg/dl (reference 0.68–1.17 mg/dl)*
- *BUN: 28.8 mg/dl (reference 18.0–55.3 mg/dl)*
- *Total protein: 6.5 mg/dl (reference 6–8.4 mg/dl)*
- *Glucose: 95 mg/dl (reference 70–125 mg/dl)*

"All right," said Dr. Eldridge, "we'll get you off to sleep now." Turning to me he said I should close my eyes, think of something pleasant, like being on a warm beach, and not worry. After that, I didn't remember much of anything.

8.1.1 Which Form of Anesthesia Would You Choose and Why?

General anesthesia is indicated due to the planned intra-abdominal surgery. To be on the safe side, trigger-free anesthesia might be advisable with a family history of muscular dystrophy. Trigger-free means that all medications, which could trigger malignant hyperthermia, must be avoided. This includes inhaled anesthetics, with the exception of nitrous oxide, and depolarizing muscle relaxants (succinylcholine). Safe medications include benzodiazepines, barbiturates, local anesthetics, propofol, non-depolarizing muscle relaxants, opioids, and ketamine.

The incidence of malignant hyperthermia is drastically different in various geographic and ethnic populations. The incidence of malignant hyperthermia in the USA is 1:100,000 in adults and 1:30,000 in pediatric patients, with an estimated prevalence of the genetic predisposition at 1:2,000 [10]. The incidence of malignant hyperthermia is higher in Wisconsin, Michigan, West Virginia, and Nebraska [10]. Malignant hyperthermia appears in all races and in both genders; however, there is a suspected predominance in men and pediatric patients. Two diseases are linked to malignant hyperthermia, the autosomal dominant inherited central core disease (also known as central core myopathy) and the Australian King-Denborough syndrome. The defect in calcium homeostasis leads to accumulation of calcium in the sarcolemma that causes sustained contractures of skeletal muscles.

Neuromuscular dystrophies (such as Duchenne, Becker, or Emery-Dreifuss) do not have a higher risk of malignant hyperthermia compared to the general population. However, the fetal and adult acetylcholine receptors are upregulated in these disorders. These patients are therefore susceptible to massive calcium release. Especially important, succinylcholine and inhaled anesthetics may cause rhabdomyolysis and life-threatening hyperkalemia. This is the reason why patients with neuromuscular diseases are anesthetized as patients with malignant hyperthermia [7, 9]. It is unclear if a positive family history as in the presented case is also an indication for trigger-free anesthesia, but since this type of anesthesia is not risky at all, many anesthesiologists choose this technique.

Combined epidural and general anesthesia might be beneficial for post-op pain control and early postoperative ambulation; however, it is often not used in emergency procedures associated with hypovolemia. Preoperative informed consent, with discussion of risks/benefits of regional anesthesia, would also have needed to be obtained.

8.1.2 What Must You Watch Out for During Induction?

Due to the small bowel obstruction with nausea and vomiting, and urgency of surgery, the risk of aspiration of gastric (or bowel) content during the induction of anesthesia is increased. The incidence of aspiration during elective procedures is 1:3,000–4,000, compared to emergency procedures 1:600–900 [17]. Therefore, in emergency procedures rapid sequence induction (RSI) with cricoid pressure is recommended. The goal of RSI is first to reach a deep enough anesthesia for intubation and also to minimize the time that the muscles are relaxed before the airway is secured, because this time period has a high risk of regurgitation.

The efficacy of RSI with cricoid pressure in reducing aspiration is unknown as evidence-based studies are lacking [12]. Nevertheless, the standard of care is that a RSI is recommended in every anesthesia where there is increased risk of aspiration. However, every step of the RSI should be evaluated for practicality and possible side effects.

8.1.3 Name Other Indications for RSI and Discuss the Procedure!

Risk of aspiration is increased when intra-abdominal or intragastric pressure is elevated or

the esophageal pressure is lowered. On the other hand, loss of protective reflexes due to neurological diseases (e.g., increased intracranial pressure) is another risk factor for aspiration.

8.1.3.1 Indications for a Rapid Sequence Induction (RSI)

Emergency patients:

- Non-fasting patients
- Trauma patients
- Patients with intra-abdominal disease processes

Gastrointestinal (GI) reasons:

- Bowel obstruction
- Upper GI bleeding
- Gastric atony
- Gastric emptying disorder
- Gastroesophageal reflux disease, with symptomatic reflux
- Reflux esophagitis
- Esophageal diverticulum
- Esophageal atresia
- Pyloric stenosis
- Pregnant patients in the second and third trimesters

Patients with decreased level of consciousness:

- Comatose patients
- Drug-intoxicated patients
- Alcohol-intoxicated patients
- Patients with hyperuricemia
- Patients with increased intracranial pressure

The following procedure is often chosen:

8.1.3.2 Gastric Emptying

Preoperative placement of a nasogastric (NG) tube is often considered in patients with bowel obstruction, peritonitis, or UGI bleeding. Known esophageal varices are a relative contraindication. Regularly monitor the flow since food clumps can block the tube. Most anesthesiologists leave the NG tube in place during induction of anesthesia. However, because a NG tube can function as a path of least resistance for regurgitation, some anesthesiologists recommend removing it prior to induction of anesthesia. All things considered, the forced gastric emptying before a planned RSI is highly debated. The placement of a gastric tube is usually very uncomfortable, and the effectiveness is questionable. Clinical trials are lacking.

Sometimes, especially with pregnant women, the stomach acid will be buffered with sodium citrate shortly before the induction of anesthesia. Clinical trials are also lacking for this topic. Sodium citrate increases the volume of gastric content and has a bad taste (disbelievers should try it once), and the patients often feel nauseated. It should not be administered in the presence of a small bowel obstruction.

8.1.3.3 Anesthesia Induction

- Consider positioning the patient in a head-up position – especially important for the morbidly obese patient.
- Secure good IV access.
- Preoxygenate sufficiently, at least 3 min with a F_iO_2 of 1.0 (see Sect. 25.1.5)
- Have suction ready.
- Intravenous drugs including muscle relaxants must be given in rapid succession.
- Avoid mask ventilation before intubation unless the patient is hypoxemic; quickly intubate in 60–90 s when neuromuscular blockade is complete.

Note:

The reverse Trendelenburg position of the patient prolongs the "safe apnea" phase after preoxygenation, especially in morbidly obese patients. The safe apnea period is the time before reaching critical levels of desaturation [5]; thus, this position should be considered not only for possible aspiration prophylaxis.

A general recommendation for RSI is to give the hypnotic and muscle relaxants as quickly as possible. Likewise, one must expect to encounter hemodynamic side effects, such as hypotension from the hypnotic and hypertension/tachycardia with endotracheal intubation. There is no study that weighs the risk of aspiration during the titrated prolonged medication injection, against the hemodynamic instability following rapid injection. Therefore, the speed of injection of each medication should be individually assessed and dosed accordingly [12].

In addition to intravenous anesthetic agents and muscle relaxants, there are other medications recommended to suppress autonomic reflexes during the intubation and to reduce the doses and side effects of RSI. These adjuvant medications include opioids, lidocaine, and esmolol. Fentanyl provides good hemodynamic stability after intubation [15]. Lidocaine, in comparison to esmolol, does not significantly reduce the hypertensive response to intubation. Lidocaine, however, more effectively suppresses cough and may reduce bronchospasm in an asthmatic patient. Both lidocaine and fentanyl reduce pain upon injection with propofol.

The use of cricoid pressure (CP) during the RSI is based on physiological and cadaver studies, anecdotal evidence, and expert opinions. Randomized controlled studies about the effectiveness of the airway protection from CP are absent. While there is debate over risk/benefits, CP is still standard care in a RSI in the patient with a full stomach, such as a small bowel obstruction or after a large meal. CP is also useful in a modified RSI when mask ventilation is necessary, such as with hypoxemia or increased ICP. The CP reduces the possibility of gastric inflation during mask ventilation. One must watch out for possible airway obstruction with difficult intubation circumstances due to the CP [12]. Always keep in mind to release CP in case of active vomiting because there is an increased risk of esophageal rupture.

In very young pediatric patients, mask ventilation is generally recommended during a RSI in order to avoid hypoxemia. An inspiration pressure no higher than 15–20 cm H_2O minimizes the risk of stomach insufflation [12].

8.1.4 How Would You Proceed in the Case of Dr. Collins, the Patient?

After placement of monitors, Dr. Collins would be preoxygenated from a tightly held face mask with 100 % oxygen.

An ideal intravenous anesthetic agent should offer a rapid onset of action, depression of sympathetic reaction during intubation, analgesia, dependable anesthetic and amnestic properties, assistance for laryngoscopy in case of an incomplete muscle relaxation, and, above all, hemodynamic stability and minimal side effects. Such a medication does not (yet) exist. In principle, the following agents can be considered for use:

- Propofol
- Etomidate
- Ketamine
- Benzodiazepine

Keep in mind individual patient characteristics and possible side effects or inadequate effects.

Most anesthesiologists prefer propofol for RSI, especially in combination with non-depolarizing muscle relaxants, since propofol suppresses the reflexes in the airway especially important in an asthmatic patient and it may increase intubation success [16]. The classic anesthetic for RSI is thiopental, which is no longer available in the USA. Etomidate or ketamine may be chosen if the patient is hypovolemic or has significant myocardial dysfunction. Ketamine reduces reflex-induced bronchoconstriction and may also be useful for induction in patients with asthma or chronic obstructive pulmonary disease.

For the muscle relaxation required for RSI, there are two drugs currently in use:

- Succinylcholine
- Rocuronium

Succinylcholine, administered at ≥1 mg/kg, assures excellent conditions for intubation and is the drug of choice when no contraindications are present [12]. Rocuronium, administered at 0.9–1.2 mg/kg, would obtain almost the same intubating conditions [14]. Due to her son's muscular dystrophy, succinylcholine is contraindicated for Dr. Collins due to the risk of hyperkalemia.

>> *I came back to consciousness in the ICU, sometime during the night or early morning hours of the next day. As it became bright outside, I felt better and could concentrate a little on other things. There were strange memories in my head, so I was very curious to see the anesthesia*

and surgical records. The nurses were kind enough to allow me to see them, so I began to read:

After preoxygenation, I received 200 µg fentanyl, 150 mg propofol, and 100 mg rocuronium IV.

Intubation was uneventful; there was no suspicion of aspiration. Post-intubation hypotension of 85/50 mmHg was treated with phenylephrine. The anesthesia was then continued as…

8.1.5 Which Medication Would You Use to Maintain Anesthesia? Which Would You Avoid and Why?

Volatile anesthetics were avoided due to the predisposition for hyperkalemia. Hence, TIVA with fentanyl and continuous propofol infusion was used.

Dr. Collins was ventilated with a mixture of 50 % oxygen and air. This is because nitrous oxide is contraindicated in the presence of a bowel obstruction. Nitrous oxide diffuses very easily through the air-filled spaces, because it is 35 times more soluble in blood than nitrogen. Since nitrous oxide diffuses quickly into empty spaces, faster than nitrogen diffuses out, expandable cavities such as intestines will increase in volume, and in nonexpandable cavities such as the inner ear, pressure will increase. Usually nitrous oxide diffusion into the intestines is inconsequential, since only small amounts of air are contained within the intestines. When obstruction is present, however, the volume increase can make the surgery significantly more difficult, and the bloated intestines can hinder breathing during the postoperative phase.

>> And that's how it went. After induction, I received a propofol infusion running at a rate of 80 µg/kg/min. At incision, the rate was increased to 125 µg/kg/min. In addition, shortly before incision, I was given fentanyl 300 µg. I remained hemodynamically stable. The operation only took 50 min. From the records, I confirmed that a small bowel obstruction was the cause of my symptoms. Thank goodness, it was only necessary to lyse the adhesions. Then why am I in the ICU? Why wasn't I extubated at the end of the operation? The record showed that at the end of the procedure I didn't have any twitches present on my train of four (TOF). The reason must have been a muscle relaxant overdose.

8.1.6 Why Was Dr. Collins Left with a Postoperative Residual Neuromuscular Blockade, and Why Wasn't It Reversed?

Dr. Collins received during the course of the RSI twice the standard amount of rocuronium, as compared to a normal anesthesia induction. In such high doses, rocuronium takes effect after about 1 min. However, the duration of neuromuscular blockade can be profoundly increased.

The main location of action of the nondepolarizing muscle relaxants is the acetylcholine receptor of the motor end plates. Indirect antagonism of neuromuscular blockade occurs by inhibiting pseudocholinesterase, so that the concentration of acetylcholine increases. This leads to a competitive inhibition of the muscle relaxant on the receptor, so that the effect is decreased. Typical cholinesterase inhibitors/parasympathomimetics are neostigmine, pyridostigmine, and edrophonium. Before reversal of neuromuscular blockade, spontaneous recovery of 25 % of the neuromuscular impulse transmission should be present. This correlates to three to four twitches on the TOF. Since Dr. Collins lacked twitches on her TOF, there was ample justification for not reversing the blockade. After administration of cholinesterase inhibitors, the acetylcholine concentration will be increased not only on the nicotinic but also on the muscarinic receptors. The result is typical side effects such as:

- Bradycardia
- Increasing salivary and bronchial secretions
- Increased intestinal mobility
- Bronchial constriction

- Miosis
- Contraction of the urinary bladder
- Nausea and vomiting
- In myasthenia gravis, triggering a cholinergic crisis

To minimize the side effects, neostigmine or edrophonium is always given with glycopyrrolate or atropine, respectively. As complete inhibition of muscarinic effects can't be guaranteed, asthma, bradyarrhythmias, and AV blocks are relative contraindications to reversal of neuromuscular blockade. However, these adverse effects can be avoided if the anticholinergic agent is administered first. Since Dr. Collins had a deep level of neuromuscular blockade and she has asthma, reversal of the block was not done.

Sugammadex is an alternative for the reversal of rocuronium. While approved in Europe, it has not yet been approved by the FDA for use in the USA due to concern over hypersensitivity reactions, including severe bronchospasm. Sugammadex is a modified y-cyclodextrin, which has a low water solubility and builds 1:1 complexes with steroid muscle relaxants. The elimination of these complexes occurs renally or via dialysis. Recurarization is very unusual. The affinity of y-cyclodextrin is strongest for rocuronium, followed by vecuronium and pancuronium. It also works in a deep muscle block from rocuronium and was successfully used in a case of severe anaphylaxis following rocuronium administration. Sugammadex does not work on any other receptor system of the body and does not have any known side effects. A disadvantage is the high cost for the drug.

>> Near the end of the surgery, an arterial blood gas analysis was done, which showed the following:

- *Hb: 10.2 g/dl (reference 11.9–17.2 g/dl)*
- *HCT: 31 % (reference 37–47 %)*
- *Na^+: 136 mEq/l (reference 136–145 mEq/l)*
- *K^+ 3.0 mEq/l (reference 3.8–5.2 mEq/l)*
- *pH value: 7.33 (reference 7.35–7.45)*
- *P_aCO_2: 41 mmHg (reference 36–44 mmHg)*
- *P_aO_2: 105 mmHg (reference 70–100 mmHg)*
- *BE: –4.8 mEq/l (reference ±2 mEq/l)*
- *HCO^{3-}: 20.4 mmol/l (reference 22–26 mmol/l)*
- *S_PO_2: 95 % (reference 95–98 %)*

I had received fentanyl 200 µg and 5 mg midazolam and then I slept a little in the ICU. During the night I was weaned and extubated. I had, however, a terribly bad dream in my memory after I woke up.

The next morning Dr. Eldridge came to the ICU to see me. I was waiting for his visit, and I feared that I was going to have to tell him something very difficult. "Good morning to my dear colleague, Dr. Collins," he greeted me warmly. "How are you? Did you sleep well?" I shook my head. "I can't complain about the anesthesia, but I wasn't completely asleep. I remember someone moving a large instrument around in my abdomen. Dr. Eldridge stared at me in shock.

"Were you in pain?" he asked.

"No, I didn't feel any pain, but it was a very uncomfortable feeling."

"Can you remember anything else?" he asked.

I said that I remembered a man standing beside me. I wanted to give him a sign, but I didn't have the strength to move. Then I fell asleep.

8.1.7 Which Anesthesia Complication Is the Patient Talking About?

Dr. Collins is describing awareness (explicit recall).

8.1.8 Name the Incidence of, the Causes for, and Risk Factors for Awareness!

The prevalence describes how common a characteristic is in the target group. Incidence describes the new occurrence of this characteristic. On average, taking general anesthesia as a whole, the incidence of undesired awareness with pain is 0.03 % and without pain is 0.1–0.2 %. However, awareness is more common in certain surgical procedures, and awareness incidence in different types of anesthesia varies immensely [1, 13].

Table 8.1 Prevalence of conscious memories from an awareness episode

Reported memory	Prevalence (%)
Hearing	85–100
Seeing	27–46
Fear	78–92
Helplessness	46
Operation details	64
Paralysis	60–89
Pain	39–42

Adapted from Daunderer and Schwender [4]

Coming to consciousness without remembering it and subconscious memories are even more common and are estimated to occur in up to 70 % of all cases [4]. Table 8.1 shows the prevalence of various memories in the case of awareness4.

The main causes of awareness are light anesthesia, increased anesthetic requirement, and equipment malfunction. In most cases, awareness arises in patients who have planned light anesthesia or when lower doses of anesthetic agents are required. These patients/surgeries include:

- General anesthesia for cesarean section
- Scoliosis operations with intraoperative wake-up testing
- Cardiac surgery patients, especially during cardiopulmonary bypass
- Bronchoscopy
- Patients with severe cardiac dysfunction
- Trauma surgery

Although light anesthesia is commonly manifest by hypertension and tachycardia, hemodynamics are usually unpredictable indicators of awareness. Reliance on observation of clinical signs fails as soon as autonomic reactions are masked by medications such as catecholamines, antihypertensives, parasympatholitics, etc. The pain response is masked by opioids and movement is prevented by muscle relaxants. Don't forget the increased anesthesia dose required in younger patients, patients with any history of drug abuse, and patients taking chronic opioids and benzodiazepines. Many episodes of awareness occur in hypotensive patients in whom anesthesia doses are reduced to avoid cardiovascular depression.

8.1.9 What Are the Psychological Sequelae of Awareness in Patients?

The psychological sequelae are dependent on the remembered experiences. They include, with increasing severity:

- Anxiety
- Sleeping disorders
- Nightmares
- Constant reliving of the intraoperative perceived events
- Death-like experience
- Avoiding situations which are similar to the intraoperative awareness: doctors, hospitals, and surgery
- Full-blown posttraumatic stress disorder (PTSD)

Minimal impairments are common, but some patients develop PTSD. The incidence of PTSD after an awareness episode is unclear and may be associated with more distress, pain, and near-death experience.

8.1.10 What Are Some Strategies to Prevent Awareness?

In surgical procedures with an increased risk of intraoperative awareness, increased vigilance is needed. In these patients, the possibility of awareness should be discussed preoperatively. In most patients without an increased risk, failing to bring up the possibility of awareness can be justified by the desire to avoid causing undue worry in countless patients.

8.1.10.1 Prophylaxis Against Awareness

The most important preventive strategy against awareness is possibly the pharmcological premedication with bezodiazepines [13]

Benzodiazepines are administered before induction of anesthesia for most elective procedures. However, benzodiazepines are often avoided in emergency surgery. Limiting muscle relaxants to the minimum necessary is recommended. The loss of consciousness must be maintained for as long as the patient is still paralyzed.

Measuring the degree of neuromuscular blockade with a TOF monitor is important to avoid complete neuromuscular blockade.

Furthermore, operating room discussions should be kept to a minimum, with conversations in quiet voices. Comments or insults about the patient are strictly inappropriate.

Anesthetics depress consciousness and unconscious perceptions in a dose-dependent manner. Therefore, high enough doses should be given, which is especially important for RSI patients [8]. In order to prevent awareness, the end-tidal anesthetic concentration of the volatile anesthetic needs to be 0.7 MAC or greater, adjusted for age [2]. MAC levels decrease with age. The monitoring of the end-tidal volume of anesthetic concentration only shows that the concentration is high enough, not necessarily guaranteeing a protective effect against awareness. In most cases of anesthesia care, lower MAC levels are given in combination with opioids and occasionally nitrous oxide.

Until now, there has been controversy over which type of anesthesia has the highest incidence of awareness: balanced with volatile agents or total intravenous anesthesia (TIVA). New data show a higher incidence during TIVA [6]. TIVA requirements vary considerably among patients. In addition, the lack of end-tidal anesthetic gas concentrations and greater chance of administration errors also increase the risk of awareness with TIVA. Brain function monitors may be useful in assessing anesthetic depth. Most are based upon analysis of EEG activity, as changes in consciousness create predictable EEG changes. Even acoustic evoked potentials are done for determining anesthesia depth. Titration of anesthesia using the bispectral index (BIS) monitor, a brain function monitor using cortical EEG, reduced the incidence of awareness, but did not completely prevent it, in patients at high risk for awareness [11]. While similarly effective in reducing awareness as use of end-tidal anesthetic gas levels during inhalational anesthesia [2], use of a brain function monitor is recommended to assess the degree of hypnosis during TIVA [3].

Many patients with awareness remember conversations during surgery as the block of sound impulses during general anesthesia is not reliable. For this reason, it may be beneficial to cover the ears of patients at increased risk for awareness.

If awareness occurs, the complaints of the patients must be taken seriously. An empathetic discussion with the patient and an explanation of the events is important. Subsequently, patients should be referred to a mental health specialist for counseling. It is unclear if immediate counseling can reduce the likelihood of serious psychological sequelae, but referral is standard care.

>> Dr. Eldridge took a good hard look at me after our discussion about awareness. "This is a very uncomfortable situation for me. I am very sorry. Honestly, I offer you my deepest apologies. If you develop nightmares or anxiety attacks as a result of this experience, please contact me immediately. I also recommend you obtain psychological counseling, as it may help prevent severe psychological problems from developing."

I assured him it was OK, that I did not need psychological counseling, and I promised to contact him no matter what within the next few months. Somehow, however, it took a year before I managed to get him on the phone. During the first 2 weeks after surgery, I sometimes dreamed of the "equipment moving around in my stomach," but then the dreams went away. Today I can talk about it all normally, without emotional difficulty and without breaking out in a cold sweat. Thanks for listening.

And so ended Dr. Sharon Collins's story about her surgery for bowel obstruction and her awareness episode.

8.2 Case Analysis/Debriefing

8.2.1 Which Medical Errors Do You See in the Presented Case?

8.2.1.1 Intravenous Volume Replacement Therapy

This patient was hypovolemic due to the long period of small bowel obstruction symptoms and to the massive secretion of fluid in the abdominal cavity in combination with decreased

reabsorption. The underlying cause is a regional perfusion disorder and the resulting inflammation reactions, including the secretion of fluid in response to the increased osmolarity. Before surgery, isotonic fluid replacement therapy should be performed to counteract hypovolemia. Dr. Collins should have received IV fluids during the presurgical waiting period.

8.2.1.2 Hypokalemia

Mechanical bowel obstruction often causes nausea and vomiting. Due to the loss of acidic stomach acid, metabolic alkalosis and hypokalemia arise. The non-compensated metabolic alkalosis is defined by an increased pH value, increased bicarbonate, and a positive base excess. As compensation, the breathing effort is minimized; this leads to an increase in PCO_2 and possibly a normalization of pH value. Furthermore, potassium is exchanged with hydrogen ions from the extracellular space moved into the cell. This increases the plasma H^+ concentration while decreasing the plasma potassium. Recurrent vomiting further aggravates the extracellular hypokalemia.

Aggressive replacement therapy is not necessary, since removing the source of the metabolic alkalosis will reverse the electrolyte imbalance. However, severe hypokalemia should be corrected to prevent cardiac arrhythmias and prolongation of neuromuscular blockade.

Due to the reduction in neuromuscular excitation, the effect of muscle relaxants is increased. Typical ECG changes such as flattened or inverted T waves and ST depression, U waves, and extrasystoles can occur. A third reason to correct the hypokalemia is to correct the metabolic alkalosis due to the H^+/K^+ imbalance. Rapid administration of K^+ is dangerous as it may cause ventricular fibrillation (see Sect. 1.1.1).

8.2.1.3 Blood Gas Analysis

As discussed above, vomiting with a small bowel obstruction might cause a metabolic alkalosis. When the bowel obstruction has been present for a long period of time, arterial blood gas analysis should be performed before induction of anesthesia.

8.2.1.4 Premedication

Our patient, Dr. Collins, wanted to be fully conscious for the anesthesia and surgical procedures, which is understandable. The lack of a benzodiazepine premed, however, facilitated the occurrence of awareness. In hindsight, since no volatile anesthetics were used, the awareness episode might have been prevented by premedication with midazolam just before induction of anesthesia.

8.2.2 Which Systems Failures Can Be Found in the Presented Case?

8.2.2.1 Algorithm Awareness Prophylaxis

Certain surgical and anesthetic combinations (listed in Sect. 8.1.8) increase the risk of awareness in high-risk cases; you should think of strategies to prevent awareness in advance, such as use of a brain function monitor during TIVA to indicate periods of light anesthesia.

8.2.3 You Made a Mistake, and You Await the Patient's Complaint. What Should You Do?

There is enough literature about the procedure to follow, including legal aspects, after making an error. We want to concentrate on the psychological aspects in this case.

In the presented case, Dr. Eldridge didn't yet know that he had made a mistake, when he visited the patient. Regardless of the cause of a complication, it is always important to talk to the patient and family if complications occurred. By direct communication, the patient can describe symptoms, the anesthesiologist can discuss possible etiologies and show empathy, and the patient is given the chance to express any negative feelings. If an error was made, an apology is indicated. In his postoperative discussion, Dr. Eldridge acknowledged that he was responsible for the awareness episode and he apologized for the complication arising from an inadequate dose of propofol.

Since he didn't make excuses and he didn't try to justify himself or his actions, the doctor–patient trust was not compromised. Legally, Dr. Eldridge responded correctly; he made no mention of fault, he tried to maintain transparency about the situation, he tried to convey accountability, and he offered help for the future.

What value do his mistakes have for our clinical care? We learn from errors, our own and others' mistakes (see Case 1 to Case 33)! It is therefore important not to sweep them under the rug, but to discuss them openly and learn from experience. An incident report to hospital risk management should be submitted, as the Joint Commission deems awareness during anesthesia a sentinel event. In addition, Dr. Eldridge should report the case to his department's continuous quality improvement (CQI) committee, and then a root cause analysis could be performed. Discussion of findings with other anesthesiologists within the department would increase the sensitivity of the group to this problem and determine changes in care in future cases. The analysis of the chain of events leading to this awareness occurrence would help to detect systems failures and brainstorm about solutions.

A requirement for all of this is a safety culture open for mistakes and a non-punitive response to errors. Also, an anonymous, critical incident reporting system is a valuable tool, in that it lowers the threshold which must be overcome to report mistakes. The Anesthesia Quality Institute (AQI) maintains a confidential anesthesia incident reporting system (AIRS) in the USA (www.aqiairs.org).

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Case 9: D&C

9

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9.1 Case Introduction

>> Mrs. Abigail Taylor was 57 years old and had a long medical history. Five years ago, she had had an anterior–posterior (A/P) rectum resection with a colostomy placement. At that time, hepatic cirrhosis (Child–Pugh Score B) was diagnosed, and since that time she has remained abstinent from alcohol. The abstinence was tough for her. Before this diagnosis, neither she nor anyone else had ever suspected that she was an alcoholic. Nevertheless, she had now conquered alcoholism.

In daily activities, Mrs. Taylor was limited due to her obesity. Despite this, she enjoyed a full life within her family circle, even if her strength had failed lately. For about a year, she had been complaining of recurrent vaginal bleeding. Three D&Cs had been necessary, and that was again the reason for her preoperative visit today. Mrs. Taylor's past surgeries were uneventful, and she was relaxed about the upcoming surgery. She was drinking a cup of coffee and chatting with another patient in the waiting room waiting to see the anesthesiologist. It was already afternoon, and she wanted to take a walk outside.

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Dr. Casey, a young anesthesiologist, was in charge of evaluations in the preoperative clinic. It was almost 4 p.m., and Dr. Casey moaned as he opened Mrs. Taylor's thick medical record. Thank goodness, there was an anesthesia record from the previous month, so hopefully the evaluation wouldn't take so long. After all, Dr. Casey became an anesthesiologist because he placed great importance on getting off work on time.

After studying Mrs. Taylor's records, he noted the following on the preanesthesia form:

Past medical history:

- *S/P A/P rectum resection with colostomy*
- *Alcoholic liver cirrhosis Child B with portal hypertension and esophageal varices*
- *Systemic hypertension*
- *Aortic valve stenosis*
- *Diabetes mellitus, type 2, diet controlled*

Medication:

- *Propranolol 50 mg PO QD*
- *Ramipril 5 mg PO QD*
- *Furosemide10 mg, PO QD*
- *Pantoprazole 20 mg PO QD*
- *Vitamin K 10 mg PO QD*

9.1.1 What Are the Possible Indications for Propranolol, and Why?

Propranolol is a noncardioselective β-receptor antagonist and is prescribed as a therapy for hypertension, along with ramipril, an angiotensin-converting enzyme (ACE) inhibitor. Mrs. Taylor's β-blockers helped to maintain her basal heart rate. In aortic stenosis, the heart rate should be between 60 and 80 beats/min [1]. Extreme brady- or tachycardia must be avoided. Generally, a cardioselective β-blocker with primarily β-receptor affinity is chosen for the treatment of hypertension with heart rate control and in some cases also to treat congestive heart failure.

In Mrs. Taylor's case, decreasing the portal hypertension is an additional indication for propranolol. Seventy-five percent of patients with cirrhosis have portal hypertension and some form of portacaval anastomosis. Therapy is aimed at treatment of the primary cause [5]. Unfortunately, due to Mrs. Taylor's advanced stage of liver disease, causal therapy is no longer possible. To reduce the risk of bleeding from esophageal varices, the portal pressure should be kept below 12 mmHg or at least reduced by 20 % of the original pressure. Various studies have shown that this goal can be reached in 20–30 % of patients by administration of a noncardiac selective β-blocker [3]. The recommended dose of propranolol is around 70 mg, whereby therapy should be begun with a low dose and slowly increased.

>> Meanwhile, Dr. Casey figured he wasn't going to get off work on time today, because the preoperative evaluation of Mrs. Taylor was going to take a little longer, even though he had the previous anesthetic records. Something was missing, something he had to find before he could go to see the patient.

9.1.2 In the Presented Case, Which Preoperative Evaluation Would You Insist on Having?

9.1.2.1 Lab Values

Coagulation Tests

In advanced liver cirrhosis, a decreased synthesis of liver products is to be expected. Apart from the typical reduction of cholinesterase in serum, there is a disorder in the formation of vitamin K-dependent coagulation factors of the prothrombin complexes (factors II, VII, IX, and X); protein C, S, and Z; as well as antithrombin III (AT III). A determination of the synthesis disorder is done by checking of PT and PTT in serum. The current therapy with vitamin K hints to a coagulation disorder in Mrs. Taylor. In addition, pathological values can appear for albumin, alanine transaminase (ALT [also known as SGPT]), aspartate transaminase (AST, [also known as SGOT]), bilirubin, and lactate dehydrogenase (LDH).

Electrolytes

When a patient takes diuretics regularly, electrolyte values should be checked preoperatively.

Furosemide is a potent loop diuretic, which can cause hypokalemia.

Renal Function Tests
Renal function should be evaluated when kidney disease is known, suspected, or when there are conditions present which increase the risk of renal impairment (e.g., diabetes, hypertension). Advanced liver cirrhosis can also lead to hepatorenal syndrome.

Complete Blood Count
Increased postmenopausal bleeding can lead to anemia. Advanced liver disease is often associated with thrombocytopenia.

ECG
In moderate to severe aortic stenosis, possible typical changes in the ECG might be seen.
- Left axis deviation
- Signs of left ventricular hypertrophy
- Signs of myocardial ischemia (also a sign of pressure hypertrophy)
- Inverted T waves in chest leads V_4–V_6

In long-standing aortic stenosis, echocardiographic checkups to determine severity are recommended, especially when the patient's clinical fitness cannot be determined due to other diseases. With increasing severity of the aortic stenosis, the perioperative risk also increases.

>> …and so Dr. Casey was sorting through the pages of the very disorganized medical record once more. A recent ECG was easy to find, revealing a sinus rhythm, normal frequency, a first-degree AV block, and signs of ischemia. The rest of the work-up was missing. At just this moment, the pretty young ward nurse Anna appeared with the missing lab report. "I thought you might want to see this, hmm?" she said with an adorably mischievous smile. Dr. Casey nodded, turned red, and then quickly huddled over the results:
- *Hb: 11.6 g/dl (reference 12–14 g/dl)*
- *HCT: 35 % (reference 37–48 %)*
- *White blood cell count: 7,200 cells/mcl (reference 4,300–10,800 cells/mcl)*
- *Platelets: 120,000/mcl (reference 150,000–400,000/mcl)*
- *PT: 15.7 s (reference 9.5–13.5 s)*
- *PTT: 41 s (reference 25–40 s; laboratory dependent)*
- *Bilirubin: 1.6 mg/dl (reference 0.2–1.1 mg/dl)*
- *ALT: 13 IU/l (reference 10–40 IU/l)*
- *AST: 8 IU/l (reference 10–34 IU/l)*
- *LDH: 280 IU/l (reference 120–240 IU/l)*
- *Creatinine: 1.1 mg/dl (reference 0.68–1.17 mg/dl)*
- *Na^+: 138 mEq/l (reference 136–145 mEq/l)*
- *K^+: 3.4 mEq/l (reference 3.8–5.2 mEq/l)*

9.1.3 How Do You Interpret the Lab Values?

The Hb and HCT values are most likely decreased due to postmenopausal bleeding; no intervention is necessary at this time. PT and PTT are abnormal as well. The cause could be the limited hepatic function due to cirrhosis. This is also the cause of the abnormal liver function tests. The diuretics could cause the hypokalemia.

>> "Good, I finally have everything together," thought Dr. Casey. With the help of the past records, the preoperative evaluation form was quickly filled out so that he could talk to and examine the patient and obtain informed consent. After looking through the preanesthetic checklist, there appeared to be no new aspects of Mrs. Taylor's condition.

Mrs. Taylor admitted to only being able to climb one flight of stairs for about the last 6 months. Before that she could go up three flights without stopping for breath. "It's just because of my love handles," she said with a laugh. "There's a little more love there this year. I love to eat and my tummy becomes a little bigger, especially without exercise."

Mrs. Taylor was 5′2″ tall and weighed about 85 kg. Dr. Casey began to listen very attentively. In addition to the 3/6 systolic heart murmur and obesity, he identified a new finding by his careful physical exam, ascites! He also noted bruises on her arms and legs. After careful questioning, Mrs. Taylor admitted to getting bruises after a simple bump. Dr. Casey moaned to himself: "Why

do I always get the most difficult patients at the end of the day?" *A few minutes later*, *he had all his questions answered*; *he had formed a clinical picture of this patient. Mrs. Taylor was looking at him expectantly*, *waiting to hear what he had to say next*.

9.1.4 Which Form of Anesthesia Would You Recommend for Mrs. Taylor and Why?

Generally, subarachnoid blocks can be used for short procedures. Low-level spinal anesthesia has less effect on the pulmonary function in adipose patients. Alternatively, monitored anesthesia care (MAC) with sedation and a paracervical block can be considered. However, due to Mrs. Taylor's comorbidities, general anesthesia is recommended. Aortic stenosis is by itself a relative contraindication for spinal anesthesia. By decreasing the venous return, due to venous pooling (especially in areas innervated by the splanchnic nerve), there is reduced filling of the left ventricle.

Simultaneously, the decrease of systemic resistance reduces coronary perfusion and therefore decreases myocardial oxygen supply. This can result in a significant reduction of cardiac output, leading to a further reduction of myocardial perfusion. Reflex tachycardia may occur with shortened diastole which further reduces myocardial perfusion. Furthermore, the history of easy bruising and the abnormal coagulation studies are contraindications for neuraxial blocks.

9.1.5 Which Pathophysiological Changes to the Heart and Hemodynamics Are Expected, Due to the Aortic Stenosis?

Aortic stenosis is a narrowing of the opening of the aortic valve. The normal size is 2.5–3.5 cm^2. When narrowed to more than one-third of this, the stenosis becomes hemodynamically significant. Classification of the severity of stenosis depends on the valve opening area and the systolic pressure gradient between the left ventricle and the aorta; the pressure gradient depends on the cardiac output and therefore must be interpreted with the cardiac output in mind (see Table 9.1).

Table 9.1 Classification of the severity of the aortic valve stenosis

Severity	Systolic pressure gradient [mmHg]	Area of valve opening [cm^2]
I	<40	>1.5
II	40–80	0.8–1.5
III	81–120	0.4–0.8
IV	>120	<0.4

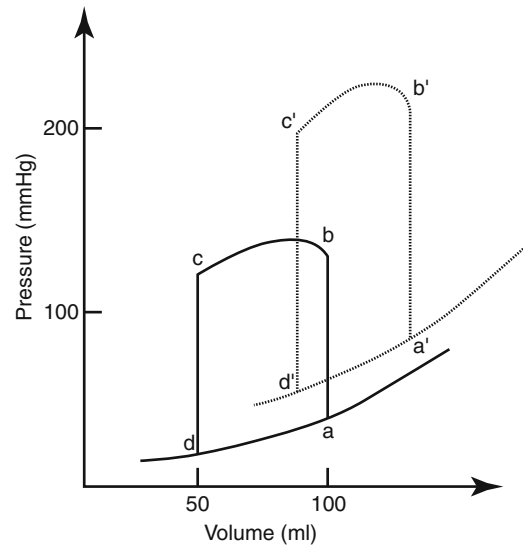

Fig. 9.1 Pressure–volume curve of the left ventricle. In aortic valve stenosis, the thickened myocardium of the left ventricle is less flexible (reduced compliance). Therefore, the pressure volume curve moves up and to the right (*dotted line*). There is increased end-diastolic pressure and volume ($a \rightarrow a'$). Since the left ventricle must pump against a fixed gradient (increased afterload), the endpoint of the isovolumetric contraction also shifts ($b \rightarrow b'$)

Aortic valve stenosis increases systolic resistance. The increased left ventricle pressure strain causes concentric left ventricle hypertrophy. The pressure volume curve is pushed up and to the right. With increasing hypertrophy, a compliance disorder develops so that over the clinical course, the left ventricular end-diastolic pressure and, secondly, the pressure in the left atrium increase (see Fig. 9.1).

The increased basal myocardial energy and oxygen demand are a result of the pressure burden by the increased cardiac myocardial mass [4]. Coronary perfusion may not be sufficient for the increased demand so that myocardial ischemia may arise. Reasons for decreased myocardial perfusion are:

- Inadequate vascularization of the myocardium for the proportionately large muscle mass
- A reduced coronary perfusion gradient due to increased end-diastolic pressure and decreased post-stenosis pressure in the aorta and due to an increased diffusion pathway through the hypertrophied myocardium

If myocardial ischemia occurs, the left ventricle may react with serious arrhythmias, including ventricular fibrillation. The clinical symptoms, which tend to appear during physical exertion, are:

- Angina pectoris without evidence of coronary artery stenosis
- Fainting (cardiac syncope)
- Acute heart failure

>> *After all these thoughts went through Dr. Casey's head, he discussed general anesthesia with Mrs. Taylor. She became a little upset, because her previous surgeries had gone well with spinal anesthesia. Dr. Casey explained why a spinal was now contraindicated, and he promised to prescribe a sedative for her to take before bed.*

Mrs. Taylor actually slept well. The surgery was planned for the morning, so she didn't eat breakfast. However, as she was about to leave for the hospital, a nurse phoned her to tell her that her surgery would be delayed until 1 p.m., due to an emergency.

Dr. Diana was assigned to do the anesthesia, a resident with 2 years of anesthesia experience. Reviewing the medical record and preoperative evaluation, she stumbled upon the patient's comorbidities. Dr. Diana wondered if she should call the attending anesthesiologist, Dr. Eldridge, to discuss the case; but then she decided not to. She was sure that he had been informed about Mrs. Taylor yesterday. In addition, he was supposed to see the patient himself in the preoperative area. Dr. Eldridge told Dr. Diana to get started with the patient's anesthesia and he would be in shortly. "We'll use the ProSeal laryngeal mask airway," she said to the anesthesia technician Freya. Due to Mrs. Taylor's unremarkable previous anesthetics and the expected short duration of the surgery, Dr. Diana saw no reason to deviate from routine anesthesiology techniques, which she had mastered.

Mrs. Taylor was positioned in the lithotomy position; a 5-lead ECG, blood pressure cuff, oxygen saturation clip, and an IV infusion were all set up. The blood pressure was 150/80 mmHg; the heart rate was 55 beats/min. After preoxygenation, she received 150 µg fentanyl and 150 mg propofol IV.

Dr. Diana placed the ProSeal laryngeal mask airway, size 4, without incident and mechanically ventilated the patient with pressure-controlled ventilation due to the patient's obesity. In order to maintain anesthesia, Dr. Diana turned on the desflurane vaporizer. "Doing well," she thought and informed the experienced gynecological team that they could begin.

The next blood pressure reading, the first after induction, showed pronounced hypotension, at 70/50 mmHg, with a pulse of 65 beats/min (which was a slight increase from the preinduction pulse). Dr. Diana increased the IV fluids and gave phenylephrine 200 µg IV. Dr. Diana briefly considered giving more fluids, but then she remembered that because of the left ventricular pressure load, the left atrium was probably strained, and a relative mitral valve regurgitation could occur, leading to pulmonary edema. So, she decided against it.

The next blood pressure was 80/60 mmHg, and Mrs. Taylor received additional phenylephrine 250 µg. "She's responding, but slowly," thought Dr. Diana. The gynecologists picked up on her increasing nervousness. "Is something wrong?" asked one of them. "No, no." Dr. Diana replied and then asked, "Is it bleeding a lot?" The gynecologist responded, "No more than usual. In 3 min we'll be finished."

Dr. Diana relaxed upon hearing the news and shut off the desflurane vaporizer. Suddenly Mrs. Taylor's heart rate fell to 30 beats/min. Dr. Diana hit the off button for the monitor's alarm

and opened the medication drawer of the anesthesia cart as fast as she could to get some atropine. She yelled for Freya to help out. The anesthesia technician was in another OR helping during induction of anesthesia. When Freya did enter the OR, she saw Dr. Diana with a vial of atropine and the monitor: the ECG showed a flat line. There was no alarm – asystole.

9.1.6 Why Didn't the Monitor Give an Alarm?

There are two ways to turn off a monitor's alarm:
- Cancellation of the alarm
- Shutting off the alarm tone (the "silence" button)

The cancellation of an alarm changes the setting for the alarm software. Most monitors are programmed so that continuation of a cause for an alarm (such as the bradycardia in this case) will not set off the alarm again for a certain time period, usually about 2–3 min. Other causes for an alarm, like hypotension or a reduction in oxygen saturation, cause a new signal to sound.

The difference is that in some monitors, cancellation of one alarm signal cancels all alarm signals. This means that new events may not trigger an alarm. Newer monitors are equipped with software which sound alarms for defined events, such as asystole and or ventricular fibrillation.

The basic rule: Canceling an alarm is preferred to shutting off all alarms.

>> "*Do you realize that the patient no longer has a pulse*?" *asked the gynecologist*. "*That can't be true*," *said Dr. Diana, who dropped the atropine vial and turned pale. The gynecologist jumped up from his stool. He was, thank goodness, finished with his operation*. "*What should I do*?" *he asked everybody in the room. Anesthesia technician Freya had begun chest compressions. Dr. Diana jolted back from her shock*. "*Please tell my attending to get in here*!" *she yelled to the gynecologist*.

In the blink of an eye, or maybe a few blinks, attending anesthesiologist Dr. Eldridge appeared, with a CRNA at his side. He raised his eyebrows in shock, asked what on earth was going on, analyzed the cardiopulmonary resuscitation efforts, and, without waiting for an answer, took over from there. He instructed Dr. Diana to assist the anesthesia technician with ventilation during the resuscitation. Next he set the inspiratory oxygen to 100% and intubated Mrs. Taylor with help from a CRNA. Finally, appearing cool and calm from the outside, he asked for the crash cart and ordered epinephrine.

After about 8–9 min of chest compressions and a total of 4 mg of IV epinephrine, Mrs. Taylor was again in sinus rhythm and had a measurable blood pressure. To further stabilize the blood pressure, a norepinephrine infusion was started. "*I'll get a bed in the ICU. Would you please prepare for insertion of an arterial line*?" *Dr. Eldridge said to anesthesia technician Freya. Turning to Dr. Diana, he said* "*We'll talk about this later*."

Dr. Diana accompanied Mrs. Taylor to the ICU; the patient received an infusion of norepinephrine during the transfer to maintain sufficient blood pressure. Humiliated, Dr. Diana went back to the gynecology OR, where her attending, Dr. Eldridge, was waiting her attending, Dr. Eldridge.

9.2 Case Analysis/Debriefing

9.2.1 Which Medical Errors Do You See in the Presented Case?

9.2.1.1 Preoperative Evaluation

Echocardiography could have easily been performed preoperatively. In our patient, an echo should have been done because the patient complained of a decrease in exercise tolerance. This decrease is a hint to the progression of aortic stenosis. Another cause of the increased dyspnea upon exertion could be ascites. The preoperative anesthesiologist completely ignored the diagnosis of aortic stenosis.

9.2.1.2 Choice of the Anesthesia Technique

In addition to obesity, Mrs. Taylor had substantial ascites. The result is an increased risk of aspira-

tion and indication for RSI. Laryngeal mask airway or similar upper airway devices do not protect against aspiration. Due to the water retention and tendency to develop edema, a difficult intubation is to be expected. Tachycardia during endotracheal intubation should be especially avoided to prevent myocardial ischemia.

Obesity and ascites create an elevation of the diaphragm and mechanical impingement of its function, leading to a restrictive lung disease. This restrictive ventilation pattern is further compounded by general anesthesia so that the total lung and functional residual capacities are reduced. The reduced lung surface area for the gas exchange increases the danger of hypoxemia. To safely provide for the increased myocardial oxygen consumption, due to the aortic valve stenosis, ventilation with PEEP is recommended. Increased ventilation pressures are expected.

9.2.1.3 Monitoring

Mrs. Taylor's monitoring during the anesthesia was insufficient. Despite the brevity of the procedure, more extensive monitoring should have been used. In patients with aortic stenosis, the biggest danger is myocardial oxygen deficiency. Therefore, the hemodynamic stability of the patient is the critical factor for coronary perfusion.

Hemodynamic changes must be detected early and treated quickly. Therefore, direct arterial blood pressure monitoring should have been done. The arterial line should be inserted before induction of general anesthesia. The blood pressure needs to be maintained within the patient's preoperative range, and extreme changes in heart rate must be avoided. Especially important is maintaining synchronized atrial contraction for maximum ventricle filling, which is why supraventricular arrhythmias must be immediately treated. Even with small amounts of anesthetics, patients can react with drops in blood pressure and cardiac output, leading to reductions in coronary perfusion. The cardiac output can increase only minimally with tachycardia, since the ejection fraction is relatively fixed by the aortic stenosis. Even short-term blood pressure decreases must be immediately recognized and treated.

9.2.1.4 Choosing/Dosing the Anesthetic Agent

There is a basic rule of all anesthetic agents: they depress the cardiovascular system. The extent of the depression of the cardiovascular system is dose dependent. The anesthetic agents should be very carefully administered. The initial bolus of 1.5 mg/kg of propofol was obviously too much here. An alternative agent, such as etomidate, causes less cardiac depression and hypotension. While ketamine also tends to preserve hemodynamic stability, it may result in tachycardia and hence is not a good choice in this patient.

9.2.1.5 Hypotension Therapy

Patients with aortic stenosis are dependent on sufficient intravascular volume due to the poor left ventricular compliance. The ejection fraction can't be increased as needed, and tachycardia simultaneously shortens the duration of diastole and the already reduced coronary perfusion. Euvolemia is necessary to assure sufficient ventricle filling, and tachycardia and hypotension must be avoided in order to assure coronary perfusion.

The biggest danger, therefore, is not pulmonary edema, but hypovolemia. Hypotension should be treated with volume replacement. If unsuccessful, vasopressors should quickly be used, but again, there should be sufficient volume replacement. Only aggressive therapy may prevent cardiovascular collapse. While phenylephrine is often the initial choice for treatment of hypotension due to its ready availability, vasopressin should be quickly given if no response. If the hypotension is severe and unresponsive to vasopressin and phenylephrine, norepinephrine is indicated. Norepinephrine is an inotrope and it increases systemic vascular resistance. It causes tachycardia less frequently than dopamine or epinephrine and usually only with higher doses.

9.2.1.6 Cancellation of the Monitor Alarm

This point was already discussed in Sect. 9.1.6.

9.2.2 Which Systems Failures Can Be Found in the Presented Case?

9.2.2.1 Preoperative Evaluation

Due to the serious comorbidities, the anesthesiologist-in-charge ("board runner") should be notified about the upcoming case after the patient is seen in preoperative clinic. Ideally, the case should be discussed with the attending anesthesiologist prior to surgery. On the other hand, part of the attending's job is to be informed about the patients he/she is supervising.

9.2.2.2 Changes in the Surgery Schedule

Also here, the attending anesthesiologist should have been informed of the delay for this high-risk patient. Only then would he have had the chance to order enough volume replacement therapy, since the deficit occurred because of the late time slot and longer fasting period. As explained earlier, because of her aortic stenosis, Mrs. Taylor was especially endangered by hypovolemia.

9.2.2.3 Supervision

Dr. Diana was a resident in her third year. The American College of Graduate Medical Education (ACGME) requires resident supervision for all anesthesia procedures in accreditation of residency programs. Billing procedures require the attending anesthesiologist to perform a preoperative evaluation; be present on induction, emergence, and all critical phases of anesthesia; and be immediately available throughout. In addition, he/she (or partner) is responsible for care in the postanesthesia care unit. The attending anesthesiologist Dr. Eldridge did not fulfill his part; however, Dr. Diana didn't discuss the case with him nor wait until he was available before inducing the patient.

9.2.2.4 Cardiopulmonary Resuscitation Training

Dr. Diana was obviously overwhelmed with the emergency situation, as anesthesia technician Freya began with the compressions. Maintenance of advanced cardiac life support (ACLS) certification is required for anesthesiologists, including residents, for hospital/ambulatory center privileges. Emergency training must be done regularly in the simulation lab in order to internalize the algorithms and overcome hindrances. Checklists for intraoperative emergencies are also becoming popular as they increase adherence to guidelines and recommended therapies [2]. Enlisting the circulating nurse or an anesthesia technician in reading the checklist during the emergency situation can also be helpful. Maintenance of certification in anesthesiology (MOCA) requires simulation training during each 10-year cycle.

9.2.3 Dr. Diana Neglected to Obtain Help from Her Attending Before Beginning. She Assumed He Had Already Been Informed. Has That Ever Happened to You?

Despite wondering about the patient's comorbidities described in the medical record, the resident decided to go ahead with the anesthesia without discussing the case with her supervisor. She assumed that he knew about the patient's serious comorbidities. In addition, the resident who performed the preoperative evaluation the day before should have notified the in-charge anesthesiologist, and appropriate assignments could be made. This behavior is known as "**responsibility diffusion**." Neither the resident who saw the patient in preoperative clinic nor Dr. Diana took responsibility for informing their supervisors.

When people assume that there are others responsible for carrying out actions, the perception of their actual responsibility goes along with the motto: somebody else will do it; it obviously needs to be done. How can such "responsibility diffusion" be avoided?

The solution is so simple and so obvious that it is often not done: we must talk to one another! By explicitly checking with the other anesthesiologist, the board runner, or the attending anesthesiologist for the case, the strategy would have been different, hopefully.

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Case 10: Inguinal Hernia Repair

10

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Contents

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10.1 Case Introduction

>> Two months had already passed since Dr. Clare finished her anesthesia residency and had become a junior attending. Near the end of her residency, Dr. Clare had done an additional pediatric anesthesiology rotation so that she would be competent anesthetizing children under 2 years of age. Although she had experience anesthetizing children on other rotations, the pediatric anesthesia rotation had so many young babies. Therefore, this rotation was something special, even for a specialist.

Today was hernia day; there were a total of 5 newborns to care for, all younger than 3 months. The work in pediatric anesthesia was so enjoyable for Dr. Clare. The whole team – herself included – worked well together. Most hands-on manipulations were standardized as well as easy to do so that extensive communication was not necessary. Just now, CRNA Patricia brought the third infant into the OR. His name was William; he was 5 weeks old and a former "preemie."

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10.1.1 How Is Preterm Infant Defined? Which Characteristics Must Be Kept in Mind During the Postoperative Time Period?

Newborns born before the 37th week of gestation are referred to as premature. An inguinal hernia is a typical disease of former premature neonates. Due to the risk of incarceration, the hernia often requires operation during the first weeks of life.

The brain respiratory center is more sensitive to analgesics and hypnotics in babies who were born prematurely. This is one reason respiratory complications after general anesthesia are more commonly seen, as compared to mature newborns [15]. Therefore, adequate postoperative monitoring is mandatory for the 24 h following the anesthesia. From an anesthesia perspective, postponing surgery as long as possible is desired; with increasing age, the respiratory center becomes more robust and less influenced by analgesics and hypnotics.

>> *Hernia surgery is commonly performed with a combination of general anesthesia and a single-shot caudal anesthesia. Dr. Clare had now done about 20 cases with this technique and had slowly begun to feel more relaxed.*

10.1.2 What Do You Think of Combining Anesthetic Techniques for This Surgery?

Due to the immature respiratory center of the former premature infants, it is theoretically beneficial to abstain from or use as little as possible respiratory depressant drugs, including opioids, hypnotics, and general anesthetics [9]. However, there is no definitive scientific proof of benefits for the combination procedure [4]. Regional anesthesia techniques for inguinal hernia operations can be performed alone (e.g., spinal or epidural) or are employed with a "light" general anesthesia.

>> *Little William was born during his 35th gestational week and had developed well, apart from the temporary newborn jaundice. Two weeks after birth, he was happily discharged with his mother, and he now weighed about 4,000 g.*

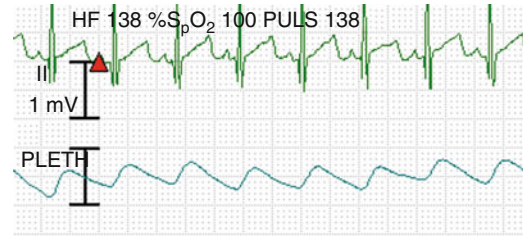

Fig. 10.1 ECG and pulse oximetry waveforms after induction of general anesthesia

Dr. Clare had already seen William, because the anesthesiologists visited all pediatric patients with scheduled surgeries before surgery. To make sure, she reviewed the preoperative evaluation once more, confirming that there was nothing remarkable about his family history. "With so many little patients with the same diagnosis, one can't recheck too much," she assured herself as she turned to the infant.

William already had an IV inserted by a pediatric surgery colleague. There was a crystalloid infusion running at 40 ml/h. CRNA Patricia wrapped an oxygen saturation sensor around William's hand and hooked up the ECG.

The monitor showed a sinus rhythm with a rate of 160 beats/min and a pulse oximetry reading (S_pO_2) of 100 %. Anesthesiologist-in-charge, Dr. Eldridge, peered through the OR window. He gave Dr. Clare a sign that the surgeon was outside the OR, so she could begin with her induction. Dr. Clare sat on the head of the OR table and held the mask for preoxygenation over William's nose and mouth. "Please administer 20 mg of propofol," she said to CRNA Patricia. "That is 2 ml of propofol," answered Patricia, and began the injection. William's heart rate dropped to 138 beats/min (*see* Fig. 10.1). *The blood pressure was 60/30 mmHg.*

10.1.3 Isn't That a Little Too Much Propofol?

The usual induction dose for adults is 1.5–2.5 mg/kg body weight. Small children and newborns need higher doses of propofol and other hypnotic

agents. Among other things, this is due to significantly larger volume of distribution.

>> Until now William had been quiet, but now with the propofol injection he began to cry and hiccup a little. "It is such a shame that we can't give the guy a little lidocaine," Dr. Clare said to Patricia. The hiccups remained, even after William had gone to sleep.

Dr. Clare tried to ventilate him carefully, but without success. The S_pO_2 value was now 99 % and the heart rate was 170 beats/min. The sliding door to the OR opened. Anesthesiologist-in-charge Dr. Eldridge glanced at William and Dr. Clare's attempts and announced: "The most common cause of difficult mask ventilation is lack of sufficient anesthesia depth." According to Dr. Clare's orders, CRNA Patricia administered another 20 mg of propofol, followed by 10 µg fentanyl. The hiccups disappeared, but the ventilation still was difficult.

10.1.4 What Are the Causes for Difficult Mask Ventilation When the Anesthesia Is Deep Enough?

The mask ventilation of a newborn and infants is difficult for the inexperienced. The most common mistakes are obstruction of the airway:

- Due to overextension of the neck – mask ventilation is only possible in the neutral position
- Due to strong pressure of the mask on the nose, causing obstruction of the nasal passages
- By pressure on the floor of the mouth, causing closure of the choana by the tongue

>> Dr. Clare heard the oxygen saturation of the pulse tone dropping, and she started to get nervous.

"We'll be fine," whispered Patricia calmly. "Don't extend the neck quite so much, and don't press on the floor of the mouth."

Dr. Clare was thankful for the advice, as William then allowed himself to be effortlessly ventilated with the mask. The saturation increased all at once to 99 %. "Now we can intubate," she said to Patricia.

10.1.5 Wasn't Something Forgotten Here?

Intubating without muscle relaxants is controversial. Not only does it make the intubation more difficult, but it is also associated with an increased incidence of hoarseness, throat pain, and laryngeal damage [3]. Despite this, muscle relaxants are often avoided in pediatric anesthesia [13]. The main reasons are the presence of a contraindication for muscle relaxants and an anticipated short duration of surgery.

In newborns and especially former preterm babies, there is a further issue: due to their increased volume of distribution in comparison to adults, relatively larger doses of muscle relaxants are needed. The hepatic and renal elimination occurs more slowly, and the duration of action cannot be accurately estimated. A possible residual relaxation increases the danger of postoperative hypoxemia.

Newborns have less muscular tone than older children and adults so that intubation without muscle relaxants is more easily performed. There is the danger, however, that the depth of anesthesia is misinterpreted as being deep enough, when in fact it is not.

>> Dr. Clare laid the mask to the side and CRNA Patricia gave her an endotracheal tube with an internal diameter of 3.5 mm. She guided the tube first into the right, then into the left nasal passage, but each time, she felt resistance. This had never happened to her before. The oxygen saturation tone of the monitor was already deeper. Dr. Clare aborted the intubation attempt and ventilated William again by mask.

"Please give me a smaller tube," she requested. CRNA Patricia responded, "It will probably leak. Perhaps you should perform an oral intubation." Thankful again for the advice, Dr. Clare was able to promptly intubate the trachea. The tube was secured and William was turned on his side. To maintain sufficient depth of anesthesia, Dr. Clare turned on the sevoflurane vaporizer.

After turning the child prone, she prepped the sacral region. CRNA Patricia had already prepared ropivacaine 0.2 % as the local anesthetic and had set up the caudal tray.

10.1.6 How Much of the Local Anesthetic Would You Use for William's Caudal Anesthesia?

The dose is calculated according to height, weight, and the planned procedure. Usually the Armitage formula is used (see Table 10.1). Interestingly, the Armitage formula is not based on any scientific tests, but from a "Letter to the Editor" that Armitage wrote in 1979 [1]. Since then, the formula has been cited as evidence-based knowledge.

With a body weight of 4 kg and a planned inguinal hernia operation, 4 ml of local anesthetic should be injected. To increase the duration of action, 1–2 µg/ml of clonidine can be added [11]. In newborns, however, clonidine should not be given due to possible post-op respiratory depression [2].

>> Dr. Clare's placement of the caudal catheter was unremarkable. As no cerebrospinal fluid and no blood returned, she injected 4 ml of ropivacaine 0.2 %, removed the needle, and taped a sterile dressing over the injection site. Suddenly an alarm sounded from the ventilator; "decreased end-expiratory CO_2" was blinking on the screen. "I hope the endotracheal tube didn't come out," she said to Patricia. Together they rolled William supine, and Dr. Clare changed the ventilator to manual ventilation, increased the inspiratory oxygen to 100 %, and checked the placement of the endotracheal tube. "The tube is properly placed." Dr. Clare announced, as the next alarm went off – this time for bradycardia. Pulse was now only 110 beats/min.

Table 10.1 Dosing with the Armitage formula [1] for caudal application of a local anesthetic

Dose [ml/kg]	Dermatome affected
0.5	Lumbar 1
0.75	Thoracic 12
1	Thoracic 10
1.25	Thoracic 6–8

10.1.7 What Would You Do Now?

A decrease in heart rate is life-threatening in infants and toddlers; cardiopulmonary insufficiency and a functional circulatory arrest occur with a heart rate under 100 beats/min.

The most common cause is hypoxemia, which was already ruled out by Dr. Clare in this case. The goal of therapy now must be to increase the heart rate as fast as possible.

>> Dr. Clare was now very nervous and hectically squeezed the bag. "Give me 0.1 mg atropine," she said to Patricia, "and call for help!" Anesthesiologist-in-charge Dr. Eldridge was in the PACU with another baby. He dropped everything (except the baby) when the call for help reached him.

"What is the problem?" he asked Dr. Clare as he ran into the OR. She gave an overview of the events. Anesthesiologist-in-charge Dr. Eldridge studied the monitor (see Fig. 10.2).

10.1.8 How Do You Interpret Fig. 10.2 and What Action Would You Now Take?

Compared to Fig. 10.1, wide ECG complexes are visible with a rate of about 100 beats/min. The pulse oximetry curve is flat. Taking into consideration the decreased end-tidal CO_2, the baby is in cardiopulmonary arrest and CPR must begin immediately and advanced cardiac life support begun immediately [5, 7].

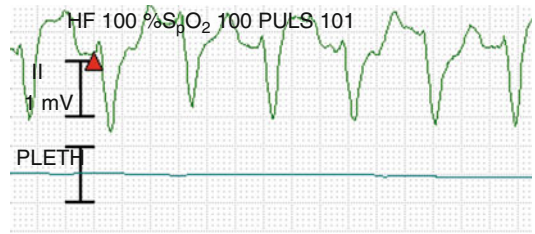

Fig. 10.2 ECG and pulse oximetry waveforms (see Sect. 10.1.8)

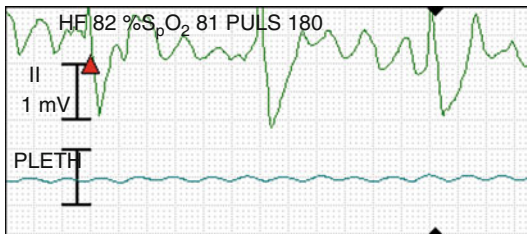

Fig. 10.3 ECG and pulse oximetry waveforms (see Sect. 10.1.9)

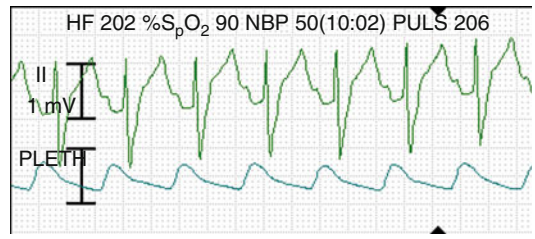

Fig. 10.4 ECG and pulse oximetry waveforms. Compared to the previous waveforms, these ventricular complexes are narrower. The T waves are significantly more peaked than in Fig. 10.1. The pulse oximetry waveform shows adequate cardiopulmonary function

>> Dr. Eldridge checked placement of the endotracheal tube once more. William now appeared cyanotic. Dr. Eldridge encircled William's torso with his fingers and placed his two thumbs side by side over the lower third of the sternum. Then he began chest compressions, compressing the sternum to approximately one-third of the anterior–posterior diameter. William quickly became rosy again. The monitor showed the following (*see* Fig. 10.3).

10.1.9 What Do You See on the Monitor Now? What Do You Do Now?

The monitor now shows a pulse oximetry waveform as a sign of the adequate external chest compression. The ECG has changed again; besides the artifacts from compressions, malformed ventricular contractions with a pulse of about 50 beats/min are present. According to the American Heart Association [7] and international guidelines [5], epinephrine is indicated. The recommended dose in newborns is 10 µg/kg.

>> As he continued chest compressions, Dr. Eldridge ordered CRNA Patricia to administer 40 µg epinephrine IV. Following that, she prepared a 30 ml crystalloid infusion in a syringe as a bolus. One minute later, Dr. Eldridge interrupted the compressions and looked at the monitor (*see* Fig. 10.4). *The previously wide ventricular complexes were again narrow. He searched for the carotid artery and felt a strong pulse. The blood pressure measurement was then 70/30 mmHg. Everyone around the baby was so relieved.*

The anesthesiologist-in-charge, Dr. Eldridge, told the surgeons the surgery needed to be postponed, and then he called the ICU and arranged a bed. An arterial blood gas analysis done immediately after CPR revealed normal electrolyte values, no metabolic or respiratory acidosis, nor hypoxemia.

10.1.10 What Could Have Caused the Circulatory Changes?

10.1.9.1 Hypoxemia

As discussed in Sect. 10.1.7, the most common cause of circulatory depression in newborns is hypoxemia. In this case, hypoxemia was not the cause. Despite this, one must first rule out hypoxemia.

10.1.9.2 Cardiac Causes

It was unusual that the first symptom was a low end-tidal CO_2. This sign is interpreted as circulatory failure. Although the risk of a cardiac cause is very small in newborns, such causes must be considered.

10.1.9.3 Accidental Spinal Anesthesia

Immediately after the injection of the local anesthetic, the circulatory failure occurred. A possible cause is accidental intrathecal injection of local anesthetic, followed by spinal anesthesia (a "high spinal"). The ECG changes (see Fig. 10.2), however, speak against this possibility.

10.1.9.4 Local Anesthetic Toxicity

The timely correlation with the injection of the local anesthetic and the noted ECG changes indicate local anesthetic toxicity. Considering the rapid onset of symptoms (cardiovascular collapse), intravascular injection most likely occurred.

>> *"What actually happened here?" asked anesthesiologist-in-charge, Dr. Eldridge. Dr. Clare repeated the chain of events in detail. "Patricia, please show me once again the syringe with the ropivacaine," he said to the CRNA. Patricia showed him the syringe and the vial. "Dammit!" mumbled Dr. Eldridge, after he had studied both. "The baby received 1 % ropivacaine instead of 0.2 % ropivacaine. I'm not blaming you, Patricia, the two vials look almost alike. We must somehow make a change in here so that this does not happen to anyone again!"*

Finally, Dr. Eldridge took a moment to watch little William, sedated, intubated, and ventilated on the ICU. The baby's mom was already waiting on him. Dr. Eldridge briefly explained what had happened, and he did not neglect to mention that there was a mistake with the concentration of the local anesthetic. The mom looked very upset, afraid, and angry at the same time. Dr. Eldridge couldn't do anything but apologize again and again.

10.2 Case Analysis/Debriefing

10.2.1 Which Medical Errors Do You See in the Presented Case?

10.2.1.1 Circulatory Status Monitoring

Even though blood pressure values in newborns have only limited significance, changes can give important hints concerning the volume status and cardiovascular depression from anesthetics. In the presented case, there was no blood pressure measurement before administration of general anesthesia.

10.2.1.2 Choice of Medications for Intubation Without Muscle Relaxants

As already discussed in Sect. 10.1.5, intubation without use of muscle relaxants can be sensible in some situations. There are some clinicians who completely avoid muscle relaxants in all but very exceptional situations.

Various techniques for intubation without muscle relaxants have been studied. A mandatory recommendation for the choice of anesthetic agents can't be made, but there is consensus that the intubation should be done in deep anesthesia. To avoid high doses of propofol, the combination with a potent opioid such as remifentanil is recommended [16]. The administration of propofol at 10 mg/kg should be viewed critically.

10.2.1.3 Propofol

This is not a medical mistake, but a suboptimal treatment. Dr. Clare was justified to be upset that William's vein hurts, because lidocaine isn't given in neonates. However, there is an alternative: the anesthesia induction in this case was done with 1 % propofol, which regularly causes pain on injection. This is rarely observed when 0.5 % propofol is used.

10.2.1.4 Placement of Caudal Anesthesia

This issue has been discussed and debated in scientific literature for years (decades). The caudal injection of a test dose of epinephrine can help to detect intravascular placement. However, this test is not 100 % reliable; absence of an increase in heart rate does not rule out intravascular placement [12]. There is some evidence that ECG changes – especially ST- and T-wave increases – are more sensitive parameters [8].

10.2.2 Which Systems Failures Can You Find in the Presented Case?

10.2.2.1 Drug Error

No one is immune to medication errors. Medication errors are common during anesthesia,

estimated at one error in every 133 anesthetics [10]. Unfortunately in the USA, many drug vials look identical and syringe swaps are common. International efforts recommend standardized labeling of vials and syringes [6, 14]. In this recommendation, various medication classes should be color coded differently. The idea is that a mix-up within the same class of drugs (e.g., fentanyl with sufentanil) is less serious than a mix-up within substance groups (e.g., opioids with muscle relaxants).

In the presented case, a higher concentration of local anesthetic was chosen. Marking the medication group would not have helped. Systems solutions are needed so that similar-appearing medications are kept in different rooms. Furthermore, strict rules about medication application must be described. Also the "4-eye principle" is recommended, which means presentation of the used vial to a second person. Additionally a technique called "read-back" is helpful: the medication and dose is ordered, and then the medication and dose is repeated by the person administering the medication. Dr. Clare and CRNA Patricia were properly practicing this technique right up to the point of the caudal anesthesia.

10.2.3 Just Between Us: How Often Have You Experienced a Medication Error?

Medication errors are part of our daily life. Although media and press have reported so much on it that we all know it occurs regularly, we continue to have syringe swaps and other medication errors. Why?

Many factors make an error more likely. Some of the many causes are look-alike vial names and labeling, storage of medication, and individual eyesight of the practitioners involved. But even mandatory vision checks of doctors and nurses can't completely solve the problem, because the human information processing operates to conserve resources. Or do you check all syringes every time, even if someone else has drawn them up?

When our level of activation is either too low (too few demands, boredom) or too high (excessive demands, stress, and pressure), the human information search function becomes coarser, and fewer clues are considered for the information processing. Even when conflicting data – such as the wrong medication label or, in this case, the right medication in the wrong concentration – are present, the view which conforms to the hypothesis will be projected onto the variant form of reality.

In the presented case, we chose a non-stressful situation, in order to emphasize that mix-up is a problem of routine situations. Is there a solution? Of course! The solution, however, is a difficult one and difficult to establish in day-to-day hospital life:

- Working through checklists
- Read-back
- Double-sided checking and rechecking
- Always question your perceptions

Lastly, here is a challenge for you: Tomorrow, observe yourself once more; observe how the **principle of resource conservation** works in you!

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Case 11: Hip Surgery

11

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Contents

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11.1 Case Introduction

>> Mrs. Moore was 85 years old and was not intending to let herself be operated on again in her lifetime. Similarly, she tried to avoid physician visits and medications. For the past 5 years, she's had an arrhythmia, but she didn't want to take warfarin because she didn't want to constantly go to the doctor and have blood drawn. She'd managed to get along all right the past 85 years, and she was sure she'd be OK for the next 10 years. The baby aspirin were less annoying; she took these regularly with isosorbide dinitrate (Isordil), which helped her angina pectoris a lot. Her biggest problem now was pain when she tried to walk. Clenching her teeth and taking ibuprofen had not helped. The hip prosthesis was too loose and needed to be replaced.

The day in the orthopedics OR began as usual. During the first procedure, the surgeon asked the anesthesiologist, Dr. Pru, if the next patient was already called to the OR. Dr. Pru, a board-certified anesthesiologist, had taken the morning off and didn't know the patient. She promised to call for Mrs. Moore. The preoperative anesthesiologist had reserved a space on the ICU. He described Mrs. Moore as mentally oriented, but

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noncompliant. The following information was obtained:

- *85 years of age, height 160 cm, weight 54 kg, ASA III.*
- *Planned procedure: replacement of the ball and socket of the hip prosthesis.*
- *Previous anesthesia (in same hospital, 2 years ago): without incident, except for the moderate PONV.*
- *The patient had moderate dyspnea upon exertion after 1 flight of stairs; walking was not possible the past 4 weeks due to severe hip pain.*
- *ECG: atrial fibrillation*
- *Coronary heart disease (CHD) New York Heart Association class II, treated with Isordil (for the past 6 months) with subsequent alleviation of angina pectoris. Aspirin 100 mg held for the past 17 days.*
- *Echocardiography (3 months ago): Atrial dilation left and right, tricuspid and mitral valve regurgitation 2°, aortic valve regurgitation 1°, normal left ventricular function.*
- *No signs of congestive heart failure.*
- *Lab values: CRP and leukocytes slightly increased, all other values within normal range.*

Amoxicillin/clavulanic acid (Augmentan) 2.2 g IV was administered for endocarditis prophylaxis and midazolam 3.75 mg PO for preoperative sedation.

11.1.1 What Are the Risks for Mrs. Moore, and How Do You Estimate the Necessity of Postoperative ICU Care?

11.1.1.1 Risks

- Hemorrhage
- Hypothermia
- Advance age
- Cardiac history: coronary artery disease and aortic regurgitation

The replacement of both components of a hip prosthesis has a high risk of bleeding. Furthermore, the chance of hypothermia increases with the duration of the surgery. Mrs. Moore's age and cardiac history puts her at increased risk for perioperative complications. Securing a bed in the ICU is highly recommended.

11.1.2 Which Procedures, in Addition to Standard General Anesthesia, Would You Consider or Prepare for?

11.1.2.1 Monitoring Volume Status

The extent of the monitoring must account for a possible volume loss during the operation. The following types of monitoring are recommended perioperatively:

- Invasive arterial blood pressure
- Monitoring trends of the central venous pressure (CVP) via a central venous catheter (CVC)
- A measurement of urine excretion via a urinary catheter

11.1.2.2 Monitoring Cardiac Function

Due to Mrs. Moore's preexisting conditions, and the extent of the operation, careful monitoring of heart function is required. At a minimum, this includes:

- Invasive arterial blood pressure
- Monitoring trends of the CVP and
- ST segment monitoring with a 5-lead ECG

Since Mrs. Moore's preoperative evaluation revealed she was cardiovascularly stable and well compensated, additional cardiac monitoring is optional including:

- The perioperative monitoring of the cardiac function with TEE

11.1.2.3 Blood Transfusion, Crossmatched Packed RBCs

Surgical replacement of the ball and socket joint can be accompanied by significant blood loss. Therefore, a system for blood transfusion should be prepared. At a minimum, 4 units of packed RBCs must be ready for transfusion.

11.1.3 Are You in Agreement with the Perioperative Management of This Patient?

11.1.3.1 Endocarditis Prophylaxis

The latest guidelines for endocarditis prophylaxis, valid in the USA since May 2007, indicate perioperative endocarditis prophylaxis only in

high-risk patients in combination with procedures that have a high risk of causing bacteremia [4]. High-risk patients are defined as heart transplant recipients with valvulopathy, patients with previous history of endocarditis, patients with synthetic/ prosthetic heart valves, patients with inborn or palliative corrected cyanotic heart defects, or patients with corrected cardiac malformations and remaining defects after insertion of foreign material. Procedures with a high risk of causing bacteremia include dental procedures, procedures on respiratory tract or infected skin, skin structures, or musculoskeletal tissue. Mrs. Moore did not need any endocarditis prophylaxis.

11.1.3.2 Anticoagulation

Even when patients like Mrs. Moore would benefit from the current knowledge of oral anticoagulants, noncompliance is a contraindication. The patient, however, does have an increased perioperative risk of thrombosis, so that anticoagulation should be carried out with low molecular weight heparin in a therapeutic dose for this time period [6]. Mrs. Moore's preoperative lab work showed good renal function. Patients with renal disease should receive unfractionated heparin instead of low molecular weight heparin due to the danger of accumulation.

>> After assuring that there was a bed reserved on the ICU, Mrs. Moore was sent to the preanesthesia area. In the preanesthesia area, an arterial line was placed under local anesthesia. She was taken to the OR where anesthesia was induced with unremarkable intubation, and then a triple lumen CVC was placed in the right internal jugular vein. The first hour of the surgery went without incident.

Mrs. Moore maintained hemodynamic stability and adequate urine production at 0.5 ml/kg/h. Oxygenation, the electrolytes, and Hb values were normal in every arterial blood gas analysis. She bled about 600 ml, and she received about 2,000 ml of a crystalloid solution.

After 65 min, it was time to implant the new prosthesis. Suddenly her blood pressure dropped to 60/30 mmHg. The pulse dropped from 70 to 50 beats/min, and the S_pO_2 decreased from 100 to 70 %. "Good that I didn't let Jennifer go to help in the next room," thought Dr. Pru, as she was handed the first syringe. The CRNA (certified registered nurse anesthetist) Jennifer already had it ready in her hand.

11.1.4 What Is Your Treatment Plan?

The dramatic hemodynamic change requires immediate action. First, the blood pressure must be stabilized with:

- Administration of vasopressors or catecholamines
- Administration of volume
- An increase of the inspiratory oxygen concentration to 100 %

>> The immediate injection of phenylephrine made no difference whatsoever. The monitor showed a 0.5 mV ST segment depression in lead II, a 0.2 mV depression in AV_F, and no change in lead V5. Dr. Pru hooked up a pressure infusion with 1,000 ml Plasmalyte onto the IV and turned the inspiratory oxygen concentration to 100 %. "Call the anesthesiologist-in-charge," she said to CRNA Jennifer. "If she doesn't get better soon, we'll be doing CPR," she thought to herself. For the orthopedic surgeons, it was just another joint replacement.

11.1.5 What Do You Think Caused the Hemodynamic Change?

The cause is a reaction to the Palacos® bone cement. The reaction is induced by insertion of bone cement into the femur shaft. Typical symptoms include:

- Hypotension
- Heart rate fluctuation – usually an increase, sometimes a decrease
- Decrease in S_PO_2

Worldwide, the most commonly used cement consists of polymethyl methacrylate (PMMA). PMMA is mixed in the OR immediately before insertion into the bone. One substance to be mixed is a monomer MMA, which is liquid, and the other substance is a powder made of PMMA.

During or immediately after the cement is placed into the bone, the following side effects can occur [9]:

- Hypotension, all the way to cardiovascular collapse
- Anaphylactoid reaction
- Pulmonary embolus
- Pulmonary hypertension with right-sided heart failure
- Acute lung injury (ALI)

The simultaneously occurring pathophysiological processes are listed in Fig. 11.1.

The exact cause of the reaction to the cement is not fully understood. In the past, the monomer particles were thought to have direct cardiotoxic effects, when washed into the circulatory system. This theory is now believed to be unlikely. It is now known that MMA monomers stimulate the release of anaphylatoxins C3a and C5a, which cause histamine-mediated allergic reaction. To prevent this reaction, some authors recommend histamine blockade [8].

The most important trigger for a Palacos® cement reaction is probably pulmonary emboli. By using TEE in patients undergoing hybrid prosthesis surgery, 61.5 % of the patients were found to have had serious pulmonary emboli [5]. The observed micro- and macroemboli could have consisted of air, thromboembolus material, fatty bone marrow, or cement particles.

The result is acute increase of the pulmonary arterial resistance and the intrapulmonary shunt. Since the ejection fraction of the right side of the heart is tightly dependent on the myocardial filling pressure (Frank Starling Mechanism), the observed arterial blood pressure decreases are more severe when any of the following takes place:

- A volume deficit is present
- The ventricular filling mechanism – for example, in atrial fibrillation – is impaired

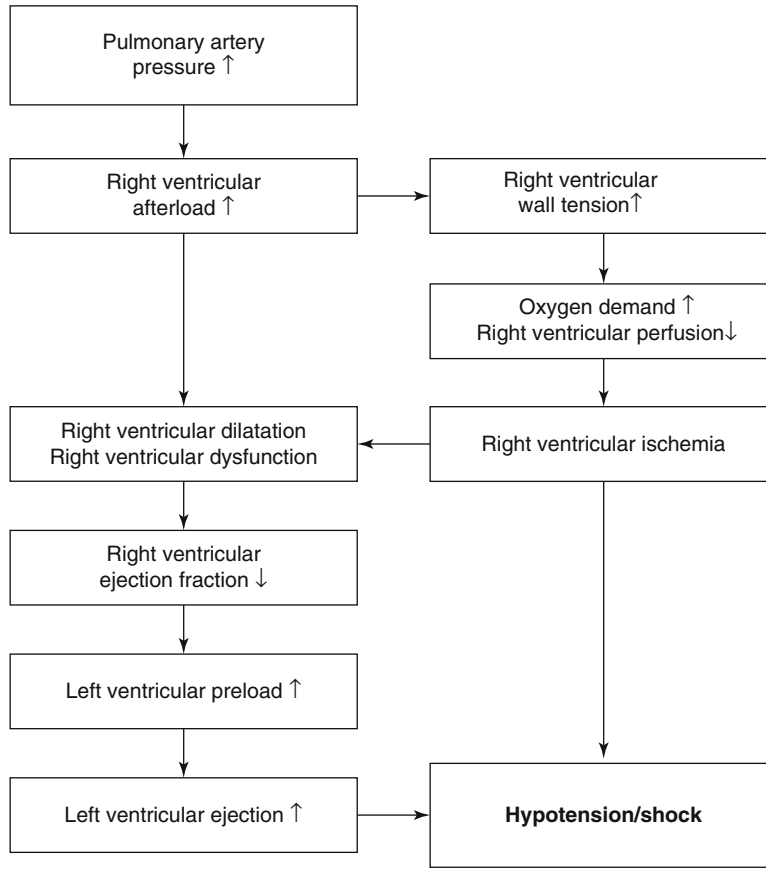

Fig. 11.1 Effects of a sudden increase of the pulmonary artery pressure on the cardiac ejection fraction

- The right-sided ventricular function is impaired

The extent of the Palacos® reaction is primarily dependent on the level of the intramedullary pressure in the shaft of the femur. The following measures are beneficial to prevent or reduce a Palacos® reaction:

- Waiting for polymerization of the bone cement monomers before inserting into the shaft.
- Using vacuum-operated mixers to minimize air bubbles within the PMMA cement.
- Copious washing out of the bone shaft before insertion of the cement.
- Airing/draining the shaft during the cementation.
- Slow, focused insertion of the prosthesis into the femur shaft.
- Balanced volume replacement and cardiovascular stability and prophylactic ventilation with 100 % oxygen are recommended.

11.1.6 How Will You Treat This Acute Situation?

The treatment is primarily symptomatic. Vasopressors and volume are administered to increase right ventricular filling. Sometimes, epinephrine or continuous infusions of another catecholamine are necessary. Increasing the inspiratory oxygen concentration to 100 %, or beginning assisted mask ventilation with 100 % oxygen during regional anesthesia, decreases the pulmonary arterial pressure and assures significant oxygenation.

>> CRNA Jennifer was now very nervous; the orthopedic surgeons could be heard hammering in the background. "What is wrong with the patient? Did she have a fat embolus?" she asked Dr. Pru. Jennifer reached the anesthesiologist-in-charge; he said he would come right away. She called other CRNAs to help, but they were busy with their patients at the moment. She questioned Dr. Pru further, "Should I do a blood gas analysis? Should I draw up more medications?" Dr. Pru declined the arterial blood gas as she had other problems to deal with at the moment.

11.1.7 Are You in Agreement with Dr. Pru's Decision? What Further Information Would You Obtain? How Is Fat Embolism Diagnosed?

Actually, a blood gas would have yielded important diagnostic information in this situation. As discussed in Sect. 11.1.5, the patient could have suffered from a fat embolus. The diagnosis of fat embolus is usually a clinical diagnosis, with consideration of the possible causes. The classic triad consists of:

- Respiratory insufficiency
- Neurological changes
- Petechiae on the upper extremities

Besides this, there are countless unspecific signs such as fever, tachycardia, renal failure, lab changes such as anemia, and coagulopathies. In serious cases, chest X-ray shows lung edema; the ECG may show signs of right heart strain. There are two theories for the pathogenesis of the so-called fat embolus syndrome.

11.1.7.1 Mechanical Theory

In the mechanical theory, it is assumed that large fatty drops cause blockage in the pulmonary arterial vessels and small drops may pass through the pulmonary circulation. This leads to microembolism in the central nervous system, the kidneys, and other organs, with the respective clinical signs and symptoms. The extrapulmonary signs could also be explained by a patent foramen ovale [1].

11.1.7.2 Biochemical Theory

The biochemical theory postulates the direct effects of the free fatty acids on the pneumocytes. Accordingly, hypoxia, hypertension, and the respective cardiopulmonary malfunction are explained.

A pulmonary embolus, besides increasing the pulmonary arterial pressure, also acutely increases dead space ventilation with an increase in the arterial alveolar CO_2 difference. Therefore, it would be interesting to know:

- The end-tidal CO_2 concentration in comparison to the arterial CO_2 concentration
- The comparison of the current CVP with previous values

In the presented situation, Dr. Pru must decide if she can manage without the presence of CRNA Jennifer. She decided correctly, because carrying out an arterial blood gas analysis requires additional personnel resources. The end-tidal CO_2 concentration and the CVP can be determined in the OR without additional effort.

>> The phenylephrine and the crystalloid infusion had no effect. Mrs. Moore's systolic blood pressure had now dropped below 30 mmHg; her heart rate was only 30 beats/min. "We must start chest compressions immediately, or Mrs. Moore will die," Dr. Pru announced loudly, so everyone could hear. The surgical team began immediate strong chest compressions. CRNA Jennifer prepared 1 mg epinephrine as soon as she heard the words "chest compressions." After 30 s, and the split administration of 0.3 mg epinephrine, the blood pressure improved. The anesthesiologist-in-charge Dr. Eldridge walked in the door just as 0.3 µg/kg min of norepinephrine was being started as a continuous infusion to stabilize the blood pressure. He affirmed Dr. Pru's decision. It took another 60 s before the S_pO_2 had normalized.

Four minutes later, an arterial blood gas sample was taken. The P_aCO_2 value was within the normal range, 44 mmHg, and also the oxygenation was reassuring, with a P_aO_2 value of 210 mmHg. The Hb value was 12.3 g/dl (Reference 11.9–17.2 g/dl). The ST segment analysis still showed pathological changes. The surgeon checked the stability of the socket prosthesis and decided against the replacement and concluded the surgery after just the replacement of the femoral head. "Whew!" thought Dr. Pru, "Good thing I didn't have to convince him of that!"

Half an hour after the cardiopulmonary resuscitation, the surgery was finished.

Mrs. Moore's body temperature was 34.9 °C. There were no further circulatory system fluctuations, and the total blood loss was about 800 ml. Mrs. Moore was transferred to the ICU, intubated and ventilated, with the norepinephrine infusion still running.

11.1.8 Why Did the Patient Go to the ICU Intubated and Ventilated?

The patient is hypothermic and still shows signs of myocardial ischemia. When anesthesia is stopped in a hypothermic patient, an increased oxygen requirement arises due to shivering. Shivering increases oxygen demand and therefore compensatory increase in cardiac output is needed which worsens cardiac ischemia. Extubation is contraindicated until the patient reaches a normal body temperature.

11.1.9 What Further Diagnostic Tests Are Recommended?

Immediately after the procedure ends, or at the latest immediately upon arrival in the ICU, the following should be done:

- A 12-lead ECG should be written
- A chest X-ray should be ordered
- Blood for lab tests should be drawn
- An echocardiogram should be ordered/performed

These diagnostic procedures are done in order to rule out or confirm a myocardial infarction or acute cor pulmonale. Troponin, CK, and CK-MB are the most important lab values. These tests must be repeated 2 h later. For differential diagnosis, D-dimers and proBNP values have a high priority in pulmonary embolism [7].

>> After the close call in the OR, Dr. Pru needed to breathe slowly and deeply for a minute. Three hours later, she went to the ICU to visit her patient. Mrs. Moore was still ventilated, but stable. The first troponin T value was in the normal range; the ECG did not show evidence of an acute myocardial infarction. Her body temperature was 35.0 °C, and hemodynamics were stable without additional catecholamines. The most recent hemoglobin value was 10.3 g/dl. Dr. Pru went home relieved.

The next morning, Mrs. Moore was awake and oriented. The endotracheal tube had been removed at midnight. Mrs. Moore was annoyed

that she had had to spend a restless night in the terribly noisy ICU. "My roommate on the ward had only partial sedation for her surgery, and she could go back to her room right after surgery!" she complained. The cardiac enzymes and the ECG were unremarkable in the follow-up tests. Dr. Pru explained to Mrs. Moore what had happened and was secretly very happy that her patient had returned to "normal," which meant noncompliant and dissatisfied with her care.

11.2 Case Analysis/Debriefing

11.2.1 Which Medical Errors Do You See in the Presented Case?

11.2.1.1 Aspirin Therapy

The risk of discontinuing aspirin must be weighed against the benefit of reducing the risk of bleeding. Discontinuation of aspirin therapy in patients should be considered only in those in whom hemostasis is difficult to control during surgery (class IIa recommendation). In less invasive procedures, preoperative discontinuation of aspirin requires interdisciplinary discussion, because interruption increases the risk of acute coronary syndrome. In high-risk procedures with anticipated significant blood loss, preoperative discontinuation of aspirin is recommended. But Mrs. Moore's aspirin intake was interrupted (100 mg held for 17 days) which was longer than necessary [2, 3]. As aspirin inhibits platelet cyclooxygenase irreversibly, a period of 7–10 days of withholding aspirin is usually sufficient to regain normal platelet function.

11.2.1.2 Optimizing the Conditions for the Placement of Palacos®

In high-risk patients undergoing general anesthesia, prophylactic ventilation with 100 % oxygen should be done before inserting bone marrow cement. Optimization of the volume status must also be accomplished. In high cardiac-risk patients, it is also desirable to have catecholamine infusions prepared.

11.2.1.3 Insertion of the Bone Cement

In Sect. 11.1.5, steps to reduce a reaction from Palacos® are listed.

11.2.1.4 Therapy for Cardiac Ischemia

With persistent ST segment changes, according to the guidelines, nitroglycerin should be administered continuously until an MI has been ruled out. Arterial blood pressure of >90 mmHg should be maintained.

11.2.1.5 Monitoring

In high-risk patients, continuous measurement of CVP measurement should be performed during surgery. Also, an intraoperative TEE would have been useful to diagnose causes of hypotension in this case.

11.2.2 Which Systems Failures Can You Find in the Presented Case?

11.2.2.1 Preoperative Evaluation

If possible, the anesthesiologist who will do the anesthetic should also do the preoperative evaluation for a complex case. If not possible for logistic reasons, the anesthesiologist should still thoroughly review the patient's medical record and perform a history and physical exam. With hospital process optimization and preanesthesia clinics, this is difficult to guarantee, especially with the tendency for anesthesiologists to work on shifts.

11.2.2.2 Resource Recruiting

The request for additional anesthesia providers failed. In emergency situations such as the one presented, one should not hesitate to recruit the help of non-anesthesiology employees, such as OR nurses or anesthesia techs, if for no other reason than to emphasize the urgent need for help.

11.2.3 Why Is Everything Always So Difficult for the Anesthesiologist?

Through the specialization of medicine, the unity between the various specialties seems to decrease. From an interdisciplinary point of view, this makes everything more difficult. For the orthopedic surgeons, it is a routine hip replacement; for the anesthesiologist, it is a high-risk patient. These different points of view lead to unequal amounts of information for each physician. Each specialty should have used a technique called the **shared mental model** (see Sect. 27.2.3). What should the surgeons have known?

The surgeons were not at all informed about the critical condition of their patient. The anesthesiologist did not convey any information; instead, she concentrated fully on the therapy of the life-threatening condition of the patient, without considering the treatment possibilities that the surgeons could have performed, such as reducing the pressure on the femur shaft.

Of utmost importance was the cooperative goal setting and task sharing that was shown during CPR. Dr. Pru informed the team loudly and clearly of the critical condition and the necessary action. By using the "we" form, she included all team players in the OR. The surgical team immediately began compressions, while CRNA Jennifer administered the emergency medications.

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Case 12: Foot Surgery

12

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12.1 Case Introduction

>> Dr. Leto had almost 3 years of experience in anesthesiology. As part of her residency, she was currently in orthopedics, a department that she enjoyed due to the many regional anesthesia techniques. Today was foot day. In her OR, there were 6 foot surgeries scheduled: one hallux valgus, one Charcot foot, and four diabetic feet. The hallux valgus procedure was first on the schedule. Dr. Leto had used general anesthesia with a LMA, which went without incident. Now Ms. Lee was in the OR.

Ms. Lee was 59 years old and suffered from Charcot–Marie–Tooth (CMT) disease, a diagnosis she received 30 years ago. Her legs were affected, and after so many years, her calves were very weak. She now had pes cavus, claw toe, and was dependent on crutches. Due to the pain, she had now decided to have surgery.

Dr. Leto didn't really have a full understanding of the CMT disease. Ms. Lee had been seen the day before in the preanesthesia clinic, and the anesthesiologist marked the preanesthesia evaluation with a red marker "trigger-free general anesthesia." Furthermore, he recorded the following:

- *60 kg, 160 cm*
- *Parkinson's disease, treated with levodopa 100 mg*
- *Paroxysmal atrial fibrillation and arterial hypertension, treated with 50 mg metoprolol per day*
- *Chronic gastritis with reflux esophagitis, treated with 20 mg pantoprazole*

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- *Chronic cervical pain syndrome*
- *Unremarkable ECG and lab values*
- *For premedication Ms. Lee received 7.5 mg midazolam PO and took her regular medications in the morning*

12.1.1 Is the Anesthesiologist's Note "Trigger-Free Anesthesia" Correct?

The question already implies it is not correct. CMT belongs to a group of hereditary motor and sensory neuropathies (HMSN) and is defined by symmetrical distal muscle weakness due to demyelination and loss of peripheral axons. The result is a reduced nerve conduction velocity and the loss of reflexes. The muscle weakness is the result of nerve deterioration and is not a symptom of a disease from the muscular dystrophy group. An increased incidence of malignant hyperthermia in CMT diseases has not been observed.

12.1.2 Which Muscle Relaxants Can Be Used with CMT Disease?

The most common complaint of CMT patients after general anesthesia is muscle weakness [1]. Since inhaled anesthetic agents also have muscle relaxation effects, avoiding these agents is justified. Patients with CMT disease suffer from chronic de-innervation of the peripheral muscles so that (as in bedridden patients) expression of postsynaptic acetylcholine receptors is increased. Therefore, the indications for use of succinylcholine are rather limited. Furthermore, the additional diagnosis of Parkinson's disease is a relative contraindication for succinylcholine. Non-depolarizing muscle relaxants such as atracurium [6], mivacurium [8], or vecuronium [2] are often used without incident. However, after the use of vecuronium, unexpectedly prolonged neuromuscular blockade has been reported [7].

>> Dr. Leto checked her little pocket anesthesiology book and found nothing about CMT disease. "We will do a trigger-free anesthesia," she said to CRNA Barbara, who was helping her in the OR. CRNA Barbara had already read the notes on the preanesthesia evaluation and prepared everything accordingly. For induction, Ms. Lee received 100 mg propofol, 150 µg fentanyl, and 30 mg atracurium IV. Ventilation via *face mask and intubation went without incident, and they were very careful not to overextend Ms. Lee's head, due to the C-spine problems. Dr. Leto started the constant infusion of propofol at 150 µg/kg/min.*

Shortly thereafter, the surgery began. Dr. Leto had increased the propofol infusion to 200 µg/kg/min before skin incision, even though she had the feeling that it was not even necessary. Ms. Lee didn't react to the surgical stimulus in the slightest. The extent of the procedure was significantly less than what was noted on the OR schedule; it only took 20 min. "Thank goodness I didn't give any more fentanyl," thought Dr. Leto, "or else it would have taken forever for her to wake up." She stopped the propofol infusion, and shortly thereafter, Ms. Lee began to breathe. "Ms. Lee," Dr. Leto said loudly to the patient, "Open your eyes!" Ms. Lee opened her eyes, but she didn't make any other movement.

12.1.3 What Would You Do Now?

The only reaction that Ms. Lee displays is eye opening and spontaneous respiration. From this, one cannot conclude that airway protective reflexes are present. Residual neuromuscular blockade needs to be excluded. Alternatively, one must rule out Parkinson's disease as the cause for the lack of movement.

>> Dr. Leto took out the peripheral nerve stimulator from the anesthesia cart and placed the electrodes on the area of the left ulnar nerve on the wrist. She chose a voltage of 50 mA and pressed the train-of-four button. Ms. Lee's hand showed no reaction whatsoever. Dr. Leto repeated the stimulation; this time putting her hand on Ms. Lee's fingers.

She was unable to detect a muscle response with this method either. "Maybe the machine is broken," she thought. She stuck two electrodes on her wrist and pressed TOF, but only with 20 mA. "Ugh!" she said, ripping it off, as her hand twitched. Then she reached for her telephone to ask for advice from her attending anesthesiologist Dr. Eldridge.

He came in shortly thereafter and repeated the measurement. To be safe, he checked Ms. Lee's other hand. The result remained negative. "If we don't even have one twitch, we can't reverse neuromuscular blockade," he said. "It's a shame that you did not use rocuronium. We could have used sugammadex, but time is not toxic," he added, "We will give Ms. Lee a little more time and let her sleep." Then he restarted the propofol infusion at 100 µg/kg/min and left the room.

12.1.4 Do You Have a Better Idea?

12.1.4.1 Judging the Depth of Relaxation

The cause a negative TOF test can be deep neuromuscular blockade. To determine this, a post-tetanic count (PTC) is recommended. PTC is performed by the application of a 50 Hz stimulus for 5 s followed by a pause of 3 s and then by 15 individual stimuli with a frequency of 1 Hz (Fig. 12.1). The number of positive muscle contractions correlates reciprocally with the depth of the block. With >10 positive single twitches, return of a positive TOF is expected immediately.

During nerve stimulation, it is important that the area of stimulation is warm enough, in order to receive a valid result. Ideally, skin temperature should be >35 °C and most certainly at least >32 °C.

12.1.4.2 Reversal of Neuromuscular Blockade

The most common medications used for reversal of non-depolarizing muscle relaxants are acetylcholine esterase inhibitors, most commonly neostigmine and edrophonium. They can be used if there is at least one twitch after the TOF stimulus, which is equivalent to a relative receptor block of about 90 %. It is, however, safer to wait until at least 25 % of the receptors have recovered, equivalent to a TOF of three or four.

A requirement for the evaluation of a valid TOF is that no PTC was performed 10 min prior to the TOF. Attending anesthesiologist Dr. Eldridge was therefore correct when he said that reversal of neuromuscular blockade could not yet be done.

Sugammadex is in Europe available for reversal of deep rocuronium blockade [3]. The medication is costly and therefore not routinely used.

>> Dr. Leto made herself comfortable next to Ms. Lee. The patient was breathing regularly and the end-expiratory CO_2 showed normocapnia. Every minute, she did a TOF measurement, always with the same negative result. Dr. Leto had often seen that atracurium worked an unexpectedly long time, but 80 min was a new personal record. She documented the last vital signs on the anesthesia record and suddenly everything started to happen very quickly. Ms. Lee moved her legs, sat up with a jerk, and attempted to extubate herself. As she fought to get her arms free, she yanked out her IV.

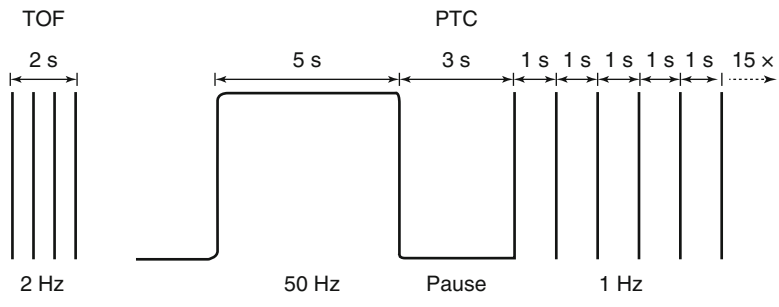

Fig. 12.1 Diagram of a train-of-four (*TOF*) stimulation and of a post-tetanic count (*PTC*). Various impulse strengths can be chosen from peripheral nerve stimulator

Dr. Leto jumped up, removed the endotracheal tube fixation, ripped off the cuff's external balloon, and extubated Ms. Lee. Ms. Lee promptly lay down again and went back to sleep. Dr. Leto stopped the propofol infusion and held an oxygen mask over Ms. Lee's face. "Strange," she thought, as she set off another TOF test, "still negative."

Ten minutes later, Ms. Lee woke up again and was transferred to the PACU. One hour later, she went to the ward.

The incident bothered Dr. Leto, so she went to see the patient the next day, with her nerve stimulator in her pocket. Ms. Lee was very happy with the anesthesia and couldn't remember a thing. The TOF measurement that Dr. Leto did once again was still negative.

12.2 Case Analysis/Debriefing

12.2.1 What Is Your Explanation for the Negative Nerve Stimulation?

CMT disease usually affects the lower extremities, especially the peroneal nerve. Neurophysiological tests show:

- A reduction of the nerve conduction velocity
- An extended latency period
- A reduction in action potential amplitude

Anatomically, axon degeneration often remains clinically unapparent for a long time. In the most serious cases with disease progression over many, many years, neurophysiological alterations on the upper extremities can be seen, especially in the area of the ulnar and median nerves. The result is that monitoring the muscle relaxation is with great difficulty or not possible at all [4].

12.2.2 Which Medical Errors Do You See in the Presented Case?

12.2.2.1 Peripheral Nerve Stimulation

The peripheral nerve stimulation is usually not a standard for monitoring muscle relaxation, even though many studies show that the percentage of PACU patients with residual neuromusclar blockade is substantial.

Note: A peripheral nerve stimulator is essential when patients with peripheral nervous system or muscular disorders receive muscle relaxants. In such cases, a measurement must be made before administration of muscle relaxants in order to minimize surprises, such as the one which occurred in this case. Please note that this also applies to all patients with secondary neuropathy, as a result of systemic diseases such as diabetes [5].

Checking the skin temperature was already discussed in Sect. 12.1.4.

12.2.2.2 Remarks Unrelated to This Case

Besides negative nerve stimulation, the necessity of higher voltage nerve stimulation in these patients is often difficult with regional anesthesia as well.

12.2.3 Which Organizational Weaknesses Can You Find in the Presented Case?

12.2.3.1 Trigger-Free General Anesthesia

As discussed in Sect. 12.1.1, it was not necessary to do a trigger-free anesthetic. When, however, "trigger free" is ordered from the doctor who evaluated the patient preoperatively, then it should be performed first on the OR schedule, after running air through the anesthesia machine all night. Appropriate algorithms for team communication and scheduling are necessary.

12.2.3.2 Train-of-Four Measurement/ Peripheral Nerve Stimulation

The value of nerve stimulation was already discussed in Sect. 12.1.4. The use of this monitoring technique as standard procedure must – at least in certain patient populations – be specified in anesthesia protocols.

12.2.4 What Could Dr. Leto Have Used to Recognize This Mistake Earlier?

Dr. Leto was correct to wonder about the negative nerve stimulation, and she began to look for the reason. Her first assumption was that the device malfunctioned, which was quickly ruled out. Then she came to the conclusion that there was an unexpected long effect of the muscle relaxant. Other possibilities, such as inadequate monitoring due to the neurophysiological changes, were not taken into consideration.

Her search for information was very selective and directed to her assumption – a device malfunction is very unlikely and can be ruled out immediately. Becoming fixated on such mistakes is always easier when past actions are not examined critically. Incorrect or inadequate reflected conclusions persist and sometimes multiply.

12.2.4.1 How Can This Be Prevented?

The goal is the critical questioning of previous actions, to catch details which were ignored – or to even uncover new information – to allow new conclusions to be formed with the new information. Often, verbalizing the problem helps, for example, "Why doesn't the nerve stimulator work now?" The out-loud verbalization helps to activate thought processes which can approach the problem from another angle. Simultaneously, other aspects of the problem are clarified. You can actively involve yourself in problem solving, and you will be indirectly ordered to do so.

Through such behavior, it is easier to keep alternatives in mind. You will realize this sooner if you do not make the mental mistake of being selective about your search for information.

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Case 13: Spinal Stenosis

13

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13.1 Case Introduction

>> On Wednesday morning, as Dr. Baldwin approached the OR changing room, Mr. Hill was waiting at the door. Mr. Hill had accompanied his wife yesterday for her preoperative evaluation and recognized Dr. Baldwin immediately. "Good morning, Dr. Baldwin. My wife is already in the preoperative area of the OR. Please take good care of her! She is all I've got!" Dr. Baldwin was uncomfortable with his comment, but he promised to take good care of her. Then he went in to get changed.

In the preoperative area, anesthesiologist assistant Sarah and her intern were hooking up the blood pressure cuff, ECG, and finger clip for the pulse oximetry. They were relieved to see Dr. Baldwin, because there were no veins to be found on either of the patient's arms. Even her feet were glossy, and only mini-veins could be seen through the thin white skin. "Good that you're here, doc," said Mrs. Hill bravely. "You'll find a vein, I am sure of it. I can withstand a lot of pain if you need to search a while." Dr. Baldwin replied, "Good morning, Mrs. Hill. Just give me a moment, please; I need to check some results."

Dr. Baldwin logged onto the computer to look up the information. He was especially anxious to see the result of the echocardiography, which he had ordered yesterday.

He now had the following information about Mrs. Hill:

- *77 years old*
- *Height 1.62 m and weight 60 kg*
- *Systemic hypertension*

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- *Hypercholesterolemia and hyperlipidemia*
- *In the history, shortness of breath after climbing one to two flights, for about 6 months. Then came the symptoms of spinal stenosis with gait instability, so she quit exercising*
- *S/P tonsillectomy as a child*
- *S/P breast cancer 10 years ago*
- *S/P smoking (25 pack years – abstinence for 15 years*)
- *Chest X-ray: no pleural effusion, beginning emphysema, widened cardiac silhouette, enlarged heart*
- *ECG: SR, LT, T segment depression in V_2–V_6, Sokolow index indicates left ventricular hypertrophy*
- *Echocardiography: EF ~55%, hypertrophic cardiomyopathy, diastolic disorder, abnormal relaxation, no evidence of significant valve aortic stenosis*
- *Abnormal lab values: Hb 10.5 mg/dl (reference 11.9–17.2 mg/dl), HCT 29 % (reference 37–47 %), creatinine 1.52 mg/dl (reference 0.70–1.20 mg/dl)*
- *Medications: captopril and a thiazide diuretic*

13.1.1 Which Problems Might You Anticipate in the Planned Spinal Stenosis Procedure?

13.1.1.1 Surgery in the Prone Position

The difficult patient access in prone positioning requires careful fixation of the endotracheal tube, careful positioning of the patient, and more than enough easily accessible intravenous lines. Patients are at higher risk of brachial plexopathy and ischemic optic neuropathy, especially in prolonged procedures.

13.1.1.2 Blood Loss

Blood can be significant in multilevel procedures with instrumentation for treatment of spinal stenosis due to degenerative causes. However, for one- or two-level procedures, especially without extensive instrumentation, significant blood loss is rare. However, for tumor surgery, blood loss is often massive and coagulation disorders can develop due to the extensive vascularization of the tumor tissue. Many tumors, for example, breast and prostate, have plasminogen activating factors [1]. The induced conversion of plasminogen to plasmin leads to activation of fibrinolysis. The fibrinolysis makes it easier for the tumor to infiltrate tissue, grow, and spread. A major release of plasminogen activating factors during tumor surgery can cause hyperfibrinolysis. Generally, these metastatic tumors are embolized in interventional radiology prior to spine stabilization.

In Mrs. Hill's case, it must be noted that she had anemia preoperatively. Sufficient packed RBCs must therefore be crossmatched in advance.

13.1.1.3 Hypothermia

It is difficult to passively warm patients in the prone position, and they tend to become hypothermic.

13.1.2 What Information Did You Receive from the Preoperative Evaluation?

13.1.2.1 ECG

The patient shows ECG signs of left ventricular hypertrophy, probably due to hypertension. The T segment depression in V_2–V_6 is a sign of a reduced coronary flow reserve.

13.1.2.2 Chest X-ray

Apart from emphysema, myocardial hypertrophy is present. In the posteroanterior (PA) view, the heart should not take up more than 50 % of the diameter of the bony thorax. The enlarged silhouette indicates hypertrophy of the left atrium.

13.1.2.3 Echocardiography

The pathological finding is enlarged wall thickness and diastolic disorder, indicating that Mrs. Hill has a diastolic cardiac dysfunction. Diastolic heart dysfunction is characterized by increased resistance to ventricular filling (Fig. 13.1).

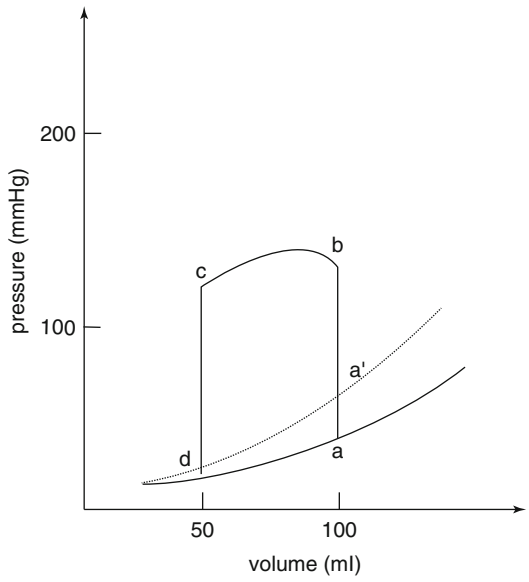

Fig. 13.1 Pressure–volume curve of the left ventricle

In diastolic heart failure, there is a relaxation disorder of the left ventricle, which causes diastolic pressure increases during ventricle filling (d → a) disproportionally (d → a′).

Diastolic heart failure is seen in the echocardiography as [5]:

- Slowed isovolumetric relaxation of the left ventricle, despite a normal ejection fraction (EF >45 %)
- Normal or low end-diastolic ventricular volume with a decreased early diastolic filling phase and decreased diastolic elasticity (left ventricular end-diastolic pressure >16 mmHg)
- Normal or decreased stroke volume
- Increased filling pressures with increased left ventricular stiffness, often stress induced

Differentiating between diastolic dysfunction and diastolic heart failure is necessary. In symptomatic patients, there is said to be diastolic heart failure, whereas asymptomatic patients are referred to as having diastolic dysfunction. Another term, which is used for diastolic heart failure, is heart failure with normal left ventricular ejection fraction (HFNEF) [13]. HFNEF requires the following conditions to be satisfied:

- Signs or symptoms of congestive heart failure such as pulmonary edema, ankle swelling, hepatomegaly, dyspnea on exertion, and fatigue
- Normal or mildly abnormal systolic left ventricular function
- Evidence of diastolic left ventricular dysfunction

Morphologically, there is a concentric hypertrophy of the left ventricle, as opposed to eccentric hypertrophy which is typical for systolic dysfunction.

Signs or symptoms of congestive heart failure in Mrs. Hill's case are difficult to evaluate due to her debilitating spinal stenosis and inability to exercise. During the preoperative evaluation, she needs to be thoroughly evaluated to assess whether she is in optimal condition for the planned surgery, in addition to obtaining informed consent for anesthesia. In Mrs. Hill's case, pulmonary edema needs to be ruled out by auscultation. In the chest X-ray, however, there are no signs of pulmonary congestion. If ordered, the lab values may show an increased proBNP level.

Risk factors of diastolic heart failure:

- Geriatric age
- Arterial hypertension
- Obesity
- Diabetes mellitus
- Obstructive sleep apnea syndrome
- Left ventricular hypertrophy
- Aortic stenosis
- Coronary artery disease

>> Dr. Baldwin now turned to Mrs. Hill. After he carefully checked all limbs and only got one small IV started in her left foot, he decided to place a CVC in the OR under local anesthesia prior to induction of anesthesia. Mrs. Hill gave informed consent. After taking Mrs. Hill to the OR, he made several unsuccessful puncture attempts of the internal jugular vein. Since he didn't want to make additional attempts on her neck, he decided to go for the subclavian vein on the right. He was successful on the second try! The Seldinger wire was inserted gently; a few extrasystoles allowed Dr. Baldwin to be sure that he had landed at the right place. Mrs. Hill

beamed up at him "That wasn't bad at all!" she said.

Then Dr. Baldwin began the induction saying, "Sarah, please administer 150 μg of fentanyl, then 70 mg of propofol." After Mrs. Hill was sufficiently anesthetized, she received 30 mg of atracurium. The next blood pressure was 68/35 mmHg. After a bolus of phenylephrine and 250 ml of a crystalloid infusion, the pressure increased to 87/54 mmHg, but then sagged again.

Anesthesia assistant Sarah pulled out the supplies for phenylephrine and vasopressin infusions and told the intern to prepare everything for invasive arterial blood pressure monitoring. Dr. Baldwin was having a hard time finding a pulse in the radial artery; he requested an ultrasound.

Finally, all invasive monitors were in place. The blood pressure, with the help of phenylephrine and vasopressin infusions, was 120/87 mmHg; the A-line was ready, the nasogastric tube in place, the eyes safely covered, and a urinary catheter placed. Anesthesia assistant Sarah had taken advantage of the vasodilation caused by the anesthetic agents to place a 16 g in the forearm. The anesthesia was maintained with 3.6 % end-tidal desflurane.

The orthopedic surgeon entered the OR: "Good morning; everything OK? Can we position the patient now? Did Mrs. Hill receive the antibiotic prophylaxis already?" The infusion with the antibiotic drip began, and Dr. Baldwin was happy that, despite the complicated induction, he was only 15 min late from the scheduled start of surgery.

13.1.3 Would You Have Chosen Another Procedure?

13.1.3.1 Invasive Blood Pressure Monitoring Before the Induction of General Anesthesia

Due to her comorbidities, Mrs. Hill should have received invasive arterial blood pressure monitoring prior to induction.

13.1.3.2 Placement of the CVC with Ultrasound Guidance

Since an ultrasound was available, it should have been used for the placement of the CVC. The ASA Practice Guidelines for Central Venous Access recommend use of ultrasound guidance for CVC insertion because it reduces complications and increases the success rate of internal jugular puncture [14].

13.1.3.3 Phenylephrine and Vasopressin Infusions Prepared Prior to Induction of General Anesthesia

Again, due to the Mrs. Hill's preexisting conditions, it should have been assumed that continuous administration of vasopressors would be necessary. With this in mind, a phenylephrine infusion should have been set up before induction. Given Mrs. Hill was taking captopril, an angiotensin-converting enzyme inhibitor, a vasopressin infusion should also have been prepared in advance, as phenylephrine alone may not restore blood pressure with this class of drugs. Vasopressin is very effective in treating perioperative hypotension in patients on renin–angiotensin system antagonists preoperatively [16].

13.1.3.4 Etomidate?

Many anesthesiologists prefer etomidate as the induction anesthetic agent in patients with significant cardiac disease, since there is less negative inotropic effect than with propofol.

13.1.3.5 Timing of Prophylactic Antibiotic Administration

Administration of intravenous antibiotic was started shortly before Mrs. Hill was positioned prone. This was inappropriate timing. Mrs. Hill had to be disconnected from the monitors just as the drip began. Allergic reactions causing circulatory changes could therefore not be detected as quickly. In addition, perioperative antibiotic prophylaxis should not be given more than 30 min before skin incision, in order to work most effectively. An exception to this timing is vancomycin, which is given by slow infusion beginning within 60 min of skin incision. In prone spine surgery, especially when neuromonitoring is placed after prone positioning, it is best to wait to start the antibiotic after the patient is prone.

>> After the team turned Mrs. Hill to prone position, and after the monitoring cables were quickly reconnected, the blood pressure was 78/43 mmHg

and heart rate 45/min, but it quickly increased to 95/min. The S_PO_2 was 94 %.

Dr. Baldwin doubled the vasopressin infusion, and his assistant Sarah hooked up 500 ml of 5 % albumin infusion. The surgeon asked why the alarm was sounding and asked if he could help. "*No, no,*" *said Dr. Baldwin,* "*the blood pressure will be better in a second.*" *And what do you know, the blood pressure increased to 105/65 mmHg. The student intern shyly asked Dr. Baldwin if Mrs. Hill might have just had a pulmonary embolus.*

13.1.4 How Do You Explain to the Student What Just Happened?

The cardiopulmonary changes could have been caused by the following.

13.1.4.1 Relative Hypovolemia Due to Positioning

Relative hypovolemia due to the prone position is often seen. It is influenced by preoperative hypovolemia, the vasodilation effect of the anesthetic agents, and the simultaneous lowering of the legs, which is often done in this position. Patients need to receive adequate volume before induction, and there must be sufficient intravenous access available. Correction of hypovolemia is especially true for patients taking ACE inhibitors and angiotensin receptor antagonists, as hypovolemia severely exacerbates hypotension with these drugs. In addition, patients should only be disconnected from the monitor for the shortest amount of time possible. Vasoactive substances must be prepared and ready to administer.

13.1.4.2 Cardiac Event

Myocardial ischemia or infarction can't be completely ruled out. The ECG and ST segments must be carefully monitored. The rapid response to the vasoactive substances and volume administration makes hypovolemia the most likely cause.

13.1.4.3 Pulmonary Embolus from a Preexisting Thrombosis

During the positioning, an embolus could have been dislodged from a thrombosis in the leg. In such an occurrence, a lowered end-tidal and relatively high arterial CO_2 concentration would have occurred, due to the increase in dead space ventilation (see Sect. 11.1.7).

13.1.4.4 Vagal Reflex in Conscious Patients

The patient received relatively little anesthetic agents, and the end-tidal desflurane concentration was under the 0.7 MAC value. Therefore, the patient could have returned to consciousness during the positioning and reacted by fighting against the ventilator and suffered a vagal reflex. Fighting the ventilator is unlikely, however, since sufficient muscle relaxant was given.

13.1.4.5 Stimulation of the Carotid Sinus by Improper Positioning of the Head

Stimulation of the carotid sinus with the accompanying vagal reflex is possible and requires checking the head and neck positioning.

13.1.4.6 Allergic Reaction to the Antibiotic

An allergic reaction to the antibiotic is an important differential diagnosis. Allergic exanthema is not always the most predominant symptom [3], nor is bronchospasm always present.

The immediate therapy to stabilize the hemodynamics is identical to treating hypovolemia. If the symptoms persist, anaphylaxis must be considered in the differential diagnosis.

As a rule, antibiotic administration in anesthetized patients should always occur with simultaneous vital sign monitoring. The immediate actions in case of an allergic shock would be to remove the trigger (stop the antibiotic infusion), give 100 % oxygen, titrate IV epinephrine (50 µg boluses) to hemodynamic effect, and give a rapid fluid bolus of 500–1,000 ml. Antihistamines and steroids are second-line treatment in anaphylaxis.

>> Dr. Baldwin explained to the intern that the most likely cause was relative hypovolemia due to the prone position. Myocardial ischemia was very unlikely because there were no additional ST segment changes. He recommended that the intern take a blood gas in order to rule out pulmonary embolism.

The ABG showed:

- *Hb*: *8.5 g/dl* (*reference 11.9–17.2 g/dl*)
- *HCT*: *26 %* (*reference 37–47 %*)

- P_aO_2: *272 mmHg* (*reference 70–100 mmHg*)
- P_aCO_2: *37 mmHg* (*reference 36–44 mmHg*)
- *BE*: *–1 mEq/l* (*reference ±2 mEq/l*)
- *Lactate*: *1.1 mmol/l* (*reference 0.5–2.2 mmol/l*)
- Na^+: *141 mEq/l* (*reference 135–150 mEq/l*)
- K^+: *3.7 mEq/l* (*reference 3.5–5.0 mEq/l*)

"*Oh*, *Sarah*," *said Dr. Baldwin*, "*Have you checked if the blood bank has crossmatched some bags for us*?" *His anesthesia assistant Sarah responded sheepishly that she had not yet had time to do so*. "*Then please call quickly*," *said Dr. Baldwin. The bags of blood were not yet ready. The ward nurse had been unable to draw blood yesterday*, *due to Mrs. Hill's bad veins. The blood would be ready in 25 min*. "*All right then*," *said Dr. Baldwin*.

The surgery began. At skin incision, *Mrs. Hill was given fentanyl 250 µg IV. Her blood pressure had stabilized*, *and the vasopressin infusion was discontinued*.

The next 2 h were unremarkable. Dr. Baldwin took some time to teach the student intern about differential diagnosis of sudden hypotension under anesthesia. In the meantime, *his assistant Sarah had drawn a new ABG. The values were only slightly different from the first blood gas*, *and Dr. Baldwin was satisfied. He shot a glance at the suction container*, *which only had about 350 ml of bloody fluid. Mrs. Hill's temperature was 35.8 °C*.

"*Oh*," *asked the student intern*, "*shouldn't we measure the CVP*?" *Dr. Baldwin replied* "*Yes*, *the absolute value won't tell us much at this time*, *but we can still measure it. The CVP was 6 mmHg. A CRNA came in to give Dr. Baldwin a break. I'll be right back*; *I just need a quick cup of coffee*." *He disappeared around the corner*.

13.1.5 What Does the CVP Indicate at This Time?

The CVP is an easily identified parameter. The CVP is defined as the pressure within the right atrium or within the vena cava as it enters the right atrium. Traditionally, CVP is measured with help from a central line, which is hooked up to a water column. The transition between the ventral to the middle third of the anterior posterior thorax diameter at the height of the sternum is used as a zero reference point (reflecting the right atrium/tricuspid valve). More information can be obtained with the help of a pressure transducer showing a central venous pressure curve (Fig. 13.2).

Fig. 13.2 Normal CVP waveform, characterized between two descents in pressure (x/x' and y) and three upstrokes (a, c, and v). The a wave reflects the pressure increase in the right atrium during atrial contraction. At the end of atrial contraction, the pressure decreases (x'). This decrease in pressure is briefly interrupted when ventricular contraction begins, and the tricuspid valve closes, resulting in an upstroke (c wave). As the ventricle contracts, it moves toward the apex with a correspondent downward movement of the tricuspid valve, resulting in a further decrease of the CVP (x). The filling of the right atrium as the tricuspid valve is still closed causes the v wave. The second pressure decrease (y) begins with the end of ventricle contraction, followed by opening of the tricuspid valve (Hübler [9] with permission)

The CVP is used as surrogate parameter for the cardiac preload. In the CVP curve, this point in time is just before the ventricle begins to contract, in other words, the curve before the c wave. This point is marked as the z-point (Fig. 13.2), which is usually hard to identify.

In this case, the horizontal part of the curve before atrial contraction starts (before the a wave) is taken as a reference point.

In volume-depleted patients, the CVP is especially sensitive to intrathoracic pressure changes. It is therefore standard to read the value at the end of expiration.

The CVP curve offers not only the possibility to exactly determine the CVP value, but more importantly it allows changes in the waveform to be assigned to specific pathophysiologic conditions (Table 13.1) [11].

An "abnormal CVP waveform" should not put the value of the CVP tracing into question. The interpretation should always take into consideration the underlying pathology. Possible influencing factors – apart from those listed in Table 13.1 – include atrioventricular dissociation, asynchronous atrial contractions during ventricular stimulation with a pacemaker, tricuspid

Table 13.1 Meaning of changes in the CVP tracing

Waveform changes	Possible cause
Loss of the *a* wave	Atrial fibrillation
Large *v* waves	Tricuspid regurgitation
Very deep *y* decrease	Volume deficiency
Loss of *y* decrease	Cardiac tamponade

stenosis, pulmonary hypertension, and right-sided heart failure.

The measurement of the CVP is based on the theoretical concept that pressure and volume are related. Therefore, CVP is viewed as a measurement of intravascular volume, i.e., of cardiac preload. Basically, this correlation naturally exists, but unfortunately, the Boyle–Mariotte law is only applicable for ideal gases and not for fluids (blood) in elastic vessels. Various studies show that the correlation between the absolute CVP value and the intravascular volume is minimal [10]. The absolute measured value, therefore, has little significance even if it is in normal range. Variation of systolic blood pressure and pulse pressure during positive pressure ventilation is predictive of hypovolemia and responsiveness to intravascular fluid administration. During positive pressure ventilation, right ventricular stroke volume is decreased during inspiration with decreased venous return. Subsequently, the left end-diastolic volume is reduced during expiration. These effects are exaggerated during absolute and relative hypovolemia [12].

Nevertheless, changes in CVP over the perioperative time period can show a tendency to volume deficiency/volume overload/heart failure. Most useful are the values before induction in the supine position, as compared to postoperative value. Also helpful are repeated measurements or, better yet, a continuous measurement in high-risk patients.

"*>> Everything goin' OK*?" *Dr. Baldwin asked as he returned to the OR*. "*I think so*," *answered the CRNA*. "*The procedure shouldn't take too much longer. I just gave 150 μg of fentanyl*." *Dr. Baldwin replied*, "*Shouldn't be a problem*," *and he stood on his tiptoes to look over the sterile drapes. The surgery was finished 30 min later. During the last stitches, the blood pressure dropped again. The arterial pressure curve showed 68/45 mmHg and heart rate 91 beats/min*.

"*Why are the drapes hung so high*?" *asked the student intern, as the sterile drapes were being removed. Dr. Baldwin mumbled*, "*Later…*," *and he asked the anesthesia technician to bring the bed into the OR. As more sterile drapes were removed, two ECG electrodes were ripped off*. "*We want to roll the patient onto the bed now anyway*," *said the surgeon*. "*With this low blood pressure, I don't know if that's such a good idea*," *protested the anesthesia assistant Sarah. But Dr. Baldwin nodded to the orthopedic surgeon, turned the vasopressin back on, disconnected the monitor, and gave the sign to turn the patient. With great effort, Mrs. Hill was rolled over to her side and into bed. Sarah worked quickly to hook up the monitors again, as Dr. Baldwin reconnected the ventilator. The arterial measurement showed a blood pressure of 30/20 mmHg and heart rate of 110 beats/min, and the* S_PO_2 *was no longer measurable*.

Dr. Baldwin glanced quickly at the blood soaked abdominal swabs hung up across the OR. Then he ordered anesthesiologist assistant Sarah to double the vasopressin infusion and restart the phenylephrine and to check to be sure the IV hadn't become disconnected during the turn. To the student he said, "*Draw up 1 amp of epinephrine in 10 ml, but first call the anesthesiologist-in-charge at 3-335. He should come immediately and send extra help. Say it's a CODE*." *As he pressure infused a bag of PlasmaLyte, he ordered Sarah to call up the RBCs from the blood bank. Tell the blood bank that they need to crossmatch an additional four bags*!" *The orthopedic surgeon interrupted*, "*Should I start CPR*?" "*Wait 10 s – the pressure is coming back*," *answered Dr. Baldwin. And sure enough, it increased, even though the systolic remained below 80 mmHg. Another nurse arrived, with blood in hand*: "*These are for you. Should I hang them*?" "*Yes, please check them and then administer under pressure, please*," *answered Dr. Baldwin. Then he called the anesthesiologist-in-charge to cancel his request for additional help*.

The blood pressure slowly rose to 130/80 mmHg. The oxygen saturation was measureable again, and showed 95 %. The heart rate remained about 110 beats/min. Anesthesia assistant Sarah had

already hung another bag of PlasmaLyte, this time without pressure. The first unit of packed RBCs was in and the arterial pressure was now 180/95 mmHg. Dr. Baldwin stopped the vasopressin infusion and turned down the phenylephrine infusion. "Can we help anymore? Can we go now?" asked the orthopedics team. "One moment, please" replied Dr. Baldwin. In silence he recapitulated the facts once more.

"What's your estimated blood loss?" asked Dr. Baldwin. "Well, we suctioned hardly anything, maybe 300 ml. The amount in the compresses is hard to say, maybe half a liter," answered the surgeon. "Dr. Baldwin?" interrupted his assistant Sarah. "One moment please, Sarah." "Did you irrigate a lot?" he asked the surgeons. "Yeah, about a liter" they answered.

"Dr. Baldwin?" interrupted Sarah again. "The pressure is now over 200!" Dr. Baldwin looked at the monitor and promptly shut off the phenylephrine. Mrs. Hill had now received two units of packed red blood cells, 500 ml of 5% albumin, and 2,500 ml of PlasmaLyte solution.

Dr. Baldwin turned the desflurane off. The student checked CVP once more. "It is now 18 mmHg. But we did reposition her." Together they waited for Mrs. Hill to wake up. The arterial pressure stayed over 190/95 mmHg; the heart rate was about 120/min.

Five minutes later, Mrs. Hill was awake and was extubated. She appeared a bit tired and had labored breathing. She responded to voice commands and could move her legs. Dr. Baldwin decided to take her to the PACU which was only 30 m away. Anesthesia assistant Sarah hooked up the monitor for transport, and placed an oxygen mask on Mrs. Hill's nose and mouth. Even so, the oxygen saturation was only 91%.

Upon arrival in the PACU, Dr. Baldwin took his stethoscope from his pocket and listened for breath sounds; he heard rales on both sides. Mrs. Hill continued to have labored breathing; her blood pressure was now between 90 and 85 mmHg systolic and 40 and 45 mmHg diastolic. Her heart rate increased to 130 beats/min. The PACU attending Dr. Finn came to the bedside and said, "Looks like hypovolemia. She probably just bled a little more than…" Dr. Baldwin turned beet red and began to shake in anger.

13.1.6 What Do You Think the Problem Is?

The most probable cause is volume overload, caused by the overly excessive and speedy replacement for the blood that was lost, followed by cardiac failure and pulmonary edema in a patient with preexisting diastolic cardiac heart failure.

13.1.7 How Can You Be Sure of This Diagnosis?

What is now necessary, in order to confirm this diagnosis:

- Arterial blood gas analysis
- CVP measurement
- ST segment analysis/12-lead ECG
- Possible chest X-ray
- Echocardiography

13.1.8 What Are the Most Important Differential Diagnoses?

The differentials to consider include:

- Transfusion reaction
- Acute coronary syndrome
- Pulmonary emboli and accompanying heart failure

>> *"No, I don't think so," said Dr. Baldwin to the young PACU doc. "If you take a look at her neck veins, you'll see that you shouldn't give Mrs. Hill another drop. Let's do an ABG, and call radiology for a chest film, and please get me a 12-lead ECG as soon as possible."*

"Then we could just measure the CVP," responded Dr. Finn in a less than friendly manner. "Have you even got an ICU bed?"

"*No*," *confessed Dr. Baldwin*, "*I'll get something on the intermediate care ward*."

The ABG showed, *in addition to hypoxemia and hypercapnia*, *a hemoglobin value of 9.5 g/dl* (*reference 11.9–17.2 g/dl*). *The CVP was at 20 mmHg*, *and the chest film showed acute pulmonary edema. The ECG showed no signs of an acute myocardial infarction*, *but there were ST segment depressions anteriorly*.

13.1.9 What Would Your Course of Therapy Now Be?

The basic principles of acute cardiovascular decompensation in diastolic heart failure include the following [15].

13.1.9.1 Provide Sufficient Oxygen

The first priority is increasing the inspiratory oxygen concentration. Simultaneously, the use of a CPAP mask should be considered as CPAP ventilation decreases preload (see below) and afterload.

13.1.9.2 Decrease Preload

An acute preload decrease can be achieved by administering a loop diuretic.

Furosemide is one of the medications given as initial therapy of pulmonary edema. The first clinical changes occur within 10–15 min; this is before a diuretic effect is seen [4]. The effect is caused by pulmonary arterial dilation [7], which decreases the diastolic pressure. The cause of dilation seems to be a direct effect on endothelial kinins, nitric oxide, and prostacyclins in the veins [17]. The acute decrease of the pulmonary arterial pressure by furosemide can also lead to a reduction of the hepatic portal pressure, which leads to a decrease in the CVP. A prerequisite of this effect is an increased pulmonary arterial pressure before administration of the loop diuretic.

Alternatively, the preload can be decreased by continuous administration of nitroglycerin, which is sometimes necessary. Morphine also decreases preload.

13.1.9.3 Decrease Afterload

The afterload should only be decreased when the patients are hypertensive during acute decompensation. This is usually the case, but not with Mrs. Hill at the moment [6]. Continuous administration of nitroglycerin is the first choice. Nitroprusside is an alternative treatment, but not appropriate in this circumstance.

13.1.9.4 Rhythm and Rate Treatment

The classic trigger for acute decompensation in diastolic heart failure is tachycardia, as the reduced compliant ventricle cannot properly fill during shortened diastole.

Tachycardia from atrial fibrillation leads to decompensation faster than sinus tachycardia, since the active ventricle filling from atrial contraction ceases. Rhythm and rate control can be achieved with β-blockers or calcium antagonists. In emergency situations, a synchronized electrical cardioversion is indicated for atrial arrhythmias.

13.1.9.5 Administration of Positive Inotrope Drugs

The administration of positive inotrope agents is the last therapeutic option, and should be used with care, since the increased myocardial energy requirement can lead to further decompensation and the systolic function is often only slightly, if at all, reduced. One may consider β-agonists and phosphodiesterase inhibitors. Note that an increase in heart rate should be avoided.

>> Dr. Baldwin administered 20 mg furosemide to Mrs. Hill and requested the PACU nurse to place a CPAP mask. Then he injected 3 mg of metoprolol IV. The heart rate dropped to 100/min, *without a further decrease in blood pressure. He decided to begin continuous administration of dobutamine IV*.

Gradually, *Mrs. Hill began to improve. Immediately after transfer to the intermediate care ward*, *a transthoracic echo was performed*, *which confirmed the suspected diagnosis of acute*

decompensation and diastolic heart failure. Through the night, Mrs. Hill's heart was supported with dobutamine; the next morning she was stable, with a balanced volume status, no catecholamines, and eupnea.

Two days later, Dr. Baldwin received a thank you note from Mr. Hill and a box of chocolates. Dr. Baldwin wasn't pleased about receiving a gift.

13.2 Case Analysis/Debriefing

13.2.1 Which Medical Errors Do You See in the Presented Case?

13.2.1.1 Invasive Blood Pressure Measurement Before Beginning General Anesthesia

Due to Mrs. Hill's medical conditions, one should have expected hypotension after induction. Placement of an arterial catheter allows constant observation of the changes and was therefore indicated.

13.2.1.2 CVC Placement Without Ultrasound Guidance

In the presented case, an ultrasound was available. Dr. Baldwin had used the device to place the arterial line. There was no reason for him not to use it for CVC placement, which would have increased the chance of success and reduced complications [2]. The ASA guidelines recommend use of ultrasound guidance in placement of internal jugular CVCs [14].

13.2.1.3 Monitoring the Volume Status

In Sect. 13.1.5, the value of CVP monitoring for volume status changes was discussed. After the operation had been in progress for 2 h, one ABG was done, and the values were only slightly different from the first measurement. Even when blood loss is difficult to estimate, one must assume that Mrs. Hill didn't receive enough volume replacement. A hematocrit reduction, however slight, was to be expected.

13.2.1.4 Time Point of Antibiotic Administration

This aspect was already discussed in Sect. 13.1.3.

13.2.1.5 Excessive Volume Administration at the End of the Operation After a Change in Position

In the presented case, there was a life-threatening drop in blood pressure after insufficient administration of volume during the surgery. In the ensuing emergency situation, uncontrolled amounts of various fluids were given. The excessive fluid administration led to cardiac decompensation with pulmonary edema, due to the diastolic heart failure.

In patients with diastolic heart failure, it is important to watch out for:

- Sufficient perfusion pressure
- Avoidance of critical anemia
- Isovolumetric volume substitution

13.2.1.6 Extubation

Mrs. Hill was still in an unstable condition after transfer to supine position and after the ensuing medical intervention. It was improper to extubate this patient, as she was hypertensive and had a heart rate of 120/min.

13.2.2 Which Systems Failures Can You Find in the Presented Case?

13.2.2.1 Administration of Antibiotic

Prearranged antibiotic administration (when and what to give) is recommended in the perioperative infection prophylaxis. A preoperative discussion of the antibiotic choice and appropriate timing between the surgical and anesthetic teams did not seem to have occurred.

13.2.2.2 Checking the Availability of Packed Red Blood Cells

Checking to make sure that the blood is ready for pickup should be set as a standard procedure as well. It is recommended to establish perioperative checklists [8].

13.2.2.3 Leaving an OR During a Problematic Anesthestic with an ASA III Patient

This aspect will be discussed in Sect. 22.1.1. It was inexcusable for Dr. Baldwin to have left the OR at

Table 13.2 Motivation = value · expectation?

Consequences	Expectation	Value	Motivation = value · expectation
What are the possible consequences if Dr. Baldwin neglects to conduct proper monitoring?	How likely is it that this consequence will occur for Dr. Baldwin? (%)	What value does this consequence have for Dr. Baldwin? (scale 1–10)	
Less work	100	High = 8	800
More time	100	High = 9	900
Loss of his position as a role model	Unlikely, since he feels that he is an expert ~10	High = 10	100
Not following standard procedures	100	High, because he considers himself an expert = 10	1,000
Overlooking a volume deficiency	Unlikely, because he feels that he is an expert ~5	High = 10	50
Inadequate volume therapy	Unlikely, because he feels that he is an expert ~5	High = 10	50

that time for a nonessential break. He should have requested that the CRNA come back at a different point in time or skip the break altogether.

13.2.3 Motivation = Value · Expectation?

During the anesthesia, Dr. Baldwin neglected to properly monitor the volume status and the blood loss. This was a clear case of motivation deficiency.

Neglecting to follow up is very high on the list of most common mistakes in medicine. Why aren't important parameters followed up? What was the motivation of Dr. Baldwin? Why did he feel that the extra effort of measuring the volume was not worth it? What did he achieve by neglecting to measure?

When looking at it from a motivation point of view, people decide for or against an action based on three questions:

- What are the *consequences*?
- How likely is it that this consequence will occur (= *expectation*)?
- What *value* does this consequence have for me?

Let's answer these "volume monitoring" questions for Dr. Baldwin (Table 13.2).

The motivation for or against volume monitoring results from the likelihood (2nd column) and the value placed on it (3rd column). The highest motivation values (4th column) for Dr. Baldwin were for the following consequences: less work, more time, and deviation from the rules.

Maybe you're asking yourself now, what does this have to do with you? Motivation is especially triggered by a sense of probability. Perceived probabilities are extremely subjective and very likely completely incorrect. They often have no relation to the actual state of affairs. Do not trust your internal probability sensor!

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Case 14: Septoplasty

14

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14.1 Case Introduction

>> Dr. Pia was annoyed. It was Friday afternoon, shortly before quitting time, and she was sent to do a preoperative evaluation. Actually, she had planned to leave the hospital at 4 p.m. on the dot and set off on a weekend trip to the beach with a friend. "I bet my attending won't be working late," she mumbled to herself.

There was a 12-year-old waiting with her mother. At the last minute, the ENT surgeon had scheduled the girl's surgery for the first case on Monday morning. The preanesthesia clinic staff had just left for the weekend. "What crappy organization," thought the annoyed Dr. Pia. "I'll have to work this all out by myself. I hope it doesn't take too long."

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She disappeared into an exam room to review the medical record. The patient's name was Olivia. On Monday she was scheduled for a septoplasty due to trouble breathing through her nose. In an outpatient surgery 1 year ago, she had some complications. The report didn't have any other details. "I'd better ask the mother; she'd know what happened," thought Dr. Pia as she walked to the waiting room. "Olivia, please!" she called out, before leading patient and mom into the exam room.

Olivia – a pale petite girl – and her mother entered the exam room. Dr. Pia introduced herself and clarified the purpose of their preanesthetic visit. "During the last anesthesia, there seems to have been a problem. Do you know what that was?" she asked.

"No, I don't exactly know," said the mom. "However, I brought this." She pulled out a worn yellow patient information ID card and an even more worn patient report. Dr. Pia read each carefully. On the yellow patient information ID, a propofol and midazolam allergy was noted. The ID card was signed by a pediatric allergist. It seems that during her previous surgery, Olivia had serious cardiovascular difficulties, aspirated gastric contents, and developed pulmonary edema, which required that Olivia be admitted to the hospital. Therefore, the ENT surgeons had decided to schedule the septoplasty as an AM admit procedure.

Olivia could not remember much from the last hospital stay. "Do I have to go to the ICU again? Will I have to stay for such a long time in the hospital?" Dr. Pia tried to calm her "No, there's nothing to worry about. We now know what medications were problematic for you, and we can avoid those medications. Are you otherwise healthy? What do you do for exercise? Do you take any medication on a daily basis?"

"Olivia is on the regional table tennis team. That's the reason it was so shocking for us last time. She is so healthy – hardly ever sick. She does not take any medication," said her mother.

Dr. Pia was not sure if she should order premedication. Midazolam was standard; however, since an allergy was reported, it was better to be safe and not order anything.

14.1.1 What Do You Think of the Decision Not to Order Premedication?

This decision is wrong. The child is traumatized by her experiences during her last surgery and ICU stay and is therefore very anxious.

14.1.2 What Is the Point of Premedication?

The main goals are:

- Sedation and reduction of fear – Fear activates the sympathetic nervous system, which can cause hypertension and tachycardia. In addition, sympathetic nervous system activation delays gastric emptying and increases gastric acid secretion.
- Amnesia.
- Prevention/minimization of postoperative vomiting and its associated risks.
- Analgesia – Analgesia is usually indicated when painful positioning is required, for example, in unstable fractures or when patients have extreme pain due to their condition and therefore have an increased sympathetic activation.
- Vagolysis (inhibition of parasympathetic nervous system) – In special circumstances, inhibition of salivary and bronchial secretions and prevention of vagal bradycardia can be beneficial.
- Possible histamine blockade when allergic reaction is likely.
- Possible aspiration prophylaxis/alteration of the composition of stomach acid.

>> In an attempt to discuss premedication, Dr. Pia phoned her attending. "Probably already gone for the weekend," she thought. Sure enough, no one answered the phone. "Olivia is almost an adult; she can handle the situation without premedication," thought Dr. Pia, who then informed Olivia and her mother about the anesthesia and possible complications. They felt well enough informed, and her mother signed the consent form. They said goodbye and headed for home. Dr. Pia was excited that her beach weekend was finally beginning!

14.1.3 What Medications Are Indicated as Premedication, and Which Ones Would You Have Ordered?

14.1.3.1 Benzodiazepines

Benzodiazepines are the most common group of medications to be used for premedication.

Effect: Anxiety reducing, calming, sedating, hypnotic, amnestic, muscle relaxing, and anticonvulsive.

Benefits: Minimal toxicity within a broad therapeutic range, minimal hemodynamic or respiratory side effects, and safe in malignant hyperthermia.

Disadvantages: Lack of analgesic effect, relatively long acting, duration of effect extended in liver disease and older patients, and occasional paradoxical excitation reactions.

Interactions: In chronic alcohol abuse, tolerance is expected; however, in acute alcohol intoxication, the central sedative effect is increased. Benzodiazepines decrease the MAC value of volatile anesthetics and decrease the requirement of intravenous anesthetics for induction of anesthesia.

Contraindications: Acute alcohol, opioid, or sedative intoxication, myasthenia gravis, ataxia, and known allergy.

The choice of benzodiazepines is based on the desired onset and duration of action. The substance most commonly used is midazolam, with a rapid onset of action and elimination. The substance can be given oral, IM, IV, and, in pediatrics, rectal or nasal (see Sects. 6.1.5 and 28.1.3). Midazolam is water soluble, whereas diazepam is not; therefore, injection of midazolam is not painful. Given orally, 50 % of the dose undergoes a first-pass effect. Midazolam is metabolized to 1-α-hydroxymidazolam and to 4-hydroxymidazolam, which contribute to about 10 % of the pharmacological activity of midazolam. Both metabolites have short half-life (~1 h), and although they are pharmacologically active, they do not prolong the duration of action of midazolam.

14.1.3.2 Barbiturates

The advantages of midazolam have drastically reduced the use of barbiturates. Only phenobarbital is rarely used.

Effect: Sedating, hypnotic, and anticonvulsive.

Benefits: Respiratory and cardiovascular effects minimal when given orally.

Disadvantages: Nonspecific central nervous system effects, a more restricted therapeutic range than benzodiazepines. When given in the presence of pain: excitation and confusion.

Contraindications: Acute hepatic porphyria.

14.1.3.3 Neuroleptics

Neuroleptics cause psychomotor retardation, emotional liability, and effective indifference.

The butyrophenone droperidol might be used for adult premedication and is usually combined with an opioid. There was a time when it was used for neuroleptic analgesia, but it has been abandoned today. FDA issued a black box warning for patients with QT prolongation, as some patients developed torsades de pointes following administration of droperidol. An ECG, to rule out preexisting long QT syndrome, is required prior to administration of droperidol.

The phenothiazine promethazine is extremely sedative, hypnotic, anticholinergic, antiemetic, and histamine antagonistic. There is no effect on anxiety, a major disadvantage that has limited its use.

14.1.3.4 α_2-Agonists

Representative of this class are clonidine and dexmedetomidine. They are analgesic, sedating, and decrease the necessity for analgesics by up to 40 %. They can reduce postoperative shivering but have no effect on anxiety.

>> The Allergy Patient Information reported a midazolam allergy. Allergies against benzodiazepines are very rare. There is nothing known about cross-over reactions; even so, the entire class should be avoided. A possible alternative is promethazine. A benefit of this choice is the antihistamine effect, with the patient predisposition to allergies.

On Monday morning after the team meeting, the attending anesthesiologist Dr. Hardy appeared in the preoperative area of the OR. He was assigned to provide anesthesia for Olivia. Dr. Hardy had been an attending for about a year now, and his career path hadn't always been easy. He was annoyed that he still had to perform

and provide solo anesthesia at 08:15, in this case with the patient Olivia. He felt that an attending should be handling more important duties and not be stuck with the work of a resident/CRNA.

Anesthesia tech Ann hooked Olivia up to the monitors in the OR. She had read the preoperative evaluation and didn't really know which medications to check out from pharmacy. "This is not going to be a standard induction," she thought to herself. "Dr. Hardy has his own ideas about everything anyway." She had known him for years and had watched his development from a first-year resident to an attending. It had been clear to her from the beginning that he would succeed in his career.

Dr. Hardy entered the OR and greeted anesthesia tech Ann and the very nervous Olivia. Then they began to study the patient's medical record. He reviewed Dr. Pia's preoperative evaluation and thought to himself, "Oh, it was not a good idea to deny the child a sedative; I will have to discuss it with her." Then he found a detailed patient report from the last hospital admission which occurred after the planned outpatient surgery. Dr. Pia hadn't seen this report when she had performed the preoperative evaluation.

The following events were documented in the letter:

Admission was necessary after an elective ambulatory surgery. Olivia had received 5 mg midazolam PO. During the induction, severe bradycardia developed with a heart rate of 38 beats/min. Atropine increased the heart rate to 130 beats/min. Anesthesia was induced with propofol, alfentanil, and an unspecified muscle relaxant. Intubation was unremarkable. During the course of events, Olivia vomited, and yellow fluid was seen in the endotracheal tube.

"All right, Olivia aspirated. The cuff was probably not sufficiently inflated. There is no other explanation for the yellow fluid in the tube," thought Dr. Hardy. Olivia had been extubated at the end of the procedure. Her oxygen saturation was 70%, so she was given 4 l/min O_2 via *nasal cannula and transported to the nearest hospital. The hospital preformed extensive diagnostic tests. A chest X-ray showed edema of the pulmonary parenchyma on both sides, especially in the upper lobes.*

"This makes no sense," thought Dr. Hardy. "Aspiration most often affects the right lower lobe." The pulmonary function tests revealed a moderate restrictive ventilation disorder. The echocardiography showed minimal pulmonary hypertension. The hospital stay was unremarkable, apart from a fever spike, which was treated with acetaminophen (Tylenol). No antibiotics were given. Olivia received additional oxygen for 12 h. A skin prick test was negative for propofol and pollen.

14.1.4 What Is a Prick Test, and What Conclusions Can Be Drawn from a Negative Prick Test?

The prick test is subcutaneous and elicits a type I allergic response, such as sensitivity to pollen or animal dander. A defined allergy extract is dropped on the skin, then scratched with a lancet, so that the substance enters the skin. The test can be read after 20 min and compared to 2 test pricks. A positive control prick is done with histamine, and a negative control is done without an active substance. The redness and swelling is analyzed. An allergy against the substance is not ruled out by a negative test; however, a positive prick test does prove the existence of an allergy [7].

>> In the patient's medical record, the pediatric allergist came to the conclusion that the reaction was caused by an allergic reaction to propofol, since there are published case reports of pulmonary edema after propofol administration. The allergist issued an anesthesia patient information card about the propofol allergy. However, Dr. Hardy could not figure out why the allergy to midazolam was reported.

14.1.5 What Is the Most Feared Side Effect of Propofol?

The most feared side effect of propofol is propofol infusion syndrome [2]. It is a very rare, but a life-threatening reaction, occurring mostly, but not exclusively, in children. The reaction is triggered by high doses of propofol

(>4 mg/kg body weight) and/or long-term administration (>48 h). The earliest symptom is often a metabolic lactate acidosis, caused by rhabdomyolysis of myocardial and skeletal muscle. During the course, serious arrhythmias and heart and renal failure develop, which may lead to death [1].

Typical findings are myoglobinuria, ST segment changes, and increases in creatinine kinase, troponin T, potassium, and creatinine. Pathophysiologically, disrupted electron transport in complex IV of the respiratory chain is seen with decreased activity of cytochrome oxidase.

Therapy is symptomatic and consists of stopping the propofol administration, providing cardiovascular support, and treatment for the metabolic acidosis and renal failure. It is important to avoid high doses or long-term use of propofol, especially in children.

14.1.6 Is There Such a Thing as Propofol-Induced Pulmonary Edema?

Propofol-induced pulmonary edema actually has been reported [4, 10]. It is unknown, however, if the reported findings were actually caused by propofol. Simultaneously, propofol seems to exacerbate pulmonary edema in septic patients [8, 9]. In summary, the current data is inconclusive. Propofol-induced pulmonary edema seems unlikely, but not impossible.

14.1.7 What Was the Most Likely Cause of Olivia's Pulmonary Edema?

Forming a diagnosis from a description of events in a patient medical record is difficult. The reported restrictive ventilation disorder, moderate pulmonary hypertension, and pulmonary parenchymal swelling could be the result of aspiration of gastric contents, a pulmonary infection, or pulmonary edema.

Since the child has an otherwise unremarkable history, the diagnosis of negative pressure pulmonary edema (NPPE) seems likely (see also Fig. 24.2 and Sect. 24.1.12). The NPPE is a known anesthesia complication with an incidence of 0.05–0.1 %. Usually young, healthy, and muscular patients undergoing general anesthesia are affected. The NPPE is a self-limiting, noncardiac pulmonary edema, triggered by a short closure of the upper airway with simultaneous forced inspiration, generating a strong negative intrapulmonary pressure.

Fifty percent of cases are caused by laryngospasm. Other causes include obstruction of the laryngopharynx (e.g., by upper airway obstruction), biting the endotracheal tube or laryngeal mask airway, aspiration of tumor or foreign material, epiglottitis, or croup.

Pathophysiologically, an extremely strong negative intrathoracic pressure is formed due to a forced inspiration attempt against a closed airway. This then causes an acute increase in the venous return into the right side of the heart and a volume excess in the pulmonary vessels. The increase in hydrostatic pressure leads to transudation of fluids into the pulmonary interstitial space.

Clinical signs include dyspnea, tachypnea, cyanosis, hypercapnia, a reduction of oxygen saturation, and labored respiration. In addition, paradoxical breathing, stridor, red bubbly tracheal secretions, and agitation can occur. This set of symptoms appears within minutes and requires fast diagnosis and therapy by the anesthesiologist. The diagnosis must be made clinically, due to the acute urgency of the situation. Later, radiology films can confirm the diagnosis (chest X-ray, thorax CT). Most cases of NPPE resolve within 24 h. While some patients may require reintubation and mechanical ventilation, others, like Olivia, can be managed noninvasively with supplemental O_2.

>> *The attending anesthesiologist Dr. Hardy was now sure that Olivia did not have an allergic reaction but must have aspirated a small amount of gastric contents. Maybe the cuff wasn't sufficiently inflated, maybe she aspirated during intubation. There was a laryngospasm after extubation, perhaps triggered by the aspirated material or blood. Olivia attempted to force inspiration against the closed airway, which*

caused negative pressure pulmonary edema to develop. Therefore, the results of the chest X-ray, the pulmonary function tests, the echocardiography, and the skin prick test make sense.

Dr. Hardy was pretty sure about his reconstruction of the events and asked anesthesia tech Ann to get propofol and fentanyl from the pharmacy and set out a laryngeal mask airway.

"*But in the anesthesia patient information card, it says that Olivia is allergic to propofol. You saw that, didn't you? Should I prepare a prophylaxis with H_1 and H_2 blockers?*" *asked Ann.*

"*I know what I am doing. It's all right. Trust me!*" *responded Dr. Hardy. He was thinking to himself, "That's the problem when you spend your whole career in one hospital. There are techs and nurses who meet you in the beginning and then never seem to notice that you learned something during the years of training.*"

14.1.8 How Would You Rate Dr. Hardy's Decision to Administer Propofol?

The events in the patient record, which occurred during outpatient general anesthesia, seem to make a propofol allergy unlikely. However, the actions of Dr. Hardy, from a medical point of view, are grossly negligent. Furthermore, the child's pediatrician and an allergy specialist diagnosed an allergy and gave the patient an allergy alert card to show other physicians. Dr. Hardy administered propofol intentionally, consciously ignoring the diagnosed allergy. While many patient-reported allergies, especially to anesthetic drugs, are not true allergies, Dr. Hardy's behavior was cavalier and not defensible in a malpractice suit in the event of an adverse reaction.

Furthermore, there are plenty of other medications that could have been used for the induction for this elective surgery, for example, etomidate, ketamine, sevoflurane, and benzodiazepines (however, benzodiazepines would not be recommended in this case). From a medical point of view, there is no reason to insist on propofol, without discussing further with the child's pediatricians and mother.

In essence, Dr. Hardy was planning a provocation test. One should be prepared for an allergic reaction and have emergency equipment ready.

>> Olivia heard the whole thing. She was more terrified than ever. Anesthesia tech Ann showed her annoyance as she prepared the medications and, upon command, issued 10 mg propofol IV. "Now we will wait 10 min and see what happens," announced Dr. Hardy.

14.1.9 What Do You Think of This Idea?

What Dr. Hardy is doing is a drug provocation test. A provocation test is only appropriate when the trigger of a medication allergy can't be identified through the history, skin test, and in vitro tests [6]. Informed consent for this test should be obtained in advance.

The basis of the provocation test is to apply the test substance in the way in which it was thought to have caused the allergic reaction. In order to clarify systemic reactions, oral exposition tests are preferred if possible, even if the initial reaction was triggered a different way. Provocation tests with primary parenteral substance application require strict indications. Emergency medications and equipment must be nearby; the personnel must be familiar with emergency procedures.

Olivia's mother had not been informed of Dr. Hardy's test procedure. For this reason alone, the test should not have been done.

>> "Ten minutes can go by really slowly, when you are waiting," said the impatient attending anesthesiologist Dr. Hardy. When he didn't see any signs of an allergic reaction, he injected 100 µg fentanyl and an additional 50 mg propofol. The induction of anesthesia and the short procedure proceeded without incident. Emergence from anesthesia was also unremarkable, and Olivia was transferred to the PACU. Dr. Hardy was satisfied with his performance. His decisions had been correct. He shot anesthesia tech Ann a look of triumph. Anesthesia tech Ann, however, had been in this business too long to let such behavior get to her.

In the PACU, Olivia admitted to having a little pain, which promptly improved with 1 g of acetaminophen. Her oxygen saturation and heart rate were unremarkable. There seemed to be no reason to measure blood pressure; pediatric patients were often bothered by the cuff's automatic pumping. After half an hour in the PACU, Olivia was transferred to the ward, with no sign of an allergic reaction.

14.1.10 What Do You Say About the PACU Monitoring?

Allergic reactions are divided up into four different types (see Sect. 14.2.2). In this case a type I was suspected. This reaction is immediate, appears within minutes, and is characterized by fulminant cardiovascular reactions, sometimes even cardiac arrest. However, late reactions can also be seen after hours. Therefore, the monitoring duration in the PACU was much too short and should have been extended to a few hours. Furthermore, no blood pressure was measured, which is not justified under any circumstances. Cardiovascular system depression would be discovered much too late.

14.2 Case Analysis/Debriefing

14.2.1 Are Allergic Reactions Common in Anesthesia? What Are the Most Common Causes?

Allergic reactions during anesthesia are seldom and occur with a prevalence of 1:5,000 to 1:28,000 [5]. The identification of the symptoms and diagnosis is often made more difficult by the effects of the anesthesia or the coexisting conditions, such as hypovolemia. The most common symptoms are listed below.

Symptoms of allergic reactions during general anesthesia:

- Cardiovascular collapse
- Bronchospasm
- Cutaneous reactions
- Angioedema
- Generalized edema
- Pulmonary edema
- Gastrointestinal symptoms

Allergic reactions must be differentiated into anaphylactic and anaphylactoid. Anaphylactic reactions are a type I reaction, mediated by IgE antibodies, which leads to release of histamine and mediators. Previous contact with the allergen is required.

Anaphylactoid reactions are direct, non-antibody-mediated reactions of the allergen's substrate with mast cells. Previous exposure is not necessary.

The most common causes during surgeries are [3]:

- Muscle relaxants
- Latex
- Colloid solutions
- Antibiotics
- Radiographic contrast agent
- Palacos® cement (see Sect. 11.1.5)
- Solvents

14.2.2 Which Types of Allergic Reactions Do You Know and What Are Their Defining Characteristics?

Oversensitivity reactions are divided into four categories according to Gell and Coombs.

14.2.2.1 Type I Reaction

The type I reaction is the IgE-mediated immediate anaphylactic reaction. Type I reactions include allergic rhinitis, extrinsic asthma, and anaphylaxis. The reaction requires previous contact. It occurs within seconds to minutes (anaphylaxis), and late reactions can be seen after hours. The reactions can be local or systemic and can manifest on the respiratory tract, gastrointestinal tract, skin, and cardiovascular system.

The most serious form is anaphylactic shock, during which release of vasoactive mediators leads to a life-threatening decrease in blood pressure.

14.2.2.2 Type II Reaction

The type II reaction is IgM and IgG mediated and leads to a cytotoxic reaction. This group includes ABO-incompatible transfusion reactions,

medication-induced hemolytic anemia, agranulocytosis, Goodpasture syndrome, and heparin-induced thrombocytopenia.

14.2.2.3 Type III Reaction

Type III reactions are characterized by antibody formation against soluble antigens. Immune complexes form, which can be deposited in the capillaries or activate the complement system. This group includes serum sickness, exogeneous-allergic alveolitis and immune complex nephritis.

14.2.2.4 Type IV Reaction

The type IV reaction is T-cell-mediated reactions such as transplant rejection (graft vs. host disease) and contact dermatitis. Another typical disease is atopic dermatitis.

14.2.3 Which Medical Errors Do You See in the Presented Case?

14.2.3.1 Preoperative Evaluation

The child's preoperative evaluation was inadequate (see Sect. 14.1.2). Peeking into your lab coat pocket to check your reference book or looking up background information on the computer is always allowed.

14.2.3.2 Exposition with Potential Allergic Substances

Even though the allergy to propofol was questionable, the decision to administer propofol without further consultation and evaluation was wrong (see Sect. 14.1.7).

14.2.3.3 Monitoring in the PACU

The PACU monitoring was inadequate – there wasn't one blood pressure measurement – and the PACU stay was much too short, being that there was the possibility of a delayed allergic reaction to propofol (see Sect. 14.1.8).

14.2.4 Which Systems Failures Can You Find in the Presented Case?

14.2.4.1 Preoperative Evaluation

Systems failures contributed to the inadequate preoperative evaluation. The patient was scheduled for a preanesthesia clinic visit Friday afternoon after the shift ended. The ENT physicians obviously knew that the evaluation for this patient is more complicated than a standard evaluation. In addition, it would likely be difficult for the anesthesiologist to be able to consult with the patient's pediatrician and pediatric allergist to sort out the allergy diagnosis.

In addition, there was no longer a supervising physician available to answer the resident's questions. An attending should be available at all times to assist with the preoperative evaluation. Furthermore, the anesthesiologist, who had been in the preanesthesia clinic all day, could no longer be reached. Also, the anesthesia resident who did the preoperative evaluation didn't have all the paperwork – the patient report was missing.

14.2.4.2 Information Relay/ Communication

The attending physician, Dr. Hardy, was surprised by the patient in the OR. He had not been informed of a possible propofol allergy. The anesthesia resident did not pass on to the anesthesiologist-in-charge or the Friday on-call attending about the patient's complicated history or even write a note on the schedule about the allergy concern.

14.2.4.3 Standard Operating Procedures (SOP) with Possible Allergies

Regardless of the hierarchy, the team players needed a hospital SOP, which forbids the administration of substances to which patients may have allergies, unless the details of the allergy have been appropriately considered and ruled out.

14.2.5 Children Become People, and Doctors in Training Become Attending Physicians!

Dr. Hardy had been an attending physician for 1 year and had received his residency training in the same hospital. His co-workers and all the OR staff knew him rather well. Dr. Hardy felt his promotion was not recognized by anyone in the way he had expected. For him, the career advance was simultaneously connected with competency and leadership. He interpreted anesthesia tech Ann's

question as an attack on his competency, and therefore he didn't answer her.

Do you also know of problems which arise from promotion within the same hospital?

Career advancement means a new role definition.

Every new boss must become acquainted with the newly associated administrative tasks and functions. First, a good leader needs to understand himself/herself and others. After that, an evaluation of the congruency of the current role and the new role must be considered. The new role should be accepted and communicated. Often, the changes lead to changes in the form of communication. In business, it is well established that leaders are introduced to their new responsibilities and receive appropriate training. In medicine, however, such preparation and training seldom occur. Often the usual process is model learning, connected to an admired figure, and also a process of disconnecting yourself from your roots. I am not a child anymore: I am an attending physician!

A strict hierarchical separation from "those under me" not only makes one very lonely – maybe you've heard the phrase "it's lonely at the top" – but it also makes it more difficult to learn from your mistakes and to work as a team. A good leader remembers his training, how he learned from his mistakes, the many good bits of advice, and his internal desire but also his reluctance to criticize his bosses.

14.2.5.1 The Following Sentence Is for Attending Physicians

Your co-workers have the same reluctance to criticize, and if you want to continue to learn from your mistakes and receive helpful tips, then you must overcome your reluctance to learn from criticism. Become explicit!

14.2.5.2 The Following Sentence Is for Non-supervising Physicians

You can't imagine how hard it is to be explicit when you are the boss. Don't be too shy to take the first step!

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Case 15: Cosmetic Surgery

15

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Contents

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15.1 Case Introduction

>> Linda was happy to finally be admitted to the hospital for oral and maxillofacial surgery (OMS). "Mandibular prognathism, correction with the Obwegeser-Dal Pont technique" was written at the top her informed consent form, in other words, a bilateral sagittal osteotomy of the mandible, ramus, and angle. Ever since she was a child, Linda had suffered emotionally from her underbite. It was mandibular prognathism, which is an elongation of the mandible with protrusion of the bottom front teeth, causing misalignment of the teeth and malocclusion and a protruding chin with projection of the bottom lip.

Linda had always been upset by her unusual profile, and as she grew, the situation had become worse. Now cosmetic as well as functional difficulties in chewing were serious problems for her (Fig. 15.1).

Finally the time had come! She was 17 years old; the bony growth of her jaw was complete, and the surgical correction of her underbite could be done. She came to the OMS clinic with her mother, and after a final exam, the steps of the operation were explained to her, as well as the risks. "A sagittal cut will be done on both sides of the mandible, in the area of the angle. The segments will then be moved dorsally, as is needed for the correction, and fixed in this position with wire sutures."

It became clear to Linda that bleeding, infection, and damage to nerves could occur. With dreams of having a beautiful profile, she was

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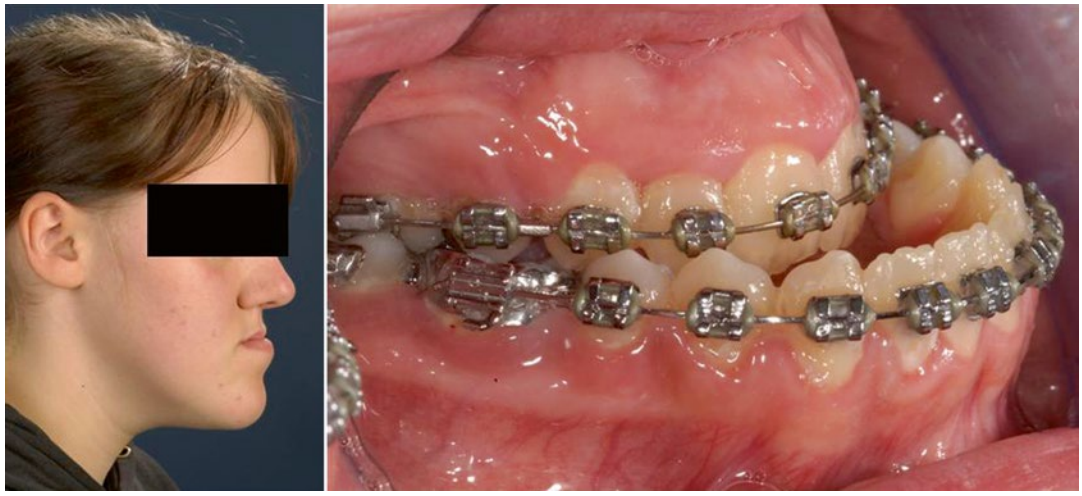

Fig. 15.1 Side view of Linda with pronounced mandibular prognathism, which led to the corresponding dental malocclusion

willing to undertake all risks. "*Nice that the entire surgery can be done through my mouth. There will be no visible scars*," *she thought to herself. Then the surgeon explained the postoperative wiring of her mouth. For 1 or 2 weeks*, *she would only be allowed to have fluids and liquids*, *and speaking would be difficult. After this discussion*, *the anesthesiologist obtained preoperative informed consent*.

15.1.1 What Are the Specific Risks to Explain Before Obtaining Informed Consent?

Apart from the usual intubation risks, such as tooth injury, hoarseness, nausea, vomiting, and aspiration, the necessity of nasal intubation should be mentioned. The specific risks of nasal intubation include injury to the nasal cavity and epistaxis. In addition to the risks of general anesthesia, the patient should be informed of possible nasogastric tube placement, which may be used for the duration of the surgery, and sometimes postoperatively.

At the end of the surgery, the patient's upper and lower jaws are wired to one another for stability. Extubation can only occur when patients are awake enough to prevent aspiration of blood or gastric fluids. Memories of the endotracheal tube still in place and the procedure of extubation are relatively common and should be mentioned during the preanesthesia discussion.

>> "*Of course it's not going to be easy*," *thought Linda*, *but getting rid of the big chin which she saw in the mirror every morning would make everything worth it. All her friends already had boyfriends…after the surgery she would be able to find a boyfriend as well*.

Dr. Greg was a third-year resident in anesthesiology. For the past 2 weeks, *he had been in his OMS rotation. In order to be well prepared for administering the anesthesia*, *he had reviewed the specifics of this surgery from an anesthesia perspective the night before*.

15.1.2 What Are the Anesthetic Considerations for This Type of Surgery?

Some anesthetic considerations were mentioned in Sect. 15.1.1. Here are a few more details [6]:

15.1.2.1 Nasal Intubation

Nasal intubation is usually performed with a nasal RAE tube. The tube is then extended with a flexible "goose neck" connector, which is placed caudally over the forehead and hooked up to the anesthesia circuit using extension tubing. In order to minimize the risk of bleeding, sympathomimetic

nasal drops can be used in both nares. The tip of the nose is in danger of developing pressure necrosis from the tube fixation. Therefore, one must take care that the tube fixation is free of tension.

Sometimes, a leaky cuff occurs after intubation, usually due to cuff damage from anatomical structures or the Magill forceps. No pressure should be placed on the cuff when using the Magill forceps. The cuff integrity must be carefully assessed after intubation, due to the limited airway access during surgery (see below).

15.1.2.2 Intraoperative Access to the Airway

Intraoperatively, the rule is "**No Airway Access**". Therefore, it is necessary to secure the tube and its connection to the anesthesia circuit tubing very well.

Fixation of the tube can be done by taping or – with permission from the surgeon – by suturing, to the side of the nose. A special problem is intraoperative tube damage by the surgical instruments. This situation must be anticipated, with the required instruments ready for use – such as a Cook airway exchange catheter. At the end of the procedure, removal of the sterile drapes must be done carefully, to assure that accidental extubation does not occur.

15.1.2.3 Eye Protection

The patients' eyes are not accessible after the surgical area is draped. The anesthesia team must therefore protect the eyes from prep solution or pressure. You should discuss how to protect the eyes with the surgical team.

15.1.2.4 Intraoperative Positioning

During surgery, the patients are positioned supine, slightly lateral, with the head hyperextended. Cerebral blood flow can be compromised in patients with arteriosclerosis of the carotid arteries.

15.1.2.5 Tamponade of the Oropharynx

At the start of the procedure, the surgeons tamponade the oropharynx. It is the anesthesiologist's job to make sure that the throat pack is removed before the jaw is immobilized and before emergence from anesthesia.

15.1.2.6 Maxillomandibular Fixation and Immobilization

Postoperatively, the jaw is immobilized by metal wires. Postoperative fixation requires the placement of the nasogastric tube, which, if all goes well, will not be used postoperatively. The anesthesiologist must know at which level (between which teeth) the intermaxillary fixation was done, in order to be prepared for an emergency. Should an emergency arise, requiring immediate severing of the fixation, wire cutters must be at the bedside, until the time at which the fixation is removed. In many cases, the surgeons use rubber bands for fixation in the immediate postoperative period, replacing the bands with wires once the patient has fully recovered from anesthesia. While rubber bands are easier to cut in the event of an emergency, the anesthetic implications are still the same.

After the osteotomy, the OMS surgeons use miniplates with tiny screws to create a new osteosynthesis of the mandible/maxilla. These screws withstand significantly less force than the intermaxillary fixation – at least when the fixation is done with wire. Forced mouth opening dislocates the screws, and patients have to understand in advance that they must not open their mouth and should breathe primarily through their nose. Therefore, patients' need to have a high level of cooperation, which is influenced by the preoperative informed consent discussion and by the choice of medication (see below).

Furthermore, it has proven to be beneficial for the surgeon to stay in the OR until tracheal extubation. In case of emergency, the surgeon can cut the intermaxillary fixation the fastest and should hold the lower jaw until extubation is complete, which, at the least, would remind the patient not to open the mouth.

15.1.2.7 Choice of Drugs

The goal of anesthesia for OMS is to have the patients as awake and cooperative as possible at the end of surgery and to avoid PONV (postoperative nausea and vomiting). Total intravenous anesthesia (TIVA) with propofol and remifentanil is an excellent choice to achieve these goals. The prophylactic administration of an antiemetic – for example, dexamethasone at

induction of anesthesia or ondansetron prior to emergence – is beneficial (see Chap. 4 Table 4.1). The postoperative analgesia should primarily consist of non-opioid analgesics, in order to reduce the risk of PONV.

15.1.2.8 Blood Loss

Significant blood loss is to be expected with many maxillofacial procedures, such as Le Fort osteotomy or maxillary advancements. Possible strategies to reduce blood loss include the administration of tranexamic acid [2] and maintaining deliberate hypotension [3]. Whether or not these procedures lead to a reduction of blood transfusion is debated. Therefore, the indication should be carefully considered according to a risk–benefit analysis. In the presented case, the planned prognathism operation rarely has blood loss of more than 300 ml, making special considerations unnecessary.

15.1.2.9 Corticosteroid Administration

In many hospitals, dexamethasone 10 mg IV is given just prior to starting surgery in order to reduce the inflammatory response, thereby reducing the local swelling. Effectiveness, however, is highly debated. Studies which show a shorter hospital stay after maxillofacial surgery with steroid administration often neglect to take into account the weakened immune defense and the danger of prolonged wound healing [4].

>> The induction and maintenance of anesthesia proceeded without incident. Dr. Greg carried out the anesthesia as "TIVA with propofol and remifentanil." Muscle relaxation was only necessary to facilitate tracheal intubation with a nasal RAE tube (6.5 mm ID). After about 2 h, the maxilla and mandible were wired together to achieve immobilization.

Dr. Greg was accustomed to the problems of intermaxillary fixation and had the OR tech give him wire cutters for the patient. "Extubation only when full consciousness has been obtained, AND sufficient spontaneous respiration, AND adequate communication!" the attending anesthesiologist Dr. Eldridge had reminded him before surgery. "Any and all pharmacological excess (hypnotics, opioids, and muscle relaxants) must be avoided. Half an hour before the end of the operation, give a little bit of a longer working opioid, such as fentanyl, and combine it with a peripheral-acting analgesic such as acetaminophen or ibuprofen. The patient should receive substantial postoperative pain relief."

Dr. Greg informed his attending Dr. Eldridge that he was ready to begin the emergence. The surgeon was present, in order to quickly and properly cut the wires, if needed. Dr. Greg suctioned the stomach through the nasogastric tube before extubation. Then Dr. Greg slowly removed the tube, all the while suctioning through the tube and finally suctioning the oral cavity.

Linda was really awake after the extubation. She denied pain or nausea. "I did it," she thought. "What will my new chin look like?"

With cardiovascular stability and an S_pO_2 reading of 95 % on room air, she was transferred to the PACU. Dr. Greg informed the less experienced (second-year) PACU resident, Dr. Niac, about Linda: "She is a young, healthy patient. She had a mandibular prognathism osteotomy, and her jaw is now wired shut. The surgery and anesthesia went without complications, with a total blood loss of 300 ml. She received 1,500 ml of a Lactated Ringer's solution. The emergency scissors to cut the wires are beside her on the bed. Linda was quite nervous before the operation, but now everything is OK."

"Wow!" thought Dr. Niac. "A young healthy female, so rare here. I haven't yet cared for a patient who is wired shut, but I won't have any problems with her. She's super healthy, which is good, since we are so busy." Nevertheless, he quickly went to Linda's bedside to form a better picture of the patient. The PACU nurse Maria was just hooking her up to the monitor, which showed:

- *S_pO_2: 89 % without oxygen*
- *Blood pressure: 160/80 mmHg*
- *Heart rate: 120 beats/min*

Linda was shivering slightly and responded slowly, but adequately, to verbal stimuli.

15.1.3 What Appears Abnormal to You?

Basically, many factors can cause the combination of systemic hypertension and tachycardia. The most common causes in the PACU are listed below:

15.1.3.1 Stress

A stress response is possible, due to pain, nausea, discomfort from a urine catheter or a full bladder, fear, or other psychological impairments, such as perioperative cerebral dysfunction (see Sect. 18.1.2).

15.1.3.2 Compensatory Tachycardia

Compensatory tachycardia can be triggered by hypovolemia or anemia and can – when the disorders are minimal – appear with hypertension.

15.1.3.3 Respiratory Disorders

Another possible reason for hypertension and tachycardia in the PACU is respiratory disorders, especially when hypoxia and/or hypercapnia are present. In the presented case, the low S_pO_2 value is a clue that there is a respiratory disorder.

15.1.3.4 Shivering

Simultaneous appearance of muscle shivering, also called postoperative shivering, occurs in up to 50 % of all postoperative patients. It is a generalized, reflexive, non-suppressible tremor, which increases the production of energy by 50–100 %. The threshold for shivering is about 1 °C below the threshold for vasoconstriction as protection against hypothermia.

All anesthetic agents tested thus far affect autonomic temperature regulation [8].

Usually, there is an extension of the temperature range encompassing temperatures sensed as normal, so that the patients begin to sweat at higher temperatures and begin to feel cold at colder temperatures.

Shivering after anesthesia is divided into two types [1]:

- Thermoregulatory shivering is accompanied by vasoconstriction and is a physiological reaction to perioperative heat loss.
- Non-thermoregulatory shivering is accompanied by vasodilation and often associated with pain. The appearance of non-thermoregulatory shivering is probably due to the return of spinal reflexes in the presence of inadequate central inhibition as the result of residual anesthetic action.

15.1.4 What Action Would You Take? How Is Postoperative Shivering Treated?

The most urgent treatment of the patient is the administration of oxygen, since hypoxemia must be excluded as the cause of the changes and also because the shivering muscles have an increased oxygen requirement. Finally, the cause of the shivering, listed in Sect. 15.1.3, needs to be clarified.

The most important preventative action to avoid postoperative shivering is sufficient protection from heat loss during the surgery by protective body coverings and the use of effective passive warming devices. With thermoregulatory shivering, passive warming must be continued in the PACU.

Apart from this, there are various pharmacological therapy options [5]:

15.1.4.1 Opioids

Several μ-receptor agonists can reduce postoperative shivering. The most reliable and popular of these is meperidine (Demerol). The standard dose is 12.5–25 mg. The number needed to treat is 3.

15.1.4.2 α_2-Receptor Agonists

Shivering triggered from the hypothalamus can be relieved by α_2-receptor agonists. The most commonly used in this class is clonidine in a dose of 75 μg. The number needed to treat is <2.

15.1.4.3 5-Hydroxytryptamine Agonists

The exact mechanism of action is not clear, but 5-hydroxytryptamine agonists can inhibit

postoperative shivering. Ondansetron or granisetron is used. The number needed to treat is about 3.

15.1.4.4 Others

Countless other drugs can alleviate postoperative shivering, including tramadol, physostigmine, or magnesium. Due to the effectiveness of meperidine, clonidine, and ondansetron, these other medications are seldom used.

>> The PACU nurse Maria gave Linda an oxygen mask with 2 l oxygen/min. Dr. Niac was shocked by the young woman's hypertension and tachycardia. "Probably stress-induced shivering," he thought, and he administered 1 µg/kg *clonidine to Linda as a short infusion. The shivering disappeared almost immediately.*

As he returned half an hour later to the PACU to make rounds, he approached Linda's bedside once more. She complained of mouth pain and slight nausea. "My whole mouth seems foreign to me," she mumbled through her teeth which were wired shut. "It's a little hard to breathe." Her blood pressure was 150/85 mmHg, the heart rate was 95 beats/min, and the S_PO_2 measurement showed 92%. Dr. Niac administered meperidine and 4 mg ondansetron IV.

Dr. Niac wondered to himself, "Strange that the S_pO_2 has not yet improved." As he increased the inspiratory O_2, he realized that PACU nurse Maria had already turned it up to 6 l/min due to the low S_PO_2. "I should have checked on that earlier," he thought. "Then I would have given her peripheral pain relief instead of the opioids." He increased the oxygen flow to 8 l/min.

At that point, he remembered the reduced effect an increase in F_iO_2 has on oxygenation in the presence of an increased intrapulmonary shunt. "Ahh, whatever," thought Dr. Niac. "That phenomenon is only seen in really sick patients with sepsis or acute lung injury, but not in this ASA 1 patient." He tried to remember which inspiratory oxygen concentration and which P_aO_2 should be achieved with administration of 8 l/min.

15.1.5 Do You Know?

The answer is presented in Table 15.1 (see also Sect. 20.1.7).

Table 15.1 Effect of the oxygen flow rate from masks on the inspiratory oxygen concentration (F_iO_2) and the arterial oxygen partial pressure (P_aO_2)

Flow rate oxygen (mask without reservoir)	F_iO_2	Expected P_aO_2 in middle-aged patients without pulmonary disease (mmHg)
Without	0.2	100
5–6 l/min	0.4	235
6–7 l/min	0.5	307
7–8 l/min	0.6	378

Note that with a simple mask, the actual F_iO_2 level is highly dependent upon the patient's minute ventilation due to entrainment of room air. If a higher F_iO_2 is desired, a mask with a reservoir (non-rebreather mask) should instead be used

Table 15.2 Relative effect of positioning on functional residual capacity

Positioning	Functional residual capacity (%)
Standing	100
Sitting	~95
Reverse Trendelenburg positioning (30°)	~80
Lying flat	~70
Trendelenburg positioning (−30°)	~65

15.1.6 What Should Dr. Niac Do in Addition to Increasing the Oxygen Flow Rates?

15.1.6.1 Auscultate the Lungs

Lung auscultation is the next step, as a simple, fast, and noninvasive action which should always be done when a respiratory disorder is suspected.

15.1.6.2 Administer Nose Drops

Unobstructed nasal breathing should be reassessed and confirmed in patients with intermaxillary fixation. Generous administration of nose drops to reduce swelling is therefore justified.

15.1.6.3 Sit Up the Patient

Every general anesthesia causes a temporary reduction in the functional residual capacity (FRC) of 15–20 %. Putting the patient in a semi-recumbent position can increase the FRC (Table 15.2). The effects are especially pronounced

when there is an additional reduction of the FRC due to increased intra-abdominal pressure or obesity.

>> Dr. Niac auscultated Linda's lungs. Her breath sounds were equal bilaterally, quiet, and without stridor, crackles, crepitations, or rales. However, Linda's respiration was labored. Respiratory rate was 21/min. Sitting up and increasing the inspiratory oxygen did not improve Linda's respiration. She was tired and simultaneously agitated; she still identified pain at the site of the surgery. Her cardiovascular status remained stable with hypertension and tachycardia. The S_PO_2 value hadn't changed either. Dr. Niac asked nurse Maria to do an arterial blood gas; shortly thereafter she brought back the following values:

- *pH: 7.31 (reference 7.35–7.45)*
- *P_aO_2: 70 mmHg (reference 70–100 mmHg)*
- *P_aCO_2: 65 mmHg (reference 36–44 mmHg)*
- *HCO_3: 21.2 mEq/l (reference 22–26 mEq/l)*
- *BE: –2.2 mEq/l (reference ±2 mEq/l)*
- *S_aO_2: 91.1 % (reference 95–98 %)*
- *Lactate: 1.2 mmol/l (reference 0.5–2.2 mmol/l)*
- *Na^+: 143 mEq/l (reference 135–150 mEq/l)*
- *K^+: 4.2 mEq/l (reference 3.5–5.0 mEq/l)*
- *Hb: 11.6 g/dl (reference 12–14 g/dl)*
- *HCT: 34 % (reference 37–47 %)*

15.1.7 How Do You Interpret the Arterial Blood Gas Values?

Linda has a respiratory acidosis with hypoxemia. The P_aO_2/F_iO_2 (Horowitz index) is roughly 115 mmHg (reference range usually between 350 and 450).

Slight hypoventilation with a slight increase in the P_aCO_2 is common after general anesthesia. Immediately post-op, inadequate ventilation is caused by two factors:

- Pharmacological depression of cerebral respiratory center by anesthetic agents
- Mechanical changes in the respiratory parameters as a result of anesthesia and surgery

Healthy patients tolerate the mild hypoventilation well and only need minimal assistance, such as an increase in inspiratory oxygen via a face mask, as the condition is self-limiting and resolves as the patient regains consciousness. If the increasing P_aCO_2 is not sufficient to cause an increase in respiratory drive, the accumulating CO_2 further reduces vigilance and increases agitation and an increased risk for cardiac arrhythmia.

>> Although Dr. Niac thought a structural cardiopulmonary problem would be very unlikely in this young healthy patient, he ordered a chest X-ray just to be on the safe side. The X-ray showed age-appropriate findings: no atelectasis, pneumothorax, pulmonary edema, or heart failure.

"Maybe Linda can't breathe deeply because she is in pain," he thought and gave her another 12.5 mg meperidine IV. Suddenly the S_PO_2 dropped to 84 %. "Dang!" thought Dr. Niac, "That was totally wrong!" Nurse Maria heard the monitor's alarm and came to the bedside. "Linda," she called out, shaking her shoulder. "You need to take a deep breath!" Linda sleepily opened her eyes. "Why don't they help me?" she thought. "I am so nauseous and I can hardly breathe. Something must have gone wrong with my surgery!" Dr. Niac gave a questioning glance to Nurse Maria and then gave in. "I better call my attending."

As the attending anesthesiologist Dr. Eldridge arrived at Linda's bedside, he knew exactly what was wrong. The young woman was agitated and pale and had slightly cyanotic lips, tachycardia of 125 beats/min, S_pO_2 of 80 %, and a panicked look on her face as she lay there in the bed.

15.1.8 What Would You Do Now?

Linda is now in grave danger. Her oxygenation and ventilation are inadequate. Simultaneously, she has a decreased level of consciousness, so that her airway reflexes are no longer sufficient. For these reasons, the maxillomandibular fixation must be cut!

>> "Maria," said Dr. Eldridge to the PACU nurse, "Call the maxillofacial surgeon and give me the wire cutters!" Then he quickly began to cut the wires, although Linda constantly fought against him, shook her head from side to side – not cooperating in the least. Linda didn't care what

happened next. She only thought of air – she only wanted air – she was oblivious to the hectic activity around her. Suddenly her teeth were split apart, and the big wad in her throat was magically gone. She voraciously inhaled and exhaled, inhaled and exhaled….

15.1.9 What Was the Problem?

Due to the operative dorsal placement of the mandible, the tongue was pushed against the posterior pharyngeal wall. This usually has no effect on breathing.

In more severe cases, posterior placement of the mandible and thus the base of the tongue (Fig. 15.2) can narrow the upper airway to such an extent that a respiratory disorder develops. An operation can be done to correct this, in which the angle of the jaw is adjusted. It is possible that the tongue size may need to be reduced.

>> As the maxillofacial surgeon entered the PACU, Linda was already rosy and had gradually returned to a normal level of consciousness. "It doesn't surprise me that the fixation had to be cut," the surgeon said as he turned to attending anesthesiologist Dr. Eldridge. "We carried out an extensive correction." Then Dr. Eldridge and the maxillofacial surgeon explained to Linda what had happened and explained that she would need another operation in order to replace her lower jaw in a position better for her tongue.

Two months after the surgery, Linda was happy that everything went without further complications. Her boyfriend found her new smile marvelous. She had never imagined, however, that the correction of her chin would be such a life or death adventure.

15.2 Case Analysis/Debriefing

15.2.1 What Causes of Postoperative Hypoxemia Can You Name?

15.2.1.1 Hypoventilation

This point was already discussed in Sect. 15.1.7.

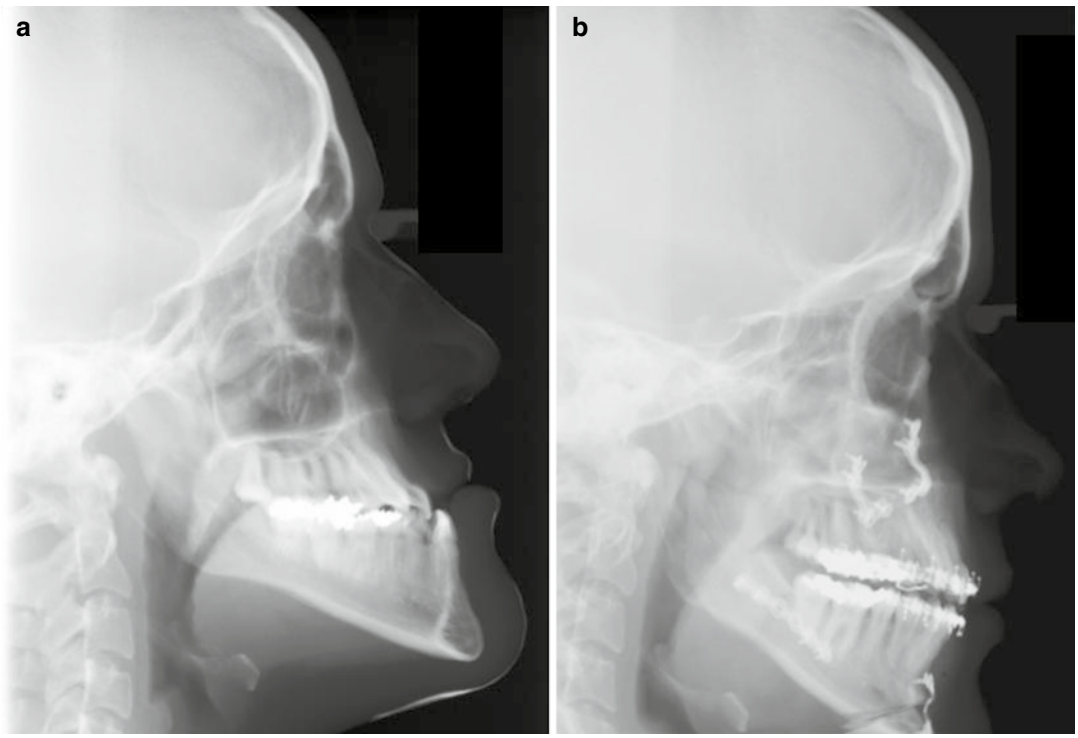

Fig. 15.2 (**a**, **b**) Lateral X-rays of Linda with the clearly visible mandibular prognathism before surgery (**a**) and the status postsurgical correction (**b**)

15.2.1.2 Ventilation–Perfusion ($\dot{V}/\dot{Q}$) Mismatch

A significant cause of a $\dot{V}/\dot{Q}$ mismatch in the postoperative period is increased dead space ventilation and atelectasis, which occur due to positioning, inadequate intraoperative ventilation (lack of PEEP), or insufficient pain relief. The closing capacity and shunt perfusion increase in the supine position, especially in older or obese patients. Secretions, abdominal distension, or preexisting pulmonary diseases can perpetuate the problem by further reducing the FRC. The cause of the problem can be treated by CPAP administered in PACU.

In the PACU, constricting chest bandages can cause a pathophysiologically similar disorder. Bandages have to be loosened in agreement with the surgeon. Principally, apart from surgical causes, all other possible causes must be considered, such as pulmonary embolism or edema (iatrogenic, heart failure, negative pressure pulmonary edema) as well as pneumo- and hemothorax.

15.2.1.3 Imbalance Between Available Oxygen ($\dot{D}O_2$) and Oxygen Consumption ($\dot{V}O_2$)

In addition to the factors described above, shivering, fever, stress, pain, and septic shock all lead to an increased $\dot{V}O_2$. Apart from that, the cardiac output and the arterial oxygen content (C_aO_2) determine $\dot{D}O_2$. Thereby, the C_aO_2 is basically only determined by the hemoglobin concentration of the blood and the arterial oxygen saturation (S_aO_2). The main factors which affect S_aO_2 are ventilation and $\dot{V}/\dot{Q}$ mismatch, already discussed in Sect. 4.1.6.

The imbalance between DO2 and $\dot{V}O_2$ can be proven by the arterial-venous oxygen saturation difference and, with consideration of the hemoglobin, delivers important information about the adequacy of cardiac output.

15.2.2 Which Medical Errors Do You See in the Presented Case?

15.2.2.1 Pain Therapy

A common side effect of opioids is that they increase the incidence of PONV. As discussed in Sect. 15.1.2, they should be administered with utmost restraint in patients with intermaxillary fixation. The repeated administration of opioids in the PACU was not appropriate, especially since Linda had a decreased level of consciousness and complained of nausea.

15.2.2.2 Rechecking the Steps

Dr. Niac first gave an opioid and then increased the oxygen flow rates. The chain of events was wrong, as he later realized.

15.2.2.3 Nasogastric Aspiration

It was correct to give Linda an antiemetic, when she complained of nausea. In addition, however, the nasogastric tube should have been suctioned quickly.

15.2.3 Which Systems Failures Can You Find in the Presented Case?

15.2.3.1 Extubation

Dr. Greg properly informed his attending about the imminent tracheal extubation. However, he had little experience with patients with wired jaws; therefore, the attending anesthesiologist, Dr. Eldridge, should have been present.

15.2.3.2 Introduction to the PACU

In the PACU, Dr. Niac encountered a patient with intermaxillary fixation for the first time. Dr. Greg had informed him about the wire cutters, but he hadn't received any further instructions. Later, he was hesitant to use the cutters. If Dr. Niac had received the proper training he needed before being placed in the PACU or if an attending was also present, then the critical problems may have been avoided.

15.2.3.3 Information Exchange with the Maxillofacial Surgeons

The maxillofacial surgeons knew that Linda could experience respiratory problems due to the intermaxillary fixation. Unfortunately, they did not forward this information to the anesthesia team about the large tongue. This type of communication is part of the preoperative checklist.

Anesthesiologists should also remember to discuss this aspect with the surgeon every time prior to extubation. In case of an adequate perioperative discussion of the risk of airway obstruction, the anesthesiologists could have asked for the jaws to be fixed with rubber bands, rather than wires, in the immediate postoperative period.

15.2.4 Why Didn't Dr. Niac Question His Working Hypothesis Sooner?

The PACU resident was caring for an intermaxillary fixated patient for the first time in his career. Therefore, due to his knowledge level, he fixated on the treatment of hypoxemia as a result of the pulmonary function and the pulmonary gas exchange, according to the philosophy: "It can only be this and nothing else." Because Dr. Niac was not aware of the possibility of upper airway problems after prognathism surgery, the option to "open the fixation" was not considered. Thank goodness, the upper airway obstruction was discovered and treated in time by the attending anesthesiologist.

In critical situations, it often happens that players stick to an oversimplified or incorrect mental model, thereby misfitting their perceptions and thinking to the model. Through the resulting cognitive **tunnel vision**, important clues to the outside world are ignored – Patient, Team, Equipment – clues which could point to the true solution. Problems with situational awareness (perception, comprehension, and projection of events) contribute to errors in management [7]. What are some strategies you can use to avoid attention fixation on the wrong thing?

Do you remember Case 7 (see Sect. 7.2.3)? Take a step back! Another cognitive strategy will be discussed in the next chapter – Case 16 (see Sect. 16.2.4).

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Case 16: Laparoscopic Cholecystectomy

16

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Contents

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16.1 Case Introduction

>> The schedule for general surgery in OR #4 on Wednesday had a total of three laparoscopic cholecystectomies. The anesthesiologist Dr. Sven thought to himself, "The usual list again." As a second-year resident, he was often in the laparoscopic OR. He felt confident enough here; he was accustomed to the sequence of events and the surgical and anesthetic considerations for these procedures. He was even acquainted with the individual surgeons and their special desires. "Thank goodness the attending Dr. Harold isn't operating today," he thought. Dr. Harold was an exceptionally adept and fast surgeon, who always thought the turnover between the patients was too slow, and, therefore, he put a lot of pressure on anesthesiologists to speed up. Dr. Buster was assigned to the OR today, a less experienced surgeon, who had just become board certified. Dr. Sven knew him from his surgical rotation as an intern. Once a week, they played soccer together on an amateur team.

The first two cases of the day went without incident. After Dr. Sven transferred the second patient to the PACU, he returned to the preoperative area.

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Here he greeted Ms. Hall, 41 years old and overweight. Dr. Sven remembered her from yesterday's preoperative visit. He glanced at her medical record once more and reviewed her preoperative assessment. Ms. Hall weighed 96 kg and was 169 cm tall (BMI = 33.6 kg/m²). She had had acute cholecystitis 2 weeks ago and was now pain-free and symptom-free. In addition, she had systemic hypertension which was treated with lisinopril and metoprolol. She also suffered from a seizure disorder, but for the past year she had taken carbamazepine and was seizure-free.

Ms. Hall's upper airway exam revealed a Mallampati score of 3 and a Wilson score of 1. Lab values were unremarkable.

16.1.1 What Do You Know About the Mallampati and/or Wilson Scores?

Both scores are used prior to general anesthesia to estimate the probability of a difficult airway. With help of the Mallampati score, the expected difficulty of intubation is assessed according to visible structures and maximum mouth opening. The original differentiation into three classes [7] was later revised into four classes (Table 16.1) [9].

Mallampati scores 3 and 4 are associated with difficult airways; however, the scoring is dependent upon the investigator.

Due to the poor correlation of individual factors in predicting a difficult intubation, the Wilson risk score was developed which combines various scores and indicators (Table 16.2) [13].

If the total of points is ≥2, a difficult intubation should be expected. The Wilson score has higher sensitivity than the Mallampati score but often leads to an overestimation of difficult intubation. The same goes for a combination of both scores. Although a reliable prediction of intubation difficulties is not possible, regular use of the scores generates important team awareness of the problem of unexpected difficult airways. The use of algorithms, such as the ASA Difficult Airway Algorithm [2], is immensely helpful if an unexpected intubation or ventilation problem occurs (see Sect. 1.1.6 and Fig. 1.1).

Table 16.1 Modified Mallampati classification

Class	Visible structures
1	Full visibility of tonsils, uvula, faucial pillars, and soft palate
2	Visibility of hard and soft palate, upper portion of tonsils, and uvula
3	Soft and hard palate and base of the uvula
3	Only hard palate visible

>> On her way to the hospital, Ms. Hall took the 7.5 mg midazolam PO she had been prescribed because of severe anxiety. The monitor in the preoperative area revealed a heart rate of 92 beats/min and a blood pressure of 164/96 mmHg.

"*Are you still nervous?*" *asked Dr. Sven. Ms. Hall nodded and said, "Well, the pill worked well at first, but now I have the feeling that the effect is gone." Anesthesia tech Carol had already placed an IV, and Dr. Sven called his*

Table 16.2 Wilson score

Points	0	1	2
Weight [kg]	<90	90–110	>110
Range of motion in head and neck	>90°	90°	<90°
Maximum mouth opening [cm] or maximum jaw protrusion	>5 or lower jaw subluxation of upper jaw	<5 or lower jaw equal to over jaw	<5 or receding mandible
Protruding maxillary anterior teeth	Normal	Moderate	Severe
Receding chin	Normal	Moderate	Severe

A score of 2 or more points is indicative of a potential difficult intubation

attending Dr. Eldridge to tell him he was heading back to the OR.

Dr. Sven was preoxygenating Ms. Hall as Dr. Eldridge walked into the OR. "Please give 300 μg fentanyl, and in 2 min, 200 mg propofol," he said to Dr. Sven. Mask ventilation was less than desirable, even with the help of an oropharyngeal airway. Dr. Eldridge asked anesthesia tech Carol to give 50 mg propofol. Afterward Dr. Sven could ventilate the patient much better. Finally, Ms. Hall received 8 mg vecuronium. Dr. Sven was a little nervous, as he was handed the laryngoscope, but the intubation was unremarkable. "Cormack II," he announced loudly, and attending anesthesiologist Dr. Eldridge nodded he had understood. After auscultation to check the position of the endotracheal tube, anesthesia tech Carol secured the tube, and Dr. Sven placed a nasogastric tube. Dr. Eldridge commented, "I'll be moving on," and he was gone.

For maintenance of anesthesia, Dr. Sven chose sevoflurane 1 MAC in 50% oxygen. He set the ventilator to volume-controlled ventilation with a tidal volume of about 600 ml, a frequency of 12/min, and a PEEP of 7 cmH_2O and an I:E ratio of 1:1.5.

After the surgical prep, Dr. Buster placed the first trocar into Ms. Hall's abdomen. "Turn on the gas" he said to the circulating nurse. "Thomas, can you place the patient in reverse Trendelenburg?" Dr. Sven did as requested. "So far, so good! Now I just have to fill out the anesthesia record," thought Dr. Sven as he began to chart on the computer screen.

16.1.2 Which Gas Is Usually Used for a Pneumoperitoneum?

In laparoscopic surgery, carbon dioxide (CO_2) is standard. CO_2 has the benefit of not supporting combustion, as opposed to oxygen or nitrous oxide. In addition, CO_2 is highly soluble in blood, as opposed to other gases like nitrous oxide or helium. This offers protection against gas embolism. The peritoneum absorbs CO_2, which can then be eliminated by the lungs.

16.1.3 Explain the Effects of a Pneumoperitoneum!

With insufflation of gas into the abdominal cavity, the intra-abdominal pressure increases, and in addition some gas is absorbed. The effects of increased intra-abdominal pressure must be differentiated from the effects of CO_2.

16.1.3.1 Effects of Pressure

Pulmonary Effects

Increased intra-abdominal pressure leads to cranial displacement of the diaphragm, which compresses the lungs and reduces lung volume. The result appears similar to restrictive lung disease with a reduction in compliance. In volume-controlled ventilation, peak pressures increase, as well as the mean airway and plateau pressure. Compression of the lungs increases the risk of atelectasis formation and ventilation–perfusion mismatch with the resulting hypoxia.

Cardiovascular Effects

Venous return to the heart is dependent upon intra-abdominal pressure. As gas flows into the abdominal cavity, venous return increases due to compression and emptying of abdominal veins. As intra-abdominal pressure increases above 15 mmHg, the reduction in venous return increases, and cardiac output decreases. Systemic vascular resistance also increases with increasing intra-abdominal pressure. In the pulmonary circulation, compression of the lungs leads to an increase in vascular resistance.

Intra-abdominal pressures of <15 mmHg are well tolerated by healthy patients and patients with minimal comorbidities. ASA III or IV patients, however, can be adversely affected by the pneumoperitoneum. For these patients, the smallest amount of abdominal distention possible should be applied, or a gas-free or open technique should be used if hemodynamic or pulmonary complications arise.

Effects on Other Organs

The intra-abdominal and retroperitoneal blood flow decreases as intra-abdominal pressure increases. The hemodynamic effect is especially

pronounced when intra-abdominal pressures exceed >15 mmHg.

16.1.3.2 Effects of the Hypercapnia

The absorption of CO_2, without an adjustment in ventilation, leads to hypercapnia and respiratory acidosis. The amount of CO_2 absorbed by the system also depends upon other things in addition to intra-abdominal pressure. The largest amounts of CO_2 resorption are during extraperitoneal endoscopic operations, such as endoscopic extraperitoneal radical prostatectomy (see Case 31). Although hypercapnia directly causes systemic vasodilation, there is an indirect sympathetic stimulating effect, which increases heart rate, cardiac output, and arterial blood pressure. Additionally, the danger of cardiac arrhythmia increases. In contrast to CO_2's vasodilation effects in the systemic circulation, hypercapnia leads to vasoconstriction in the pulmonary circulation.

16.1.4 What Effect Does Patient Positioning Have on Pulmonary Function?

After induction of anesthesia, respiratory compliance is reduced and resistance is increased. The functional residual capacity decreases due to the diaphragm being displaced cranially and a decrease in the thorax diameter.

16.1.4.1 Supine Positioning

Due to the cranial dislocation of abdominal organs, lung volume is reduced. The decrease in functional residual capacity leads to a decrease in compliance, increase in respiratory resistance, and a possible impairment of gas exchange due to ventilation/perfusion mismatch. Gas exchange is especially impaired when the functional residual capacity volume decreases below the alveolar closing capacity.

16.1.4.2 Trendelenburg Position

The pulmonary physiological changes in the supine position are exacerbated by the Trendelenburg position. A further decrease in the functional residual capacity leads to increased atelectasis formation.

16.1.4.3 Reverse Trendelenburg Position

Compared to supine or Trendelenburg positioning, the reverse Trendelenburg position increases lung volume and compliance, thereby reducing respiratory resistance.

>> *Dr. Sven had just begun charting on the computer when the ventilator sounded a high pressure alarm. The preset pressure maximum of 30 cmH_2O had been exceeded.* "*Oh*," *he thought to himself*, "*I'd better adjust the ventilation*." *He decreased the tidal volume from 600 to 500 ml and increased the ventilation frequency from 12 to 15/min.*

16.1.5 Would You Have Adjusted the Ventilator Differently?

In Sect. 16.1.3, the effects of capnoperitoneum and the resulting CO_2 resorption was described. In order to avoid the adverse effects of CO_2 resorption and maintain normocapnia, the minute volume must be increased by about 20–30 % for the duration of the pneumoperitoneum. Choosing volume-controlled ventilation makes it easier to achieve this goal. The decision to reduce the tidal volume was appropriate, in order to avoid potentially dangerous airway pressures. However, the increase of the frequency of respiration was not sufficient and increased the minute ventilation only from 7.2 to 7.5 l.

>> *Even after readjusting the ventilator settings, the high pressure alarm sounded again. The ventilator was still delivering only about 400 ml as tidal volume. Dr. Sven raised his eyebrows and thought*, "*Then I will adjust the level of the high pressure alarm*." *He thought about it for a second then increased it to 35 cmH_2O. However, even under this new setting, the alarm sounded again.* "*Is there a problem*?" *asked Dr. Buster from the other side of the green drapes.*

Dr. Sven responded after a small pause "*I can't get a big enough tidal volume into the patient. How high have you got the abdominal pressure*?" *Dr. Buster glanced at the pressure display of the insufflator and answered*, "*13 mmHg – as always. Maybe the problem is the patient's build. The visibility isn't the greatest in here either*."

16.1.6 Is the Surgeon Correct?

Obesity influences the mechanics of respiration as well as gas exchange, mimicking restrictive lung disease. The cranial positioning of the diaphragm plays an important role, resulting in:

- Reduction in lung volume
- Reduction in compliance
- Increase in airway resistance

The reduction in compliance as well as anatomical changes, such as limited movement of the diaphragm, lead to increased work of breathing in spontaneously breathing patients. The reduction of compliance in volume-controlled ventilation leads to increased airway pressures. Visibility conditions during a capnoperitoneum with unchanged intra-abdominal pressures can be affected by obesity. The surgeon Dr. Buster could be correct.

>> As Dr. Sven adjusted and checked the ventilator once more, he noticed that the end-expiratory pressure of CO_2 ($CO_{2\text{-end-tidal}}$) was only 28 mmHg (normal: 35–45 mmHg). "Maybe I don't need to ventilate Ms. Hall as aggressively as I thought," he decided and reduced the tidal volume to 300 ml. Thereafter, there was no further alarm from the ventilator. Dr. Sven was relieved and again focused on the computer to continue filling out the anesthesia record. Five minutes later there was another alarm but this time for the blood pressure.

"Systolic blood pressure is too high," thought Dr. Sven. Simultaneously, the display showed a heart rate of 96 beats/min and a blood pressure of 172/93 mmHg. The S_PO_2 was 90%. Dr. Sven saw the $CO_{2\text{-end-tidal}}$ was 37 mmHg.

16.1.7 What Would You Do Next?

Ms. Hall showed signs of sympathetic system activation and borderline oxygenation.

The possible causes of sympathetic system activation are extensive. For example, inadequate analgesia or hypnosis is possible. During a laparoscopic surgery, it is not uncommon for the sympathetic system to become stimulated at about 5–10 min after CO_2 insufflation has begun, if the minute volume is not adequately increased (see Sect. 16.1.3). Taking the borderline oxygenation into account, a ventilation problem must be considered, despite the normal ($CO_{2\text{-end-tidal}}$).

>> Dr. Sven found the entire situation strange. He was sure that the hemodynamic changes did not come from inadequate analgesia, but just to reassure himself, he gave 200 µg fentanyl IV. Then he made space under the sterile drapes to auscultate the lungs. The breath sounds were hard to hear, but on the left side he couldn't hear anything at all.

16.1.8 You Now Know What the Problem Is, Don't You?

The quiet breath sounds were caused by the patient's anatomy and the minimal tidal volume. The one-sided auscultation indicates a secondary endobronchial intubation resulting from axial compression of the trachea due to the pneumoperitoneum [4, 6]. It is a rare but typical complication of laparoscopic procedures.

>> Dr. Sven was now sure that Ms. Hall was only ventilating one lung. As he inspected the endotracheal tube fixation, he found the 24 cm mark at the teeth. "Man!" he thought, "If you don't double check every little thing yourself…" He ripped the tape off the tube and pulled it back to the 20 cm mark. Ms. Hall responded with a light cough. "Hey, Thomas, I can't operate like this!" yelled Dr. Buster. "Give me a minute," called out Dr. Sven. He was in the process of securing the tube, and he was annoyed that he had forgotten to deflate the balloon before pulling out. "Carol does a far better job at taping the tube than I do," he complained silently to himself. Then he gave 70 mg propofol and an additional 2 mg vecuronium IV.

After correcting the tube position, the ventilation pressure was only 20 cmH_2O. The $CO_{2\text{-end-tidal}}$ was 58 mmHg. Dr. Sven increased the respiratory minute volume to 9.5 l, and as the end-tidal CO_2 normalized, the heart rate and blood pressure returned to normal as well.

Dr. Sven was relieved.

The laparoscopic cholecystectomy had been in progress for about 80 min, and the gall bladder was not yet out. Dr. Sven had given vecuronium

and additional fentanyl twice already, but the peak airway pressures were again steadily increasing. With a peak airway pressure of 35 cmH_2O, the cuff could no longer form a tight seal, and Dr. Sven felt it necessary to reinflate the cuff. He wasn't the only one unsatisfied with his performance. He noticed that the surgeon was struggling. As he reviewed the entire situation in his head, he noticed that he had not yet checked the degree of neuromuscular blockade.

16.1.9 What Types of Nerve Stimulation Can You Name?

Peripheral nerve stimulation determines the extent of the neuromuscular block (see also Sect. 12.1.4 and Fig. 12.1). In most clinical settings, the N. ulnaris is stimulated, with recording contraction of the adductor pollicis muscle. A supramaximal stimulus of 40–60 mA should be used. Possible stimulation patterns are listed below.

16.1.9.1 Single Twitch

Single twitch is a single stimulus with a duration of 0.2 ms. The clinical value is minimal.

16.1.9.2 Train-of-Four (TOF)

Four twitches within 2 s (2 Hz) are applied, each with a stimulation duration of 0.2 ms. The TOF is the most useful test. During surgeries requiring muscle relaxation, a maximum of two twitches should be measurable. The TOF ratio is calculated from the ratio of the fourth to the first twitch. Using visual or tactile assessment, twitches from about 0.5 are judged to be equally strong. More accurate, therefore, are the quantitative methods of assessment, such as the mechano-, electro-, or acceleromyography, which can reliably detect differences with TOF ratios of >0.5.

For extubation, a TOF ratio >0.8 is required; with values of less than 0.7, severe respiratory complications can develop. Even at 0.8, the pharyngeal muscles are still affected and the patient can develop upper airway obstruction.

16.1.9.3 Double Burst Stimulation (DBS)

In the double burst stimulation, three impulses of 0.2 ms duration and a frequency of 50 Hz are fired. After a pause of 750 ms, new bursts of three (DBS 3.3)/two impulses (DBS 3.2) are fired. The DBS allows better visual and tactile assessment, as compared to the TOF.

16.1.9.4 Post-tetanic Count (PTC)

The PTC allows assessment of a deep neuromuscular blockade (see Sect. 12.1.4).

>> As Dr. Sven opened the door of the OR to get the nerve stimulator in the adjacent supply room, the monitor alarm went off again. This time it wasn't the high pressure alarm, but a "leak." Now Dr. Sven could hear whistling and gurgling from the patient's direction. As he inspected the endotracheal tube, he saw immediately that the endotracheal tube fixation had come loose. "It's out!" he thought as panic came over him. He tried to push the inflated tube back in, but Ms. Hall began to cough. As loud as he could, he yelled through the open door into the supply room, "Carol!" She was stocking supplies and hurried immediately to his side. "The tube is out! We've got to reintubate!"

He pulled the rest of the tube out himself, disconnected it, reached for a face mask, and turned the O_2 to 100 %. It was of no use as mask ventilation was impossible. The S_PO_2 dropped quickly to 79 %. Anesthesia tech Carol had succinylcholine drawn up and a laryngoscope in her hand. "We can begin. All of the sux?" she asked.

Dr. Sven took the endotracheal tube which had just been removed and immediately inserted it slam dunk straight into the trachea. The S_PO_2 quickly increased as ventilation resumed and was soon over 90 %. Together, they secured the tube at 22 cm. "This is really a crappy case," thought Dr. Sven. "Hopefully it won't continue this way." He had hardly finished his thought when the ventilator's alarm sounded again. "Déjà vu," thought Dr. Sven. The display read, "High pressure alarm." He changed the ventilation to manual but could only move air in with a pressure >60 cmH_2O. The endotracheal tube must be obstructed. Dr. Sven took a suction device, but after inserting it for a few centimeters, he felt a resistance in the lumen of the tube.

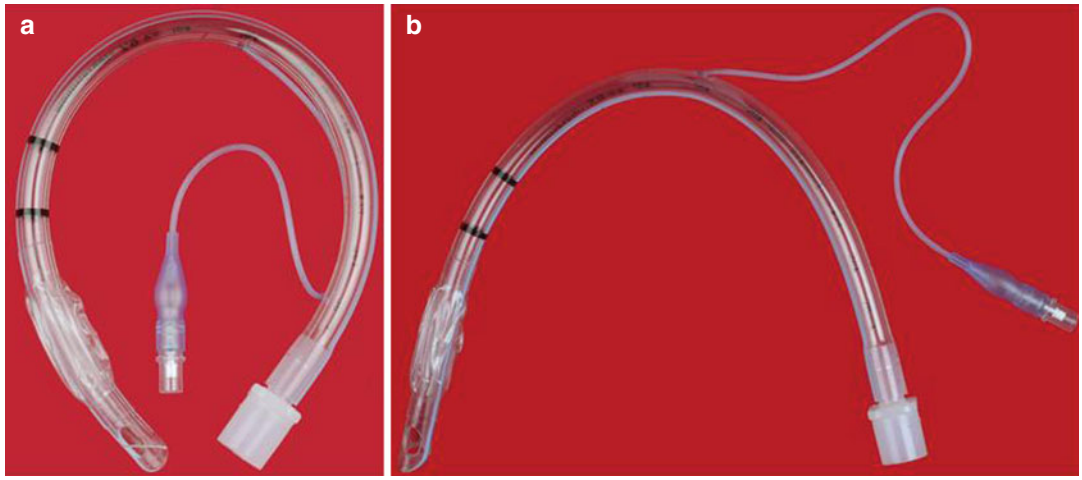

Fig. 16.1 Bending stability of polyvinyl tube at room temperature in the direction of the concave curve (**a**) and against the curve (**b**) (From Hübler and Petrasch [3], with permission)

16.1.10 What Just Happened Here?

Probing the endotracheal tube with a suction catheter is a good way to determine the patency of the tube and to remove secretions, if needed. Apart from detecting an obstruction in the lumen, one must consider a kink of the endotracheal tube [3]. The most commonly used polyvinyl endotracheal tubes are preformed and relatively stable at room temperature, even when they are bent against the preformed curve (Fig. 16.1).

The stability decreases when the polyvinyl is warmed to body temperature; small pressures against the curve of the tube can result in a kink (Fig. 16.2a). The weakest point of the tube is the exit point of the cuff inflation line. This point is at 18 cm, therefore within the oral cavity and not visible in intubated patients (Fig. 16.2b).

The temperature changes explain why ventilation problems occur with certain latency after intubation. With proper tube fixation (following the curve along the palate), natural anatomical step or sharp bend is nearly impossible. The tube was probably turned during reintubation. It is therefore important to note that the marking of the convex curve of the Mallinckrodt (Covidien) tube (light blue line) points nasally, even after fixation.

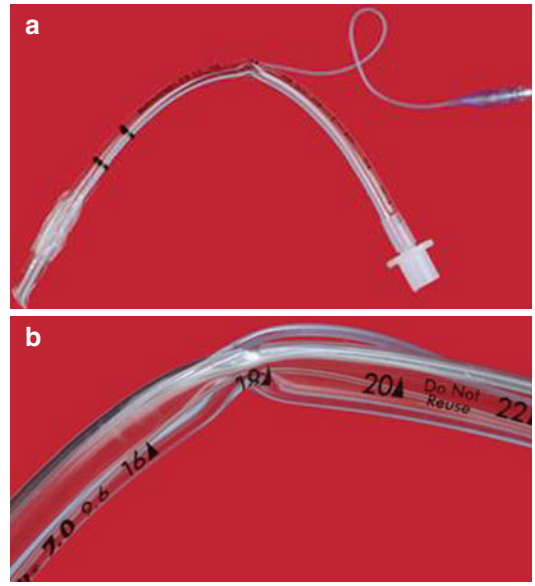

Fig. 16.2 Bending results of a polyvinyl tube after warming to 36 °C (96.8 °F). (**a**) Note the significantly smaller angle of bending before kinking as compared to Fig. 16.1. (**b**) Detail view of the location when kinks most often occur (From Hübler and Petrasch [3], with permission)

>> Dr. Sven couldn't believe that the tube was kinked. "Hand me the laryngoscope," he said to anesthesia tech Carol. As he opened the mouth he saw the problem. The tube had a kink in the mouth. Quickly he loosened the tape, and the problem was solved. "I hope that was the last difficulty of this case," he thought to himself as he sank down on his stool once more.

Attending anesthesiologist Dr. Eldridge entered the OR again. He wondered why the surgery was not yet finished. Turning to the surgeons, he

announced "I've called Dr. Harold; he should be here any minute to get things finished up in here." To Dr. Sven he said "Make sure that the patient is fully relaxed." Then Dr. Sven was alone again. As Dr. Harold entered the room, Ms. Hall received a maintenance dose of vecuronium. Dr. Harold took over the surgery and completed the cholecystectomy within 15 min. "That was fast," thought Dr. Sven. "Ms. Hall is still relaxed." He turned on the nerve stimulator, and to his total surprise, the neuromuscular blockade was completely gone.

16.1.11 Do You Have an Explanation for the Obviously Shortened Duration of Action for the Vecuronium?

The cytochrome P_{450} oxidase system is important in the metabolism of many drugs. It is composed of various types of enzymes, the so-called isoenzymes. Their activity differs between individuals due to genetic polymorphism. The isoenzymes are also influenced by medications which cause enzyme induction and inhibition.

Carbamazepine leads to induction of isoenzymes CYP3 and A4, which is responsible for the inactivation of a long list of anesthesia medications, including benzodiazepines, opioids, $5\text{-}HT_3$ antagonists, and non-depolarizing muscle relaxants with a steroid structure. The result is a short duration of action and a relative resistance to the neuromuscular blockade [1, 11].

>> Ms. Hall's extubation was unremarkable. Dr. Sven was so relieved as he brought her to the PACU with stable vital signs.

16.2 Case Analysis/Debriefing

16.2.1 What Are the Most Common Patient Complaints After Laparoscopy?

16.2.1.1 Nausea and Vomiting

Postoperative nausea and vomiting (PONV) is seen more often after laparoscopic surgery. The patients must be informed of the increased incidence, and action should be taken to reduce the incidence (see also Sect. 4.1.5 and Fig. 4.1).

16.2.1.2 Shoulder Pain

Postoperatively, about 30 % of patients report postoperative shoulder pain lasting for 2–3 days after laparoscopic surgeries. The cause is the CO_2, the stretch of the peritoneum, and irritation of the diaphragm. Treatment consists of pain relief, with opioid and non-opioid medications. The following preventative measures are often employed:

- Humidifying and warming the insufflation gas
- Slow introduction of gas and avoidance of high intra-abdominal pressures
- Mobilization of the leftover gas at the end of the surgery
- Avoidance of CO_2 as an insufflation gas
- Preemptive administration of anti-inflammatory agents
- Intraperitoneal administration of local anesthetic [8]

16.2.2 Which Medical Errors Do You See in the Presented Case?

Premedication/Choice of Anesthetics

Ms. Hall had a well-controlled seizure disorder. Immediately before beginning anesthesia, she complained that the premedication had no effect on her. As described in Sect. 16.1.11, benzodiazepine metabolism is also influenced by carbamazepine. The duration of action of midazolam (a short-acting benzodiazepine) is further decreased. If an oral benzodiazepine is desired, it is better to give such patients a longer-acting benzodiazepine such as lorazepam. Otherwise, titrate IV midazolam to effect just prior to taking the patient back to the OR.

It is known that propofol can cause – even in patients without a seizure disorder – clinical signs which resemble focal seizures [12]. This can occur:

- During anesthetic induction (34 %)
- During maintenance (of anesthesia) (3 %)
- During emergence from anesthesia (40 %)
- Postoperatively, after a latency period (23 %)

Nevertheless, propofol can be safely used in patients with seizure disorders. In this case, the preoperative sedative had lost its effectiveness prior to induction. IV midazolam is often recommended before administering propofol in order to decrease the chance of a seizure.

While sevoflurane may cause seizure activity in the EEG [5], clinical use of sevoflurane does not increase seizure activity in patients with epilepsy.

16.2.2.1 Ventilation Adjustments/ Double-Checking Tube Placement

These issues were already discussed in Sect. 16.1.5. Dr. Sven failed to set Ms. Hall's respiratory minute volume high enough, and he didn't check the tube placement early enough.

16.2.3 Which Systems Failures Can You Find in the Presented Case?

16.2.3.1 Securing the Tube

After reintubation, the tube was inadequately secured (see Sect. 16.1.10). Dr. Sven and anesthesia tech Carol both failed to notice that the tube had been rotated. A meaningful solution would be to set SOPs so that after securing the tube, the proper positioning would be rechecked.

16.2.4 You Seem to Have Already Taken a Step Back! What Should Be Done Now?

The major information Dr. Sven used in his decision-making was the $CO_{2\text{-end-tidal}}$ value. He didn't consider Ms. Hall's remarkably high airway pressures and/or the minimal tidal volumes as important, values which themselves could have been tolerated at normal pressures. Only as the S_PO_2 decreased due to the ventilation problems (endobronchial intubation) and the blood pressure increased due to hypercapnia did Dr. Sven take the most important action: check the placement of the endotracheal tube. Due to being overwhelmed, Dr. Sven limited his attention to – in his own opinion – the most important action. In this way he was able to quickly make decisions.

The disadvantage of abbreviated decision-making is obvious: once a cause is decided, due to the selective filtration of information – primarily information which fits the set model – it is very difficult to depart from the set explanation, even after information has been obtained which contradicts the set hypothesis. In this example, the recognition of the kink in the tube came too late, but thankfully his hypothesis could be revised.

Psychologically, **situational awareness** is divided into three parts: perception, comprehension, and projection [10]. In this case, Dr. Sven perceived a great deal, but he did not interpret it and he didn't understand the meaning. Which strategy should have been used to improve the situational awareness?

16.2.4.1 Thinking About Thinking

Which hypothesis should I explore? What data do I have? What do I want to avoid? Have I forgotten any information? Thinking about thinking, otherwise known as **metacognition**, sets activated thought processes into question. It is the second step after the "Step back" (see Sect. 7.2.3). Your own expectations and settings are consciously questioned. If then an additional conscious search for information pertaining to the "worst-case scenario" is performed, you can then recognize subconsciously suppressed (due to fear) and misinterpreted information.

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Case 17: The Second Inguinal Hernia

17

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Contents

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17.1 Case Introduction

>> Mr. Jones, with his medical record in hand, left the surgery admissions office and set off on the search for the preanesthesia clinic. The secretary had given him directions, but it still took a while before he arrived. The old building had so many hallways and doors; climbing the steps really exhausted him. He had to take a break after every single step. He hardly had time to fill out the yellow preoperative patient information form before his name was called.

"Good day! I'm Dr. Deborah," the anesthesiologist greeted him, shaking his hand. "I see that you have an inguinal hernia repair planned for tomorrow. Before we get into the anesthesia, let me have a look at your answers on the preanesthesia form." Mr. Jones held the form,

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properly filled out, for the doctor to take. Dr. Deborah obtained the following information:

- *72 years old, 182 cm, 84 kg*
- *Thyroidectomy 4 years ago*
- *Aspirin allergy*
- *Systemic hypertension*
- *Muscle weakness*
- *Nonsmoker*
- *Regular medications: thyroxin, pyridostigmine, ramipril*

The lab findings were all within the reference range.

17.1.1 Which Type of Muscle Weakness Did Mr. Jones Probably Have, and What Do You Know About the Pathophysiology of the Disease?

Mr. Jones is taking pyridostigmine, a cholinesterase inhibitor, which delays the inactivation of acetylcholine (ACh) in the synaptic space at the motor end plate. The muscle weakness is myasthenia gravis (MG).

MG is an autoimmune disease, in which antibodies are produced against the ACh receptors on the motor end plates or, in rare cases, against the muscle-specific tyrosine kinase [4]. As a result, the ACh receptor is no longer available for signal transduction. Additionally, the constant immune activity leads to a loss of ACh receptors. The clinical correlation is muscle weakness, which increases over time. The cholinesterase inhibitor administration lessens the symptoms but cannot stop progression of the disease. The cause of MG is not fully understood. In about 70 % of cases, there is an association with thymus disease. In such cases, a thymectomy might improve prognosis.

>> After Dr. Deborah asked the right questions, Mr. Jones explained that he had MG for 7 years. "That's the reason they took my thyroid out, but the symptoms haven't gotten any better," he said. "When I take my pills, I'm OK, but on the way here I had to stop for a few breaks." Dr. Deborah nodded. "Was your thymus also removed?" she asked. "I can never remember what that was called; I had never heard of this organ before," answered Mr. Jones. He went on to say that his blood pressure was well controlled and that he really didn't want to have another operation but that he was having so many problems with his hernia.

Dr. Deborah was satisfied with the information she had obtained. She recommended spinal anesthesia for Mr. Jones and obtained informed consent. "You can take your medications as usual, and the muscle therapy will not need to be interrupted," she explained. Mr. Jones was relieved. Dr. Deborah prescribed 3.75 mg midazolam PO as the preoperative sedative. On the preop sheet, she wrote "Trigger-free anesthesia."

17.1.2 Are You in Agreement with the Premedication?

In general, it is good to avoid general anesthesia in MG patients. The decision for the spinal anesthesia is therefore correct. Even so, the anesthesiologist overlooked a few things and made some errors.

17.1.2.1 Cardiovascular Evaluation

Mr. Jones reported that he paused many times on the way to the preanesthesia clinic.

Dr. Deborah didn't question further; therefore, she didn't know if stops were due to MG or cardiovascular problems. This patient has such serious muscle weakness, that he can't exercise. Clinical criteria for coronary heart disease or heart failure are therefore not reliable.

The American College of Cardiology/American Heart Association guidelines [6] on perioperative cardiovascular evaluation (see Sect. 5.1.1) recommend a 12-lead ECG be performed due to his age and hypertension. Preoperative echocardiography should be considered, given the poor exercise tolerance [3].

17.1.2.2 Seriousness of the Myasthenia

The questioning of Mr. Jones about his MG was inadequate. Table 17.1 shows the division of MG stages, according to Osserman and Genkins [10].

Table 17.1 Classification of myasthenia gravis

Type	Characteristic
I	Ocular myasthenia
IIa	Light, generalized form
IIb	Severe generalized form with involvement of the faciopharyngeal and respiratory muscles
III	Acute, rapidly progressing generalized form with respiratory muscle involvement
IV	Late type with generalized symptoms, which arose from type I or II within the past 2 years
V	Defect myasthenia, progression of type II or III

According to Osserman and Genkins [10]

With type IIb, respiratory musculature is involved so that great care is required. As with all regional anesthesia techniques, informed consent for possible general anesthesia should also be obtained in case the block doesn't provide sufficient anesthesia or an intraoperative emergency occurs. An additional risk factor for MG patients is postoperative mechanical ventilation, as outlined in the overview [7, 8].

17.1.2.3 Risk Factors for Postoperative Ventilatory Assistance in MG Patients

Note:

- Disease duration of >6 years
- Chronic lung disease
- Pyridostigmine dose >750 mg/day
- Vital capacity <2.9 l
- Osserman classification III–IV

Mr. Jones had suffered from MG for over 6 years; therefore, he has at least one risk factor for postoperative mechanical ventilation following general anesthesia.

Pyridostigmine treatment is usually given at bedtime, as a sustained release tablet, and during the day every 3 h, beginning at 6 a.m. A cumulative daily dose of 750 mg is quickly reached. Dr. Deborah didn't ask specifics; therefore, she couldn't note that on the preoperative evaluation. A definite classification of the disease according to Osserman classification can't be determined from the information given.

17.1.2.4 Pulmonary Function

Furthermore, pulmonary function tests were not ordered or performed. Vital capacity of <2.9 l further increases the risk of postoperative mechanical ventilation after general anesthesia [2]. A preoperative, objective evaluation of pulmonary function should be done in MG patients.

17.1.2.5 Informed Consent About the Possible Post-op Mechanical Ventilation

Even when regional anesthesia is planned, informed consent for general anesthesia is necessary. In patients with MG, the additional risk of postoperative mechanical ventilation should be described in the informed consent discussion.

17.1.2.6 Premedication

Benzodiazepines have a central-acting myotonic effect. The prescription for midazolam for preoperative sedation is contraindicated.

17.1.2.7 Trigger-Free General Anesthesia

The order for a trigger-free general anesthesia is incorrect. There is no association between MG and malignant hyperthermia.

>> Dr. Mallory felt well prepared as he entered the OR that morning. His fellows had called him from the preanesthesia clinic and told him about Mr. Jones. It wasn't every day that the anesthesiologists had an MG patient; therefore, Dr. Mallory had read up on the subject the night before. His board certification exam was a few years ago, and he noticed more and more often that his theoretical knowledge had decreased over time. "Bummer," he thought, as he noticed that Mr. Jones was scheduled for a spinal, "I already prepared myself for general anesthesia, studied up on the muscle relaxants and monitoring the neuromuscular blockade, and now I can't use a bit of what I learned."

He greeted Mr. Jones; the anesthesia technician Donald already had him sitting up for the spinal anesthesia. Dr. Mallory glanced at the preanesthesia evaluation, to see what the daily dose of pyridostigmine was. "240 mg at night, then 6 × 120 mg during the day; makes a total of 960 mg," he mumbled to himself. The prescribed midazolam was not given to Mr. Jones. Dr. Mallory had called the preoperative check-in

area from home this morning to cancel the order.

After proper preparation, Dr. Mallory placed the spinal needle into the subarachnoid space at the level of L_4/L_5 and injected 2.4 ml bupivacaine 0.5 % hyperbaric and 10 µg fentanyl. "Finished! You may lie down again," he said to Mr. Jones. After 10 min, the spinal level reached T_{12}. That was the bad news. The good news was that the sympathicolysis had no effect on the vital signs: blood pressure was unchanged at180/90 mmHg, and the heart rate remained at 65 beats/min.

17.1.3 What Would You Do Now?

The dispersal of a sensory and motor block after intrathecal injection of a local anesthetic is not entirely predictable. An important factor is the level of the puncture [11], which was rather low in Mr. Jones. Puncture below the lordosis of the lumbar region aids caudal dispersion [12] so that changing position can influence dispersal of the local anesthetic. Therefore, Mr. Jones should be positioned in the Trendelenburg position in order to raise the level of the block.

>> Mr. Jones was almost in 30° Trendelenburg position as the surgeon Dr. Martin entered the OR. "You haven't done a spinal, have you?" he questioned Dr. Mallory. "That's probably not going to work. Mr. Jones has a bit of bowel in the hernia." Then he went to scrub. "Thanks a lot for the information!" thought Dr. Mallory. The cranial dispersal of the spinal anesthesia had reached the T_6 level, and Dr. Mallory returned the patient to the supine position. Mr. Jones's blood pressure had decreased to 140/80 mmHg and his heart rate was unchanged. He was becoming anxious. He hadn't understood everything, but he did pick up on the fact that the surgeon was upset about something.

The surgery began. Dr. Martin made an incision in the inguinal region, and Mr. Jones had no pain. The hernia was actually rather large, and Dr. Mallory was annoyed with himself for not obtaining more detailed information earlier. After about an hour into the procedure, Dr. Martin mobilized the hernia sac. All of a sudden, the heart rate dropped to 40 beats/min, blood pressure 90/55 mmHg.

17.1.4 Is Mr. Jones Experiencing a Cholinergic Crisis? What Exactly Is a Cholinergic Crisis?

A cholinergic crisis is triggered by an overdose of cholinesterase inhibitors. The symptoms are:

- Bradycardia
- Warm, red skin
- Miosis
- Hypersalivation
- Agitation, confusion
- Abdominal pain, diarrhea

Since Mr. Jones had not received an extra dose of cholinesterase inhibitor, a cholinergic crisis can be ruled out.

>> Mr. Jones moaned because of extreme pain. Just as the surgeon Dr. Martin had warned, the spinal wasn't sufficient. "Could you please do your job and anesthetize the patient? I can't operate like this!" complained Dr. Martin. Dr. Mallory glanced at the surgical area: bowel loops were hanging out, and Dr. Martin looked as if he was untangling them. Mr. Jones moaned in pain again. Dr. Mallory felt sick. He must get help and quickly. He called anesthesia technician Donald, who was currently helping in the next OR. Then he gave Mr. Jones 1 mg atropine, 100 µg phenylephrine, and 200 µg fentanyl IV. The circulatory status improved immediately, but the S_PO_2 decreased to 85 %. Dr. Mallory interrupted his preparation for induction/intubation, turned off the monitor alarm, grabbed a mask and filter from the anesthesia cart, turned on the fresh oxygen, and began to preoxygenate Mr. Jones. The S_PO_2 increased to 96 %.

Anesthesia technician Donald entered the room. "Do you have to intubate?" he asked.

Dr. Mallory only nodded. With practiced efficiency, tech Donald prepared everything. "We

can begin," he informed Dr. Mallory. Just in the nick of time, because Mr. Jones was again agitated and moaning. As ordered by Dr. Mallory, tech Donald gave 160 mg propofol, 200 µg fentanyl, and 80 mg succinylcholine IV. After the last medication was in, Dr. Mallory shouted "Dammit!" Shortly thereafter, he intubated Mr. Jones without difficulty.

17.1.5 What Was the Reason for Dr. Mallory's Outburst? Would You Have Done Something Differently?

As already discussed in Sect. 17.1.2, there is no association between MG and malignant hyperthermia. There was nothing wrong with the administration of succinylcholine. However, in MG patients, the effect of succinylcholine is altered: due to a minimal number of functioning ACh receptors, the onset of effect is prolonged. Among other irregularities, an increased dose is necessary. In summary, the effect is very unpredictable.

Dr. Mallory was annoyed about something else. Almost all non-depolarizing muscle relaxants can be used safely in patients with MG. However, the dose must be decreased significantly – for example, by 10 % – and titrated. In order to properly titrate, quantitatively monitoring neuromuscular function must be done before administering the muscle relaxant so that the appropriate dose can be estimated in advance [9]. The most elegant solution is an acceleromyography with stimulation of the ulnar nerve. After taking a reference measurement without the effect of a muscle relaxant, continual neuromuscular monitoring is performed, typically using the train-of-four (see Sect. 12.1.4).

17.1.6 What Else Must Be Considered When Administering Succinylcholine to Patients with MG?

The cholinesterase inhibitor influences the activity of the plasma cholinesterase so that the duration of action for succinylcholine can be extended. For the same reason, mivacurium is contraindicated.

>> As Dr. Mallory documented the induction in the anesthesia record, he whispered quietly to himself. The conversation went like this: "You idiot! Why did you bother to review everything last night, when you do it all wrong in the deciding moment? You should give back your board certification!"

At least the surgeon Dr. Martin was finally satisfied; he could work freely without being interrupted. To maintain anesthesia depth, Dr. Mallory chose a propofol infusion, set at 150 µg/kg/min.

17.1.7 Would You Have Made the Same Choice?

Propofol can be used safely in patients with MG. Due to propofol's lack of muscle relaxation, volatile anesthetic agents are often better. It is important to make sure that there is sufficient depth of anesthesia, because avoiding the benzodiazepine increases the chances of awareness (see Sect. 8.1.10).

>> Fifteen minutes had passed and with it the surgeon's satisfaction. "The patient is moving!" he exclaimed to Dr. Mallory. "Thank goodness," thought Dr. Mallory who had already been considering how he was going to treat the postoperative residual curarization. Mr. Jones promptly received a propofol bolus of 50 mg, and Dr. Mallory increased the propofol infusion to 200 µg/kg/min. The success was short lived. Ten minutes later Dr. Martin complained of nonstatic conditions: "I feel like I'm operating during an earthquake."

17.1.8 What Would You Suggest?

Dr. Mallory might be lucky if one of his colleagues gives him a coffee break and gets him off the hook by taking over the case. Besides this wishful thinking, there are other options:

17.1.8.1 Administration of Muscle Relaxants

Due to the MG, this would not be a good choice and probably not even necessary for this operation. In addition, Mr. Jones' movements could be a result of inadequate anesthetic depth (see Sect. 8.1.10).

17.1.8.2 Deepen the Hypnosis

Increasing the depth of the hypnosis is a tried and true solution. The target hypnotic depth is the level which suppresses spontaneous movements. This can be reached by increasing the dose of propofol or by changing to an inhaled anesthetic.

17.1.8.3 Analgesic Administration

It is highly likely that Mr. Jones' spontaneous defensive movements are triggered by pain. An opioid should be administered as part of balanced anesthesia. After the surgery, Mr. Jones will only have limited pain at the surgical site due to the spinal fentanyl. Therefore, a short-acting opioid should be given.

>> Dr. Mallory supplemented the general and spinal anesthesia by giving Mr. Jones a continual infusion of 1 µg/kg/min remifentanil. There was then complete satisfaction from everyone involved. Mr. Jones couldn't exactly answer, but at least he didn't move any more, which was taken as an expression of his satisfaction.

One hour later, after a total surgical time of 2 h, the hernia was repaired. Dr. Mallory calculated silently: "Mr. Jones received one pyridostigmine dose 3 h ago. It is time for the next one, which would be 120 mg pyridostigmine PO, or 4 mg IV. I better just give him half – you never know."

17.1.9 What Do You Think of This Decision?

As with diabetics, the goal of perioperative management in patients with MG is administration of regular medication again as soon as possible [3]. It must be noted that the requirement for the cholinesterase inhibitor has been reduced. Dr. Mallory's calculation is correct: 1 mg IV is equivalent to 30 mg PO. Maximum muscle strength is desired at the time of extubation, in order to safely avoid aspiration. Even though this recommendation is not in any text book, many anesthesiologists administer half of the regular dose before extubation. Neuromuscular monitoring is highly recommended, in order to prevent an overdose or a cholinergic crisis.

>> Dr. Mallory gave Mr. Jones 2 mg pyridostigmine IV and turned off the anesthetic agents. Ten minutes later the endotracheal tube was removed without complications. He brought the patient into the PACU and secured an ICU bed for the next 24 h, just to be safe. One hour later, Dr. Mallory returned to the PACU, where he was immediately spotted by Mr. Jones. The ICU team hadn't yet had time to pick him up. Mr. Jones spoke up "The general anesthesia was a great idea. I slept well and didn't feel a thing. Now I think that I will have the other side done as well, but only if you promise to put me to sleep!" Quite humiliated, Dr. Mallory mustered up a friendly smile and agreed. Actually he very much wanted to know if Mr. Jones had had an awareness episode. Evidently not…

17.2 Case Analysis/Debriefing

17.2.1 Additional Food for Thought, Unrelated to the Case: What Does It Mean for the Baby When the Mother Has Myasthenia Gravis?

The antibodies against the ACh receptors are IgG and therefore can pass through the placenta. Depending upon the severity of the mother's disease, about 20 % of newborns develop a so-called transitory neonatal myasthenia gravis [5]. The main symptoms are lethargy, bradypnea, and flaccid muscle tone. In 2/3 of the affected newborns, the symptoms occur within the first 4 h; in one-third the symptoms occur up to 4 days later. The reason for the latency time is the half-life of the mother's cholinesterase inhibitor, which also crosses the placental barrier. The symptoms usually persist for about 3 weeks, which correlates to the half-life of IgG antibodies.

After the development of MG symptoms, the newborn must therefore receive intensive care and monitoring and perhaps receive cholinesterase inhibitor therapy.

17.2.2 You Are the Anesthesiologist on the Labor and Delivery Ward in a Hospital Which Lacks a Neonatologist. Who Cares for the Newborn Delivered by C-Section if There Are Problems Adapting to Ex Utero Life?

As a rule, pregnant patients who have myasthenia gravis should deliver in a high-risk obstetric center with a neonatal ICU, but sometimes there is no time for the transfer. If a neonatologist is not available, a nurse with neonatal resuscitation training should care for the baby in an emergency, until the emergency neonatologist or transport team arrives. The anesthesiologist and obstetrician are responsible for the mother's care, and they may not be able to leave her to care for the newborn, even when a neuraxial anesthetic is functioning adequately [1]. In rare circumstances in a stable case, the anesthesiologist may need to help in the resuscitation and to leave the monitoring of the anesthesia to a nurse or anesthesia assistant. The decision as to when this is possible is left up to the anesthesiologist, who must keep in mind that his/her primary responsibility is to care for the mother. Ideally, if available, another anesthesiologist should care for the baby.

17.2.3 Which Medical Errors Do You See in the Presented Case?

Most of the areas needing improvement have been discussed already.

17.2.3.1 Quality of the Preoperative Evaluation

See Sect. 17.1.2.

17.2.3.2 Choice of Anesthesia

The anesthesiologists were not informed about the extent of the planned hernia surgery. With proper knowledge, such as knowledge gained through a physical examination or through preoperative discussion with the surgeon, epidural anesthesia could have been seen as the better choice, in order to minimize the chance of a general anesthesia.

17.2.3.3 Neuromuscular Monitoring

The significance of quantitatively assessing neuromuscular blockade was already demonstrated in Sect. 17.1.5. Even though it was neglected before the intubation, neuromuscular monitoring should have been done after Mr. Jones began to move.

17.2.4 Which Systems Failures Did You Find in the Presented Case?

17.2.4.1 Information

An important systems failure was the anesthesia team's inadequate knowledge about the extent of the planned hernia repair. One inguinal hernia repair isn't always the same as another inguinal hernia repair. The anesthesiologist should obtain exact information, through studying the patient medical record, history, and physical exam. In addition, the surgical team should inform the anesthesia team about any unusual characteristics – e.g., the extent of the hernia, presence of bowel, or whether it is incarcerated. If the surgical team fails to volunteer any unusual aspects, the anesthesiologist should specifically ask the surgeon if anything unusual is expected.

17.2.4.2 Preparation of the Anesthesia Team Before the Day of the Surgery

Preparations to perform a quantitative assessment of the neuromuscular blockade take time. This should have been done during the first hour of the surgery. After all, the surgeon gave a warning in the beginning that general anesthesia would be necessary. The same goes for the preparation to rapidly induce general anesthesia and intubate. As a rule, every regional anesthesia requires preparation for general anesthesia and endotracheal intubation, because one must act quickly should an unexpected emergency arise.

17.2.4.3 Beds on the Intensive Care Unit

There was no bed on the intensive care unit reserved for postoperative monitoring. Organizational responsibility must be clearly set by binding agreements.

17.2.5 Dr. Mallory Forgot to Monitor the Neuromuscular Blockade, Despite His Mental Preparation. How Could He Have Avoided This Mistake?

Dr. Mallory had prepared himself in advance for caring for his patient with MG. He was a bit annoyed that Mr. Jones didn't want general anesthesia. Despite this, he forgot to apply the needed monitoring at the correct moment. How did this happen? Could he have prevented this mistake?

At the time he decided that the spinal anesthesia needed to be converted to general anesthesia, Dr. Mallory was concentrating on several factors at once: bradycardia, hypotension, hypoxemia, and preparation for the induction of general anesthesia and endotracheal intubation. This acute stress caused him to forget his planned action: neuromuscular monitoring.

Forgetting goals and intended actions which were previously decided upon (and planned to be carried out later) occurs often in high-stress work situations. It has a name: **prospective memory loss**. The strategies listed in the Overview help to reduce the chances of such mistakes.

17.2.5.1 Strategies to Help Reduce the Chances of Possible Prospective Memory Loss

- Verbal elaboration so that intention is maintained within consciousness
- Open and loud communication of the planned actions so that all team members have formed a mental picture of the plan
- Frequent reevaluation of the situation
- The use of external memory aids, such as notebooks and checklists, which indeed are only situationally rendered and must be worked through

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Case 18: Hip Fracture

18

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Contents

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18.1 Case Introduction

>> Dr. Hugh was almost finished with his anesthesiology residency and had already seen a lot. He felt that he could hold his own with the other specialists, many of whom were slow in reacting to problems. "You must believe in your competence and make decisions when the time arises" was his theory. In this manner, he had already mastered many tricky situations. Appropriately, the word in the department was "Dr. Hugh will take care of it!"

On this Sunday he was on call. After a substantial breakfast in the staff room, he was asked to care for an older lady who was in the emergency room (ER). Ms. Martinez had been in her apartment when she tripped over the carpet and had such a bad fall that she fractured her hip. The 80-year-old was brought to the hospital by

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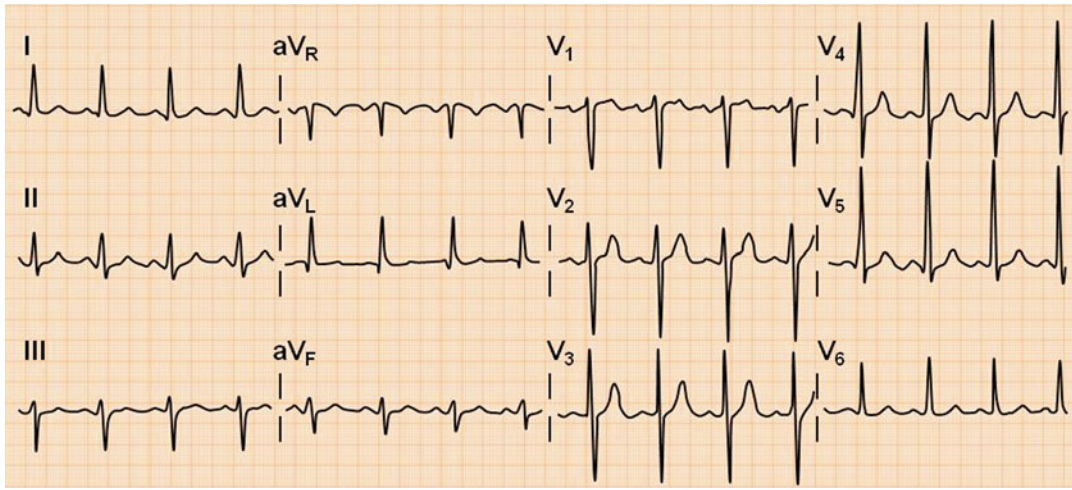

Fig. 18.1 The ECG shows a left axis deviation and a regular sinus rhythm. The only abnormality is the Sokolow–Lyon index (S in V2 + R in V5) of >35 mm, indicating left ventricular hypertrophy

two strong medics from the local ambulance service. The orthopedic surgeon had already determined that the fracture should be treated with dual head prosthesis.

"*Thank goodness I could crawl to the telephone*; *if I hadn't made it*, *I would have been laying there for a long time*," *she told Dr. Hugh*. "*I live alone and care for myself. Why does this have to happen to me now*, *at my age*?" *Dr. Hugh was in a good mood after his breakfast and took a moment to listen to the old lady's woes. Now he had heard enough*, *and he wanted to get to work on the preoperative evaluation and informed consent. He interrupted Ms. Martinez with an abrupt question. She stared at him surprised and speechless. Finally she answered his question about her past history as follows*: "*Young man*, *believe me*, *I have never ever been in a hospital before. I had my three babies at home. As far as pills go*, *I take one for blood pressure and one to thin my blood. My physician*, *Dr. Scheib on Redbush Street*, *said that the pills wouldn't hurt me*." *Actually the medics had measured a blood pressure of 210/120 mmHg*. "*That must have been due to pain and stress*," *thought Dr. Hugh. Her last oral intake was 8 h ago. She denied other preexisting conditions and allergies. Dr. Hugh reviewed the lab values*, *which were all unremarkable. He also reviewed the ECG which the ER physician had obtained.*

18.1.1 How Would You Evaluate the ECG in Fig. 18.1?

The answer is in the legend of Fig. 18.1.

>> "*Doctor*, *I am really very afraid that I won't be the same again when I wake up. My neighbor went through something like this last year. She couldn't ever be alone again after the anesthesia*, *and had to move into a nursing home*."

18.1.2 What Forms of Perioperative Cerebral Dysfunction Do You Know?

Perioperative cerebral dysfunction occurs in all age groups but is more often seen in older patients. Clinically, there are three types of dysfunction [9]: emergence delirium, postoperative delirium (brief reactive psychosis), and postoperative cognitive dysfunction.

18.1.2.1 Emergence Delirium

Emergence delirium occurs immediately following emergence from general anesthesia. All age groups are affected, but it occurs most commonly in children and adolescents. The symptoms of emergence delirium are extensive and involve changes of personality, perception disorders,

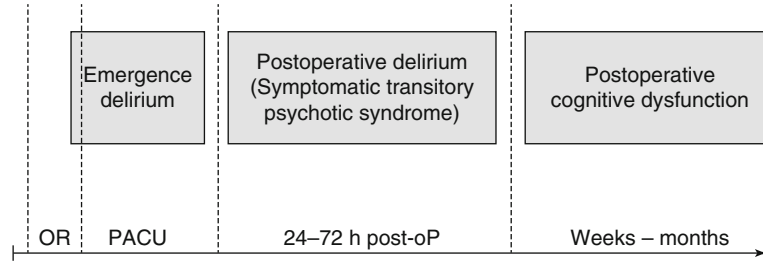

Fig. 18.2 Occurrence of perioperative cerebral dysfunction. The acute delirium is usually observed in the PACU or already in the operating room (*OR*). After a symptom-free interval, brief reactive psychosis may appear. There may be no sharp differentiation as the line is crossed to postoperative cognitive dysfunction (Adapted from Silverstein et al. [9] with permission)

disorientation, and cognitive disorders. The acute delirium imitates the excitation stage of anesthesia with ether.

The hyperactive form is usually clinically apparent, which typically keeps the personnel in the PACU busy. The hypoactive form is seldom recognized, since the patients withdraw to their inner selves. Emergence delirium has a short duration, and the affected patients later participate normally in their environment.

Since acute delirium is only seldom seen after regional anesthesia, many anesthesiologists incorrectly assume that the incidence of perioperative cerebral dysfunction after regional anesthesia is less than after general anesthesia.

18.1.2.2 Postoperative Delirium (Transient Reactive Psychosis)

Postoperative delirium develops usually after a period of clarity and awareness, typically on the first to third post-op day. The brief reactive psychosis is usually fully reversible with an average duration of a few hours or days, but it can also last for weeks and months. It is characterized by an acute onset with reduction in awareness of the environment and a disturbance in attention.

The incidence of postoperative delirium in older patients after general anesthesia is about 5–15 % [3], after hip surgeries about 35 % [4], and can also occur independent of surgery in internal medicine patients. The risk factors are age of >70 years, hearing loss, visual disturbances, malnutrition, presence of a urinary catheter, fixation devices, electrolyte disorders, volume deficiency, blood transfusions, and severe postoperative pain. Contrary to popular belief, the choice of anesthesia technique has no effect on the incidence [6].

18.1.2.3 Postoperative Cognitive Dysfunction

The term postoperative cognitive dysfunction describes deterioration in cognition, which arises following anesthesia and surgery. With emergence and postoperative delirium, there are also changes in behavior; these may not be present in postoperative cognitive dysfunction which makes the diagnosis more difficult. The comparison of the pre- and postoperative status is the deciding factor, which is seldom measured and has a high interindividual variability.

Various studies conclude that 25 % of elderly patients experience a cognitive disorder within the first 10 days after an operation. Furthermore, in 10 % of the older patients, the disorder is present 3 months after the operation and is gone at the 1-year point [9]. The one and only certain risk factor is the age of the patient. Also, the choice of anesthesia techniques has no influence on the incidence. Some data suggest that postoperative delirium is associated with postoperative cognitive dysfunction [10].

The various types of perioperative cerebral dysfunction differentiate from one another according to the time of the appearance (Fig. 18.2).

>> Dr. Hugh was uncomfortable with the topic that Ms. Martinez had raised. As an anesthesiologist, he usually had little to do with it. "I promise to carefully provide a very mild and gentle anesthesia for you. Everything will be OK" was his response.

18.1.3 Which Types of Anesthesia Are Possible for This Operation, and What Are the Pros and Cons?

The operation could be done with spinal/epidural anesthesia or under general anesthesia.

18.1.3.1 Spinal Anesthesia

A spinal anesthesia is technically easy, has a high success rate, and generally has a low rate of complications. Nowadays, reduced levels of local anesthetics are given resulting in minimal cardiovascular changes. The analgesia lasts into the postoperative phase, which many patients find comfortable. Spinal anesthesia might be especially beneficial in the presence of pulmonary or cardiac diseases which increase the risk of general anesthesia.

All regional anesthesia techniques have the disadvantage of requiring patient cooperation. This applies to the period of time required for placement – patients must sit or lie on their side for the placement – as well as for the surgery. The noises of the OR can be very upsetting for some patients. Spinal anesthesia has a limited duration of action and a less than 100 % success rate. In patients with blood clotting disorders/coagulopathies, the indication must be strictly evaluated. From the surgical point of view, a disadvantage of spinal anesthesia is delayed neurological evaluation of the operated leg.

18.1.3.2 Epidural Anesthesia

In epidural anesthesia, possible circulatory system changes occur more slowly than with spinal anesthesia. Redosing the catheter is possible at any time, should the surgery become prolonged. In addition, the catheter can be used for postoperative pain therapy, and systemic side effects of other pain medication are avoided.

A small disadvantage is that the placement is technically more challenging than spinal anesthesia. Rare complications such as hematomas or abscesses are more common. Care of the catheter on the ward is time-consuming.

18.1.3.3 General Anesthesia

As opposed to the regional anesthesia techniques, general anesthesia requires no cooperation by the patients. There is no time limit on the duration of the surgery, and neurological assessment of the extremity which was operated upon is possible usually right after the surgery.

Listing all possible disadvantages of general anesthesia would fill up this entire book. In older patients, the possible severe circulatory depression with the corresponding therapeutic consequences (including extended cardiovascular monitoring) requires special consideration.

>> Dr. Hugh had made up his mind. "*For you, the best thing to do would be spinal anesthesia. Then you may remain awake the whole time, so there is nothing to worry about,*" *he explained to Ms. Martinez.* "*Oh, doctor, you most certainly know what is best for me,*" *was her response. Now the problem was that Ms. Martinez had extreme pain in her hip with the smallest movement. Sitting for the placement of the spinal anesthesia was not an option. But Dr. Hugh had already thought of a solution.*

18.1.4 What Possibilities Does Dr. Hugh Have?

The goal of premedication is discussed in Case 14 (see Sects. 14.1.1 and 14.1.2). An analgesic component is indicated when there is pain during positioning – e.g., with nonstabilized fractures such as this one – or when the patients' condition causes enough pain to increase the sympathetic response. Apart from the systemic analgesics, Dr. Hugh also has the possibility to do peripheral regional anesthesia. A femoral block should be considered, either as a single shot or in combination with catheter placement. The catheter could later be used for postoperative pain therapy.

>> Dr. Hugh had had significant experience with the femoral block. Placement was simple, and the patients could then be positioned without pain and even sat up. "*Before you are taken into the OR, I will take care of the pain,*" *he said to Ms.*

Martinez. "I will place a small catheter in your inguinal region…" With the help of a nurse from the ER, he was able to quickly place the femoral catheter with the help of the click method. He injected 30 ml of ropivacaine 0.375 %, and Ms. Martinez's pain subsided immediately.

Dr. Hugh was in the process of documenting the unremarkable placement when Ms. Martinez called out "Joseph! Why should I take the bus?" Dr. Hugh was quite puzzled for a moment, but when he turned to the patient, she was completely normal again. Signs and symptoms of local anesthesia intoxication were not present. To be safe, he hooked Ms. Martinez up to the ECG monitor. It showed a regular sinus rhythm with a heart rate of 100 beats/min. The nurse measured blood pressure for him and it was still high, 190/100 mmHg. "That will drop when I begin the spinal and give a little sedative," thought Dr. Hugh as he pushed Ms. Martinez into the OR.

Dr. Hugh was satisfied with his performance. Ms. Martinez had very little pain during transfer to the OR bed. The short phase of disorientation did not reappear. In the OR, CRNA Pamela was waiting. She had already prepared everything for the spinal, since Dr. Hugh had given her a "heads-up" via the phone. The monitor showed an unchanged blood pressure. Ms. Martinez was sat up with the help of an OR technician, and after touching bone twice, Dr. Hugh successfully reached the subarachnoid space. Clear cerebrospinal fluid dropped from the spinal cannula, and he injected 1.8 ml bupivacaine 0.5 % hyperbaric and 10 µg fentanyl.

18.1.5 How Much Bupivacaine and Ropivacaine Did Ms. Martinez Receive?

Medication miscalculations occur again and again in daily life in the hospital, often during the calculation from percent to milliliter. The formula is simple (Eq. 18.1):

$$\% \times 10 = \text{mg/ml} \qquad (18.1)$$

This means: 1 ml bupivacaine 0.5 % equates to 5 mg, and 1 ml ropivacaine 0.375 % equals 3.75 mg. Ms. Martinez received 9 mg bupivacaine and 112.5 mg ropivacaine.

18.1.6 What Do You Think of the Intrathecal Administration of Fentanyl?

Opioids, following local anesthetics, are the most frequently used medications in the intrathecal space [8]. Of the opioids, fentanyl is the most common. Fentanyl is highly soluble in lipids and leads to segmental analgesia for up to 4 h. The analgesic potential of fentanyl is about 1:10 when compared to IV administration. As with IV administration, a feared complication is respiratory depression [7]. The effect is dependent on the dose and especially feared when given with other less lipophilic opioids.

>> "Ugh, I am suddenly very warm" said Ms. Martinez. CRNA Pamela calmed her, "That is completely normal, a sign that the spinal is working." Ms. Martinez didn't seem to understand. "I better open a window in here. Where are my house shoes?" Slowly Dr. Hugh was losing the calmness he had when he began his shift. He gave Ms. Martinez 2 mg of midazolam.

As she reached the OR, Ms. Martinez was sleeping soundly. The surgeon Dr. Leander was already waiting. Both doctors knew each other from medical school, but weren't especially fond of one another. The orthopedic surgeon thought Dr. Hugh was a show-off and a daredevil. The ever-so-understanding demeanor of Dr. Leander had always gotten on Dr. Hugh's nerves. The orthopedic surgeon was disappointed that he didn't have the opportunity to personally introduce himself to his patient. Dr. Hugh was relieved that the patient was asleep.

During positioning for the operation, Ms. Martinez woke up. She was disorientated and spoke of Christmas and plane attacks. Dr. Hugh rolled his eyes and thought to himself, "Now she's really lost it." He gave her another 1 mg midazolam. Even CRNA Pamela wondered about

the totally changed patient. She thought that a drastic drop in blood pressure was the cause, but the monitor still showed hypertension, at 175/65 mmHg.

18.1.7 What Could Be Possible Causes of Ms. Martinez's Mental Changes?

As explained in Sect. 18.1.2, it could be acute delirium. The differential includes other causes, including:

18.1.7.1 Reduced Cerebral Perfusion/ Inadequate Cerebral Oxygen Delivery

Apart from the already mentioned hypotension, increased intracranial pressure or anemia could be the trigger.

18.1.7.2 Electrolyte Imbalances or Metabolic Disorders

Electrolyte imbalances or metabolic disorders, such as hypo- or hyperglycemia, can cause decreased levels of consciousness.

18.1.7.3 Toxins

Local anesthetic intoxication is unlikely due to the timing of the events in this case.

18.1.7.4 Paradoxical Effect of Midazolam

Especially in elderly patients, benzodiazepines can have a paradoxical effect [2]. Agitation is observed instead of sedation. The paradoxical effects can be reversed by flumazenil administration. The diagnosis is confirmed if the flumazenil therapy is helpful.

>> *The surgical team had just finished the preparations.* "*Can I begin*?" *called out Dr. Leander from behind the green drapes.* "*Hours ago*," *responded Dr. Hugh. The operation began normally. Five minutes later, however, Ms. Martinez began to gag and then vomited profusely.* "*Such a mess*," *thought Dr. Hugh and asked CRNA Pamela to get it cleaned up. Most nurses liked Dr. Hugh. They liked his clear orders and big blue eyes. CRNA Pamela cleaned and dried the patient as well as she could.* "*The vase should go on the left side*," *mumbled Ms. Martinez, and then she became silent. The silence became uncomfortable for Dr. Hugh after the sudden confusion then the extreme vomiting. He went to the patient, but despite his best efforts, she could not be awakened.*

18.1.8 Which Stages of Abnormal Level of Consciousness Can You Name, and How Are They Defined?

18.1.8.1 Somnolence

Somnolence is a less serious disorder of consciousness. The patient is sleepy but can be woken up and does not usually have amnesia.

18.1.8.2 Stupor

Stupor, also termed precoma, means very deep sleep. Reactions can only be elicited by intense stimuli, such as pain. Reflexes are present, but it is not possible to wake up the patients.

18.1.8.3 Catatonia

Catatonia is rigidity of the whole body with complete consciousness. Movements are no longer or only slowly possible. Catatonia can occur with psychiatric diseases, be induced by medications, or be caused by organic brain disorders.

18.1.8.4 Coma

A coma is the most severe form of all consciousness disorders. The patient can no longer be woken up, not even by a pain stimulus. The eyes remain closed. There is no adequate reaction to bodily needs or external stimuli. In medicine, the Glasgow Coma Scale (GSC) is used to quantify such states (see Sect. 2.1.3 and Table 2.2).

18.1.9 What Should Dr. Hugh Do Now?

Ms. Martinez has a GCS of 8, at the maximum. She has vomited and can no longer be aroused. One must assume that she no longer possesses adequate

airway reflexes. Apart from a general neurological exam, securing the airway is first priority.

>> As in every regional anesthetic, everything was prepared for possible general anesthesia. CRNA Pamela held a face mask tight over Ms. Martinez's mouth and nose, and Dr. Hugh injected the meds and provided cricoid pressure for a rapid sequence induction. During all this, Dr. Hugh took a look at the patient's pupils. They were symmetrically dilated. "Dang it!" thought Dr. Hugh! Pamela placed the endotracheal tube without incident and set the ventilator, and Dr. Hugh called his attending anesthesiologist Dr. Eldridge. Upon arrival, Dr. Eldridge listened to the chain of events and inspected the pupils for himself. "You can't assess the pupils, pal. She's had cataract operations on both eyes. Have you measured the blood sugar?" he asked Dr. Hugh. "If not, then do that immediately. When the operation is over, take the patient for a CT scan. She didn't fall on her head, did she?" Dr. Hugh wasn't sure and glanced at the record from the medics. Nothing was documented.

The rest of the surgery and anesthesia went without incident. The orthopedic surgeon picked up on the problems and worked extra quickly. When it was finished, Dr. Hugh and CRNA Pamela transported Ms. Martinez to the CT suite. They had given a heads-up so that their arrival was expected.

18.1.10 Which Diagnosis Can Be Made from the CT (Fig. 18.3)?

The answer is in the legend of Fig. 18.3.

An intracerebral hemorrhage usually occurs suddenly and accounts for about 15 % of strokes. Common causes are arteriosclerosis in small vessels from long-term hypertension. Also, trauma, vessel abnormalities/malformations, and, more seldom, tumors can lead to increased intracranial pressure. A risk factor is anticoagulant therapy.

>> In the dark room beside the computer tomography, Dr. Hugh observed the CT scan. The neurosurgeon who had been called in did not see any possibility for surgical intervention. Ms. Martinez was brought into the OR again and a ventriculostomy was placed. After the procedure, Dr. Hugh transferred the patient to the ICU, intubated, ventilated, and sedated. This time he was not so satisfied with himself.

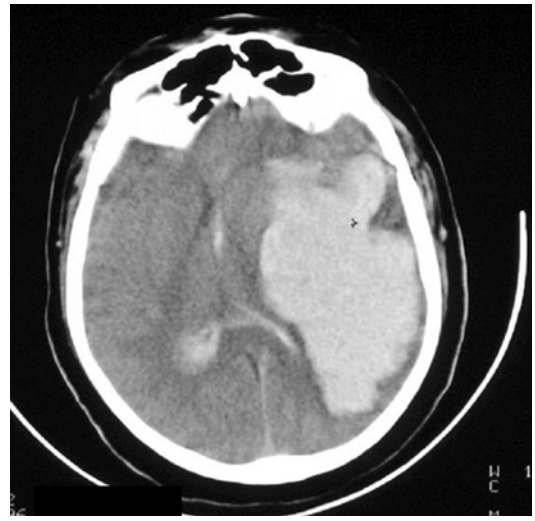

Fig. 18.3 The CT showed an intracerebral hematoma in the area of the left cerebrum. The blood obstructed the ventricles, which lead to a visible increase in intracranial pressure with a significant shift of the middle line to the right. The gyri and sulci have been flattened out

18.2 Case Analysis/Debriefing

18.2.1 What Was the Most Likely Cause for Ms. Martinez's Increased Blood Pressure?

In patients with increased intracranial pressure, there is often a reflexive increase of arterial blood pressure. This so-called Cushing reflex functions to maintain significant cerebral perfusion pressure (for the definition of CPP see Sect. 2.1.9).

18.2.2 What Are the Most Important Target Values and Actions to Take in the Acute Therapy of Patients with Raised ICP?

The Brain Trauma Foundation has established guidelines for the management of severe traumatic brain injury [5] with the goal of limiting the secondary damage (Table 18.1).

Table 18.1 Target values and treatments in the acute care of patients with increased intracranial pressure

	Target values	Measures
Respiration	S_pO_2 >90 %	Oxygen administration
	P_aO_2 >60 mmHg	Intubation and ventilation in a GCS ≤8
	P_aCO_2 35–38 mmHg	Careful P_aCO_2 monitoring
		Without ICP measurement: PEEP maximum 10–15 mmHg
Blood pressure	BPsys >90 mmHg	Invasive blood pressure monitoring
	CPP >60 mmHg	Volume therapy with isotonic crystalloid/colloid solutions
		Vasoactive substances
Cerebral pressure	ICP <20 mmHg	Semi-recumbent positioning, with sufficient blood pressure
		Temporary hyperventilation only in cases of acute neurological deterioration with danger of brain herniation
		Exceptional measures (osmotherapy, barbiturates) only if there is a possibility of measuring intracranial pressure

Modified after Brain Trauma Foundation [5]

18.2.3 Which Medical Errors Do You See in the Presented Case?

18.2.3.1 Patient History/Checkup

The history and physical performed by Dr. Hugh were very unspecific. Especially, there was no question about the accident itself. He attempted to obtain this information later in the OR after his attending asked for it. Also, he was surprised by the bilateral pupil dilation. The medication history was very superficial; when anticoagulants are taken, spinal anesthesia can be contraindicated.

Dr. Hugh noticed that the patient was hypertensive during transport to the hospital. He didn't recheck the blood pressure or begin therapy. It is possible that the intracerebral hemorrhage occurred because of the long-term hypertension.

18.2.3.2 Monitoring

The placement of the femoral block was done without monitoring. Administration of significant amounts of local anesthetics requires continual ECG monitoring, in order to recognize signs of local anesthetic overdose or accidental intravascular administration. Sometimes the abnormal values explain the symptoms, allowing a direct therapy.

In all patients with neurological disorders, blood sugar and electrolytes must be checked.

18.2.3.3 Neurological Monitoring

The repeated bouts of confusion were not taken seriously by Dr. Hugh. His only reaction was to give midazolam, which made further assessment of neurological status impossible.

18.2.4 Which Systems Failures Can You Find in the Presented Case?

18.2.4.1 Monitoring During Regional Anesthesia Placement

The standard of care is to use cardiovascular monitoring during placement of regional anesthesia with local anesthetics [1]. Failure to use cardiovascular monitoring is not only unsafe, but it is not appropriate care.

18.2.5 Dr. Hugh: Lonely Rider or Rambo VII?

Dr. Hugh, an anesthesiologist with a high level of self-confidence and an established image to protect, felt he had control of the situation. After deciding upon the diagnosis and type of anesthesia, he just wanted to prove himself. He did not take the neurological abnormalities seriously. It was not necessary to interrupt his practice of medicine to discover the possible causes. He interpreted the severe hypertension as unusual, but not as dangerous.

The feeling of having everything under control overrides thought processes which are responsible for reflection, motivation, feelings, and thoughts. A macho-man personality can endanger patients. Do you expect to now receive a tip from us about how you can deal with the macho man on your team? Apart from careful selection of team members, we can't think of anything to recommend. Creating SOPs, standards, and guidelines and conducting person-to-person discussions at regular intervals can help a little, but personalities are hard to change.

Was Dr. Hugh a Lonely Rider or a Rambo VII? He was fighting alone; the solitary ride off into the sunset fits well with his personality. Rambo VII will probably be a team player, because in Rambo V, Sylvester Stallone is already a pacifist.

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Case 19: Frozen Shoulder

19

Julia Storch, Mike Hänsel, Karen B. Domino, and Matthias Hübler

Contents

19.1 Case Introduction

>> Mrs. Baker was 86 years old. She cared for herself in her little apartment and seldom accepted assistance from her children who were living in the same city. Unfortunately, lately she had been having problems with her right arm and couldn't use it much anymore. She suffered from ankylosing spondylitis but had managed alright until now. Because her activities of daily living were so limited by shoulder pain, she made an appointment with her family physician. He diagnosed a frozen shoulder and referred her to an orthopedic surgeon who practiced at the local hospital. The orthopedic surgeon suggested, "I'll manipulate your shoulder through the full range of motion under a quick general anesthetic. Then I will decide if arthroscopy is needed. It's no big deal. I have surgical time available next week. After that you can have a few weeks of physical therapy, then you'll be like new again." Mrs. Baker was not looking forward to surgery, but she couldn't endure the pain any longer.

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Mrs. Baker listened to her children's opinions, and then she went to the preanesthesia clinic with all her medical records from her primary physician. Mrs. Baker took an antihypertensive and a tricyclic antidepressant to treat a sleeping disorder, but no other medications. Her anesthesiologist was Dr. Benjamin who worked with four other anesthesiologist colleagues in the small hospital. Together they cared for about 4,000 patients a year.

19.1.1 Which Characteristics of Ankylosing Spondylitis Should Concern an Anesthesiologist?

19.1.1.1 Skeletal System

Ankylosing spondylitis leads to inflammation of the tendon attachments – over time forming a hardening of the joint, especially the pelvis and spinal column. The resulting syndesmophytes may extend across the vertebra, resulting in stiffening of the vertebral column, forming a so-called bamboo spine.

In the thoracic area of the vertebral column, patients often have kyphosis and limited range of movement. In the cervical area, extension of the head may no longer be possible.

19.1.1.2 Pulmonary Function

As the ankylosing spondylitis progresses, pulmonary involvement may appear. Pulmonary fibrosis, interstitial lung disease, and pleural scars and effusions may form [9]. Even without direct lung involvement, the disease leads to a decrease in chest wall compliance/flexibility of the thorax. In severe cases, the patient can only breathe using the diaphragm.

In pulmonary function tests, the total lung capacity (vital capacity and forced expired volume 1-s capacity) shows a restrictive ventilation disorder [10]. The elasticity of the lungs and the diffusion capacity are normal. Therefore, patients with ankylosing spondylitis seldom suffer from shortness of breath.

19.1.1.3 Cardiopulmonary System

The markers of chronic inflammation – especially tumor necrosis factor (TNF) and C-reactive protein – are a sign of a systemic disease which can also affect the cardiopulmonary system [3]. The typical pathology includes aortitis and consequential aortic regurgitation, impairments of the intracardiac signal transduction – especially at the AV node – and myocarditis of the left ventricle [6].

19.1.2 Which Anesthesia Techniques Would You Suggest for Mrs. Baker?

19.1.2.1 Intubation

Due to her preexisting conditions, Mrs. Baker is classified as ASA class III. Due to limited neck mobility, a difficult intubation is to be expected. Fiberoptic intubation is indicated in this case, and it is easiest and safest to perform while she is awake.

19.1.2.2 Anesthesia

In addition, regional anesthesia to aid post-op physical therapy is recommended. An indwelling interscalene catheter lends good analgesia in the shoulder and upper arm area.

Basically, it is possible to do a shoulder arthroscopy under regional anesthesia only. The interscalene block must be extended to include a block of the suprascapular and supraclavicular nerves. A requirement is excellent teamwork between the surgeons and anesthesiologists. The patients must also be cooperative, since the head is fixed during the procedure in most hospitals. Should the necessity arise to secure the airway, access is severely limited during the surgery.

>> For Mrs. Baker, everything sounded so difficult and stressful – everything was a "big deal." However, the orthopedic surgeon had her full trust, and several friends had said that it was a good hospital. Her mind was made up. She would go through with the surgery, but not with regional anesthesia alone, and she made this clear to her anesthesiologist. Dr. Benjamin was becoming fond of the lively old lady. He glanced at the ECG, which was unremarkable except for signs of left ventricular hypertrophy. Then he said goodbye to Mrs. Baker and told her not to worry about a thing.

Although Mrs. Baker had taken her medication exactly as ordered, on the morning of the surgery, she presented with a blood pressure of 195/100 mmHg in the preanesthesia area. She hadn't become drowsy from the preoperative sedative. She was relieved as Dr. Benjamin entered the preanesthesia area and greeted her. Then, he placed an interscalene catheter. CRNA Alice assisted Dr. Benjamin and spoke calmly to Mrs. Baker who felt uncomfortable the whole time, although she had anticipated something much worse. Then there was this tube which was supposed to be pushed up her nose…

19.1.3 What Do You Know About Indications for a Fiberoptic Intubation?

Fiberoptic intubation can be performed nasally or orally in conscious or unconscious patients. For fiberoptic intubation in emergency settings, essential prerequisites are good logistics, equipment which can be quickly available for use, and experienced personnel. From newborns to adults, this technique can be safely used in any difficult intubation. Awake fiberoptic intubation is useful for all sorts of difficult intubations, including facial malformations, C-spine and facial injuries, upper airway masses, limited movement of the jaw or C-spine, and in patients with an elevated risk of aspiration. Fiberoptic intubation also plays an important role in patients with extreme obesity and is highly valuable in dental cases.

19.1.4 How Is Awake Fiberoptic Intubation Performed?

Awake fiberoptic intubation requires cooperative patients. Complete informed consent must be obtained, with full disclosure of all details, and the patient's fears must be addressed. The patients should receive a sedative before transport to the OR and further sedation in the OR. The combination of benzodiazepine – usually midazolam – with an opioid works well. Basically, any opioid can be used. Longer-acting opioids, such as fentanyl or sufentanil, are often used but must be carefully titrated. Some anesthesiologists prefer a controlled infusion of remifentanil. The goal is a cooperative sedated patient, and if necessary both groups of drugs can be antagonized. In some patients, such as those who become agitated with midazolam, low doses of droperidol (1.25–2.5 mg) or scopolamine (0.2–0.4 mg) may need to be added. Some anesthesiologists may alternately administer dexmedetomidine as the agent for sedation; however, the drug is not immediately reversible and some (mostly younger) patients experience dysphoric reactions. Mild hypoventilation from sedation is corrected with oxygen administration through a nasal cannula or simple face mask.

Fiberoptic intubation in conscious patients offers maximal safety, because spontaneous breathing and airway reflexes are present. Care must be given that the topical anesthetic is sufficient; if not, extreme stress reactions and laryngospasm can occur.

After the desired sedation has been achieved, for nasal intubation, a vasoconstrictor (e.g., Afrin nose drops) should be applied in each nasal passage to reduce the risk of bleeding. The application of cocaine, which is simultaneously a topical anesthetic and a vasoconstrictor, has largely been discontinued due to concerns over drug abuse.

Spray, drops, or vaporization of topical anesthetics, such as lidocaine or Cetacaine, can numb the mucus membranes in the nose and throat region. A transtracheal injection of 4 % lidocaine may also be performed in select patients. Commercial sprays have the disadvantage of being made with alcohol; therefore, nasal application is painful. The maximum limit for the various topical anesthetics should be observed. It is also good to place a soft nasopharyngeal airway (lubricated with lidocaine jelly) first to dilate the nasopharyngeal passage, then remove it, and insert the fiberoptic scope.

The bronchoscope with a lubricated, threaded endotracheal tube is placed, with full visual control, through the nostril into the oropharynx. The vocal cords are visualized and topical anesthesia is sprayed from the additional lumen of the bronchoscope. After 1 min, the vocal cords are numb and relaxed in the open position. The bronchoscope then passes through the cords

and local anesthetic is applied into the trachea. Due to the common occurrence of a cough, it is recommended to pull the fiberoptic scope back to remain cranial to the larynx. If detection of the trachea is difficult, the fiberoptic can also be left in the trachea. Now the optic from the trachea will be projected further to the bifurcation.

The bifurcation should remain in sight in order to check placement, as the tube – easiest with the help of a second person – is slowly pushed further, slowly, with a gentle rotation, until it appears in the visual field of the fiberoptic. The bronchoscope is then removed and general anesthesia induced after verification of the lack of leak.

An awake oral intubation is technically more difficult, but the procedure is similar. In some cases, superior laryngeal nerve blocks may need to be performed in addition to topical spray, nebulized, or gargled lidocaine. In order to protect the fiberoptic scope, a bite block should be used. The presentation of the larynx is made easier by a slit oropharyngeal airway, such as an Ovassapian airway. If an unconscious patient needs to be fiberoptically intubated, ventilation during the procedure can be done via an adapter.

>> Dr. Benjamin was satisfied that the placement of the interscalene catheter went without problems. In the OR, Mrs. Baker had received so much midazolam and fentanyl by now that she was very sleepy but maintained good respiration. Dr. Benjamin noticed that CRNA Alice was very nervous and constantly checked the monitor. "Everything OK?" he asked. "Yes," she replied, "it's just that I haven't done a fiberoptic intubation in so long." "There's nothing to worry about," replied Dr. Benjamin, "I'll talk you through every step."

Dr. Benjamin removed the nasopharyngeal airway from the right nostril and inserted the bronchoscope. The laryngeal inlet was easily visualized, and after application of local anesthetic, the fiberoptic was inserted into the trachea until he could see the bifurcation. "You've done well, Mrs. Baker. You can go to sleep now." Then Dr. Benjamin said to CRNA Alice, "Please give 100 mg propofol, and 200 µg fentanyl." Mrs. Baker fell sound asleep and ceased breathing spontaneously. "Now slide the tube, Alice. I will hold the bronchoscope in position." The tube slid off without difficulty. After 8 cm, however, Alice felt resistance. "It won't go any further," she said to Dr. Benjamin. "Let me try," he replied. "Please hold the bronchoscope." Exactly as reported, the tube would not budge despite Dr. Benjamin's attempts to turn and push. He suddenly heard a loud crack during his attempts. The monitor's alarm sounded: oxygen saturation was only 85%!

19.1.5 What Should Dr. Benjamin Do Now?

The most important goal is securing oxygenation because Mrs. Baker is no longer breathing, and since ventilation over the fiberoptic scope is not possible, it must be removed.

>> Dr. Benjamin removed the bronchoscope and the tube and obtained a face mask from CRNA Alice. After giving a few ventilations, blood appeared under the mask. He lifted it up and saw that bright red blood was bubbling out of the right nostril. Now he got nervous. The beeping tone of the monitor continued to get deeper; he looked up to see "S_PO_2 75%" on the screen.

19.1.6 What Would You Do in This Situation?

The most important goal is still oxygenation. Obviously, the tube has caused a serious nosebleed. Nasal ventilation should still be possible, but there is the danger of blood aspiration. It is better, therefore, to ventilate through the mouth or through a laryngeal mask airway. Until the airway is secured, a suction catheter should be placed in the left nostril to prevent blood from draining into the lungs during mask ventilation.

Furthermore, it is obvious that more assistants must be recruited to gain control of the situation.

>> Dr. Benjamin had really become nervous now. He had imagined the chain of events a little differently. "Quick – give me the laryngoscope!" Carefully, but quickly, he slid it down the back of Mrs. Baker's mouth. The mouth could be opened

wide, but no extension was possible. "*This is not going to work! I can't see anything!*" *The S_pO_2 had fallen to 60%. Dr. Benjamin laid the laryngoscope to the side and frantically ventilated with the mask, while CRNA Alice attempted to suction blood with a catheter.* "*This is not going to work,*" *said Dr. Benjamin out loud. The S_pO_2 had increased to only 65%.* "*Alice – do you have any idea what we can do now?*" *Without saying a word, Alice took a laryngeal mask airway out of the drawer. Dr. Benjamin accepted it and placed the laryngeal airway without a problem. As he began to effortlessly ventilate Mrs. Baker through the mask, the door of the OR opened and in walked a fellow anesthesiologist, Dr. Salvador.* "*Do you need any help? The alarm has been constantly going off in here.*"

19.1.7 What Are the Options Now?

With the laryngeal mask airway, at least the upper airways are protected and the danger of aspiration of blood is reduced. Basically, several options can be considered now.

19.1.7.1 Continuing the Anesthesia with the Laryngeal Mask Airway

Shoulder arthroscopy can be done under general anesthesia with a laryngeal mask airway. A problem, however, is that an urgent intraoperative intubation is not possible, or only with extreme difficulty (see Sect. 19.1.2). In this case, there is danger of aspiration of blood if the laryngeal mask airway becomes displaced during the surgery. Therefore, continuation of anesthesia with only a laryngeal airway is not recommended.

19.1.7.2 Intubation via the Laryngeal Mask Airway

Intubation through a laryngeal mask airway is possible, either blind or with fiberoptic visualization. Disadvantages are:

- Only a small tube can be used.
- Sliding the tube through the laryngeal mask airway may be difficult.
- A few laryngeal mask airways have a filter build in, which makes passage of the tube difficult. Only experienced practitioners should explore this option.

19.1.7.3 Use of an Intubating Laryngeal Mask Airway

Alternatively, an intubating laryngeal mask airway could be exchanged for the current one, so that intubation can be done blindly or with fiberoptic assistance. Again, only experienced practitioners should explore this option.

19.1.7.4 Nasal Tamponade and Fiberoptic Oral Intubation

With sufficient nasal tamponade – perhaps done by a colleague in the ENT – fiberoptic intubation can be done orally. Special masks are recommended, which allow simultaneous ventilation (Patil–Syracuse face mask or a Mainz universal adapter). Assistance from a second assistant is recommended.

19.1.7.5 Allow the Patient to Wake Up Again

This option should always be considered when experiencing intubation difficulties. After airway reflexes have returned, and the epistaxis has stopped, a new fiberoptic intubation attempt can be made with spontaneous ventilation.

>> *As Dr. Benjamin reported the problems to his colleague, Mrs. Baker's saturation increased to 95%.* "*Now we can do it as I learned it,*" *said Dr. Salvador with confidence and determination in his voice. He was given a 5.0 tube and placed it without incident into the bleeding nostril of Mrs. Baker. Carefully, he inserted until the end came out behind the internal nasal opening, and blocked the cuff, and pulled the tube back.* "*At least now the blood will not flow down the throat,*" *explained Dr. Salvador. Then he led a second 6.5 tube into the other nostril until he had passed the internal nasal opening. The laryngeal mask airway was displaced a little, but the ventilation of Mrs. Baker was unaffected.* "*You didn't have any difficulty finding the laryngeal inlet, did you?*" *he asked Dr. Benjamin, who shook his head. Dr. Salvador requested the fiberoptic scope. While he inserted this into the larger tube, CRNA Alice injected another 100 mg*

propofol in order to maintain the depth of anesthesia.

Dr. Benjamin removed the laryngeal mask airway, dislocated Mrs. Baker's mandibular joint, and pulled, with the help of a compress, her tongue out of her mouth. "*Very good*," *said Dr. Salvador,* "*now I have good visibility*." *The vocal cords were wide open, and Dr. Salvador could slide the fiberoptic until he saw the carina. This time, the tube slid off easily. Dr. Salvador checked placement of the tube by slowly pulling out the fiberoptic scope. The airway was secure.* "*It is best to leave the second tube in place after the operation*," *said Dr. Salvador before he left.*

After the successful intubation, Mrs. Baker was positioned for the surgery. General anesthesia was maintained with desflurane and fentanyl. As Mrs. Baker was sat up in the beach chair position, her blood pressure dropped to 80/45 mmHg and her heart rate decreased to 42 beats/min. Dr. Benjamin gave phenylephrine, 500 ml of a crystalloid infusion, and 0.5 mg atropine. The desired effect appeared quickly. The arthroscopy went well.

19.1.8 What Should One Watch Carefully for During Emergence?

In the presented case, there is the danger of blood aspiration. The danger is significantly increased, as compared to a routine intubation, because the pharynx, larynx, and trachea were treated with a local anesthetic. Therefore, diminished airway protective reflexes are to be expected. The patient should have a sufficient level of consciousness and be able to cough upon request and spit blood, or extubation should be postponed.

>> Dr. Benjamin first removed the small tube. When he was sure that the bleeding had ceased, he discontinued the desflurane. The emergence from anesthesia went without complications. Mrs. Baker was still sleepy, but she promptly followed all commands.

After an hour, the call came from the PACU. Mrs. Baker had become difficult and was constantly arguing with an imaginary farmer. Guard rails had been put on her bed. Apart from that, she had a heart rate of 120 beats/min. Dr. Benjamin had feared exactly this problem.

Table 19.1 Medications and substances which can trigger postoperative delirium

Medication class	Agent
Analgesics	Codeine, morphine
Antibiotics/antiviral drugs/ antimycotics	Acyclovir, amphotericin B, cephalosporin, ciprofloxacin, imipenem, metronidazole, rifampicin, penicillin
Anticonvulsants	Phenytoin, phenobarbital
Cardiovascular active medications	Clonidine, captopril, digoxin, nifedipine, propranolol
Drugs	Alcohol, amphetamines, cannabis, cocaine, etc.
Corticosteroid	Dexamethasone, methylprednisolone
Others	Ketamine, metoclopramide, theophylline, atropine, scopolamine, benzodiazepines, propofol, volatile anesthetics, H_2 receptor blocker

According to Cavaliere et al. [1]

19.1.9 What Must Be Done Now?

The various forms of postoperative cerebral dysfunction were discussed in Case 18 (see Sect. 18.1.2). Mrs. Baker has an acute delirium, which has an incidence of about 11 % in elderly patients following elective surgery [8]. However, before a psychiatric diagnosis is given, other factors which could cause agitation and hallucination should be ruled out, including the following [11]:

19.1.9.1 Risk Factors for Agitation and Hallucination

- Disorders of vertebral oxygen perfusion, e.g., low cardiac output or anemia or hypoxia
- Electrolyte changes such as hypo- or hypernatremia
- Disorders of the acid base balance

- Metabolic disorders such as hypo- or hyperglycemia
- Hypothermia
- Extended effects of anesthetic agents
- Side effects of medications (Table 19.1, according to [1])
- Pain
- Full bladder

Furthermore, the tachycardia must be evaluated – is it due to the psychological excitation or is there another cause?

- *The PACU nurse returned with the results of the arterial blood gas, and none of the values were outside of the normal range. Mrs. Baker's body temperature was 36.8 °C, also normal. The blood pressure was 190/95 mmHg, and hypovolemia was not evident.*

19.1.10 What Differential Diagnoses Are You Considering?

The homeostasis values are unremarkable; central neurological causes must be considered. Even though there is no suspicion of a cerebral perfusion disorder, hypotensive or hypoxic phases during the anesthesia – especially caused by the abrupt alleviation of long-standing hypertension – could explain the confusion.

Clinical experience shows, however, that there isn't always a clear, direct correlation between postoperative confusion and hypotension and hypoxemia during general or regional anesthesia techniques. A possible explanation is that pulse oximetry and blood pressure are very unreliable measurements of brain perfusion. In order to detect perioperative cerebral ischemia, the markers for neuronal damage, e.g., the neuron-specific enolase of the calcium binding Protein S100B, must be measured [5]. Both markers, however, have the disadvantage that false-positive elevations lead to incorrect diagnosis. They are therefore only recommended in organic brain disorders which result from traumatic brain injury, stroke, or subarachnoid hemorrhage.

Apart from the organic changes, residual anesthetic agents or known paradoxical medication effects (such as that of benzodiazepine in elderly patients) must be taken into consideration.

Lastly, a central anticholinergic syndrome (CAS) could explain the signs and symptoms.

>> *Mrs. Baker was now beet red in the face, felt hot, and was flailing about wildly in bed. The nurses made every attempt to calm her and explain where she was, but it was useless. Mrs. Baker believed that she was on a farm and she was shooing away all the nurses who had nothing to do with her farm.*

Dr. Benjamin was impressed with the nurses' determination to calm the patient, but the nurses gave him accusing looks. He must find help.

19.1.11 What Would You Do Next?

The findings can't be explained by any other cause, so CAS is the most likely diagnosis.

The pathophysiological cause of CAS is a functional block of central and peripheral muscarinic choline receptors or deficiency of acetylcholine in synaptic space [4]. The functional block can occur directly (e.g., by belladonna alkaloids, antipsychotics, antidepressants, antihistamines, Parkinson's medications) or indirectly (through opioids, IV and volatile anesthetics, local anesthetics, benzodiazepines, H_2 receptor blockers).

Trigger substances are lipophilic and penetrate the blood–brain barrier. The diagnosis is difficult, because the clinical picture is complicated and the clinical presentation can vary. The fearful, agitated type symptoms with hallucinations – sometimes accompanied by myoclonus and seizures – is differentiated from the type predominated by a decrease in the level of consciousness – and possible coma and apnea. Following anesthesia, the prolonged somnolence is more common than the agitated form [7]. The symptoms of CAS are divided into central and peripheral (Table 19.2). When CAS is suspected, at least one central and two peripheral symptoms must be present for diagnosis. If the CAS suspicion arises after an anesthesia during which muscle relaxants were antagonized with peripheral cholinesterase inhibitors, the peripheral symptoms may be absent.

Table 19.2 Central and peripheral symptoms of central anticholinergic syndrome (CAS)

Central symptoms	Peripheral symptoms
Fear	Tachycardia, arrhythmia
Uneasiness	Mydriasis
Disorientation	Warm, red, dry, skin
Hyperactivity	Urinary retention
Excitation	Dry mouth
Reduction in level of consciousness (LOC)	Reduced peristalsis
Dizziness	Difficulty speaking
Coma	Hyperthermia
Ataxia	
Seizures	
Myoclonus	
Respiratory depression	
Nystagmus	
Shivering	
Hyperpyrexia	
Hallucination	

For the diagnosis of CAS, ≥1 central and ≥2 peripheral symptoms are required. Peripheral symptoms can be absent after use of peripheral cholinesterase inhibitors for reversal of muscle relaxants

Mrs. Baker had uneasiness, disorientation, and hallucinations as central symptoms. Peripherally, she had tachycardia, and warmth and redness of the skin.

The suspected diagnosis of CAS is tested by a dose of physostigmine, a central cholinesterase inhibitor. The dose is 0.04 mg/kg body weight slow IV push. After a maximum of 20 min, improvement in the symptoms should be seen. Due to the short duration of action of physostigmine (25–40 min), a second dose may be given if the symptoms reappear. An infusion pump can be set up at the rate of 1–2 mg/h. Due to possible side effects such as bradycardia, bronchospasm, cerebral seizures, and severe nausea, physostigmine should only be given under cardiovascular monitoring.

19.1.12 Mrs. Baker Received Atropine for Her Bradycardia. Was There an Alternative?

The different anticholinergics are presented in Case 28 (see Sect. 28.1.5). Atropine and scopolamine belong to the major cause of CAS, which is also called "atropine intoxication." Atropine causes the agitated type symptoms and scopolamine causes the depressive type. For this reason, glycopyrrolate is recommended for bradycardia and hypersalivation in elderly patients. Glycopyrrolate does not cross the blood–brain barrier. However, there are reports of CAS after glycopyrrolate [2].

>> The surgeon, who had gotten wind of elderly, but independent Mrs. Baker's personality changes, came to the PACU. From the look on his face and in light of the show that Mrs. Baker was now putting on, he was clearly rethinking the indications for her surgery. He walked up to Dr. Benjamin and asked, "She'll be OK again, won't she?"

"We'll see," said Dr. Benjamin as he began to inject the physostigmine. After a few minutes, Mrs. Baker calmed down. The tachycardia disappeared, and she returned to her normal self again.

19.2 Case Analysis/Debriefing

19.2.1 What Medical Errors Do You See in the Presented Case?

19.2.1.1 Patient History and Exam

As explained in Sect. 19.1.1, ankylosing spondylitis can affect the cardiovascular system. Dr. Benjamin neglected to auscultate Mrs. Baker's heart.

19.2.1.2 Awake Fiberoptic Intubation

Mrs. Baker was given general anesthesia before the endotracheal tube was secured in the trachea (see Sect. 19.1.4). Many anesthesiologists prefer blind nasal insertion of an endotracheal tube into the pharynx. This technique reduces undesired surprises such as the impossibility to get the tube through the nose. However, it may be associated with a nosebleed and obscured visualization of the larynx. General anesthesia should not have been induced until the tube was successfully passed and ventilation verified.

19.2.1.3 Procedures After Placing the Laryngeal Mask Airway

Even under circumstances such as the one presented, when no harm was done to the patient, the safest variation would have been to let her wake up and then reintubate her with spontaneous ventilation.

19.2.1.4 Atropine Administration

Mrs. Baker was taking a tricyclic antidepressant prescribed by her general practitioner. Due to this, and due to her age, intraoperative administration of atropine, although not incorrect, should be viewed critically.

19.2.2 Which Systems Failures Can You Find in the Presented Case?

19.2.2.1 Management of a Difficult Airway

After the first fiberoptic intubation was unsuccessful, Dr. Benjamin had no Plan B. As presented in Case 1 (see Sect. 1.1.6), it is sensible to establish algorithms and practice in advance, in order to properly react when such a situation arises.

Additionally, CRNA Alice had announced that she felt very unsecure. It is not a good idea to start teaching in a real-life situation. Instead, mandatory training in a simulation lab is advised.

19.2.3 What Was Dr. Benjamin's Solution Strategy When the Nosebleed Arose?

He had none. In Sect. 19.1.4, the safest procedure in awake fiberoptic intubation is detailed: general anesthesia is induced after the tube is safely in the trachea. In this case, the procedure was altered: Mrs. Baker was given the general anesthesia too soon, which resulted in a very tricky situation.

CRNA Alice admitted that she was not feeling competent with the procedure of a fiberoptic awake intubation. As we later noticed, neither was Dr. Benjamin. Instead of saying, "I'll talk you through every step," he should have immediately given a detailed list of the procedures and later given the instructions for individual steps.

Why was Dr. Benjamin overwhelmed with the situation? He had not prepared himself in advance for the possible problems; he had just gone into the day with full optimism. When he was later under pressure, he did not develop the necessary overview, and he reacted without a plan. The various problems he encountered were only resolved because he had help from the team, from CRNA Alice, and from his colleague, Dr. Salvador, who heard the alarm going off constantly and came in to investigate and to help.

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Case 20: Forearm Fracture

20

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20.1 Case Introduction

>> Anesthesiology resident Dr. Blue had been in anesthesia for about a year. So far, she had rotated on general surgery and cared for some urology patients. Now she was excited to begin trauma surgery. There were many regional anesthesiology procedures done here, and the hands-on techniques were her favorite. Most of the nerve blocks in her department were done under ultrasound guidance. "The days of blind flight into the plexus are over" was one of the favorite sayings of her favorite attending, Dr. Eldridge.

As Dr. Blue returned from her lunch break, she was surprised to see she was assigned an unexpected patient, who was waiting in the preop holding area. Anesthesia technician Maria vaguely remembered a forearm fracture being mentioned, which was supposed to be on today's

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OR schedule. Dr. Blue wondered why no one had informed her, but personally she was happy to finally have a chance to do a regional anesthesia technique. After further questioning, Dr. Blue found out from the orthopedic surgeons currently operating that 37-year-old Mr. Graham had an open distal forearm fracture from a bicycle accident. He needed an open reduction, and the expected OR time was 1.5 h.

Dr. Blue charted the following information on the preoperative assessment after performing a history/physical and obtaining informed consent from Mr. Graham:

- *175 cm tall, 70 kg*
- *Allopurinol therapy for asymptomatic hyperuricemia*
- *Latex allergy*
- *Smoker with 20 pack years, chronic bronchitis*
- *Breakfast 2 h before the accident*

20.1.1 Evaluate the Patient's Comorbidities

20.1.1.1 Hyperuricemia

Gout is one of the purine metabolism disorders with pathological accumulation of urates in organs. In most cases, there is an asymptomatic hyperuricemia due to multifactorial-inherited reduction of the tubular uric acid excretion. The clinical manifestation (gouty arthritis) is triggered by purine-rich foods, alcohol, and fasting. Chronic recurrent arthralgia, joint disorders, limited function, gout tophi, nephrolithiasis, and urate nephropathy with progressing renal failure can occur [7].

20.1.1.2 Latex Allergy

Latex is part of our daily life due to its excellent elasticity, flexibility, stability, ruggedness, and sealing properties.

The milky colloid is produced by the bark of the African Malaysian caoutchouc tree, Hevea brasiliensis, also called rubber (natural latex) tree. The pure latex is 99 % *cis*-1,4 polyisoprene, a protein-rich mass which is treated with preservatives (ammonia, formaldehyde) to create a stable, liquid, natural rubber which is sometimes called raw latex. The further processing is done either to soften or to dry form-pressed latex products. Gloves, balloons, condoms, pacifiers, and catheter tourniquets are made of soft latex and contain 1 % of the original protein. The hard form-pressed vulcanized latex products such as tires, shoe soles, rubber stamps, and ampule sealing caps contain less than 1 % protein, due to the denaturing process.

Clinically, one differentiates between:

- Latex hypersensitivity
- Latex allergy

Latex hypersensitivity is present in 1–2 % of the population. It is asymptomatic but evident in in vivo or in vitro tests. A latex allergy, on the other hand, has clinical symptoms. It is caused from an IgE-mediated hypersensitivity type 1 (immediate hypersensitivity) against the Hev b 1–13 allergens (see Sect. 14.2.2). Until 1980, contact dermatitis was quite common. It was a delayed type IV reaction caused by chemicals such as preservatives, softeners, antioxidants, or powders.

Patients with atopy have a four times higher risk of latex allergy. Epidemiologically important groups are children with spina bifida or meningomyelocele. One to two percent of health-care professionals have a manifested latex allergy (Hev b 2, 5, 6, 7, 13), and 40 % are hypersensitive to latex allergens. In children with spina bifida or meningomyelocele, the incidence of latex allergy is 20–65 % (Hev b 1, 3). The exposition can occur transcutaneously, inhalative, parenteral, or over mucosal membranes. The clinical severity of the reaction can be dermatitis, angioedema, conjunctivitis, asthma, or anaphylaxis. More than half of all patients have an additional food allergy to bananas, kiwis, avocados, or chestnuts. Causally, crossover reactions have been discovered to these as well as other foods, such as papaya.

20.1.1.3 Note

- A latex allergy cannot be cured. The only treatment is avoidance of contact and reduction of exposure.
- In the medical setting, the use of latex-free equipment is required in patients with a history of latex allergy.

- If at all possible, patients with severe latex allergy should receive the first slot in the OR schedule, in order to reduce the exposition to aerosols which contain latex [4].
- More information can be found at www.latex-allergyresources.org.

20.1.1.4 Smoker

Pack years is a measurement of the lifetime dose of nicotine from cigarettes. The risk of bronchial carcinoma is 11 times higher after smoking >40 pack years, as compared to a nonsmoker. Additionally, smoking causes a chronic inflammation, inactivates protective proteolytic enzymes and repair mechanisms, and is the main risk factor for chronic bronchitis and COPD. After age 30, there is a yearly reduction in expiratory 1 s capacity (FEV_1). In smokers, the FEV_1 decrease is dependent on the pack years, and is 40–200 ml per year more than in nonsmokers (25–30 ml/year) [8].

20.1.2 Evaluate the Patient's Fasting State

Apart from the time point of the last oral intake or the last cigarette, the time point of the accident must be known. After a traumatic event, sympathetic stimulation slows gastric emptying.

20.1.2.1 Note

Following trauma, the amount of time needed for a patient to be considered "fasting" is unknown. More important than the time between trauma and surgery is the time between last oral intake and the trauma.

This period of time should be used for determining the Nothing by Mouth (NPO) time. In emergencies – such as an open fracture – a fasting state has less importance. The urgent nature of surgery does not allow for a delay, as it would impair patient outcome.

>> Dr. Blue found out that the accident happened about an hour ago. Mr. Graham was wearing his bike helmet and described the full sequence of events. He was caught off guard by a car opening its door, and he couldn't swerve away fast enough.

20.1.3 What Type of Anesthesia Will You Use?

The choice of anesthesia varies with the patient condition and the expected duration of surgery. For this procedure, regional anesthesia is a reasonable choice, provided the patient consents to a block. Mr. Graham had breakfast 2 h before the accident, and thus, he had a full stomach and an increased risk of aspiration of gastric contents. Therefore, regional anesthesia, perhaps with catheter placement, such as an axillary plexus block, with mild sedation, is a good anesthetic choice. In complex fractures with long surgery times or with uncooperative patients, general anesthesia is necessary, using a rapid sequence induction with cricoid pressure to reduce risk of aspiration.

>> Dr. Blue was not sure which equipment was latex-free, but anesthesia tech Maria calmed her with the words: "We will exchange the latex gloves for the vinyl gloves; the rest of our equipment is free of latex." Since the second anesthesiologist was caring for a patient still in the OR, Dr. Blue began preparations for the axillary block in the preop holding area.

20.1.4 A Totally Latex-Free Environment Is Not Possible in the Medical Environment. Which Other Hidden Latex Products Must You Watch Out For?

Hidden latex products include injection and infusion elements, various dressings, adaptors, blood pressure cuffs, tourniquets, syringes, nasopharyngeal airways, bite blocks, and ventilation bags on anesthesia machines. Latex particles on the seals of infusion bags, bottles, and medication ampules can dislodge and be released into the solution during penetration or through direct contact during positioning [3].

A separate case or trolley with only latex-free materials should be available.

>> As Dr. Blue shaved the patient's armpit, anesthesia tech Maria asked which local she should

prepare. Dr. Blue hadn't been in trauma surgery very much and she didn't know. "What do we normally use?" she asked. "In short procedures, prilocaine, in longer procedure, ropivacaine" was anesthesia tech Maria's answer. With a predicted surgical duration of 60–90 min, Dr. Blue decided upon prilocaine 1%.

After 30 min, Dr. Blue began identification of the ulnar, median, and radial nerves with the help of an ultrasound and nerve stimulator. After negative aspiration, 12 ml of local anesthetic was injected into every nerve. As Dr. Blue attempted to identify the musculocutaneous nerve, Mr. Graham complained about pain in his shoulder and wiggled around on the litter. Dr. Blue asked anesthesia tech Maria to inject 2 mg midazolam. Then the orthopedic surgeon entered the preop holding area and tapped on an imaginary watch on his wrist. Fifteen minutes later, the musculocutaneous nerve was also blocked with 10 ml prilocaine 1%. Dr. Eldridge, the attending anesthesiologist and anesthesiologist-in-charge, came to the preop holding area and that Dr. Blue hadn't called him earlier to talk about the case and was surprised that the block was already performed. He then was called away to deal with the OR schedule.

20.1.5 What Dosage Limits Do You Know for Prilocaine or Other Local Anesthetics?

Local anesthetics should be individually dosed for each patient using the smallest possible dose which delivers a sufficient effect. Furthermore, the various absorption rates depending on location of injection should be considered, along with individual factors such as hypoalbuminemia. The doses listed in Table 20.1 are given as guidance.

By using ultrasound guidance during the placement of the block, a smaller volume of local anesthetic can be used to obtain the same effect, which results in improved patient safety [5]. Prilocaine is an aminoamide and is metabolized hepatically and extrahepatically.

Table 20.1 Maximum doses of local anesthetics

Local anesthetic	Without epinephrine	With epinephrine (1:200,000)
Lidocaine	3–4 mg/kg (300 mg)	7 mg/kg (500 mg)
Mepivacaine	4 mg/kg (300 mg)	7 mg/kg (500 mg)
Prilocaine	5–6 mg/kg (400 mg)	8–9 mg/kg (600 mg)
Articaine	5–6 mg/kg (400 mg)	
Ropivacaine	3–4 mg/kg (250 mg)/to 37.5 mg/h continually	
Bupivacaine	2 mg/kg (150 mg)/ up to 0.4 mg/kg/h continually	2–3 mg/kg (150–225 mg)

>> Just as the last milliliter of local anesthetic was injected, Mr. Graham was pushed into the OR. Dr. Blue quickly injected 2 mg midazolam. Whether or not the block had fully taken effect could not be checked, but Dr. Blue was quite confident in her success. The inflatable cuff for the tourniquet was wrapped in cotton, because no one was sure whether or not it contained latex. Mr. Graham slept, but as the surgeons made their first incision, he jolted. "Look, at least make sure the patient stays still and doesn't feel any pain, OK?" called the surgeon.

20.1.6 What Options Do You Have Now?

Mr. Graham could possibly have moved by chance, just at the same moment of the incision, but a check of the block needs to be performed immediately. If it is insufficient, then the surgeons can try to supplement it with more local anesthesia, being careful not to exceed the maximum dose. Apart from the additional local, it is also possible to administer systemic pain relief, for example:

- Bolus administration of fentanyl
- Continuous remifentanil infusion
- Bolus administration of ketamine (in combination with a benzodiazepine)

One should not attempt to rescue an inadequate nerve block at any cost. Such action is unethical and traumatic for the patient. General

anesthesia should be immediately induced. If the patient moved only by chance, then the sedation can be increased, such as by:

- Bolus of midazolam
- Continuous low-dose propofol infusion

>> Dr. Blue gave 3 mg of midazolam IV, and anesthesia tech Maria brought knee rolls, but neither helped for long. Dr. Blue was annoyed that she had actually believed the procedure would only take 1.5 hr. She was now informed that the surgery would take at least another hour. A continuous infusion of propofol 50 µg/kg/min was started. Mr. Graham slept so deeply that Dr. Blue had to hold up his jaw with one hand, while she filled out the anesthetic record with the other. Anesthesia tech Maria placed an oxygen face mask on Mr. Graham, with 4 l O_2/min. With these measures, the pulse oximetry showed (S_PO_2) 93 %.

20.1.7 Which Inspiratory Oxygen Concentration (F_iO_2) Can Be Reached with a Face Mask?

Through the basic nasal cannula system, a maximum F_iO_2 of 30–40 % can be reached (see Table 15.1). With a simple oxygen mask without a reservoir, the maximal F_iO_2 value can be 50 %. In order to prevent inhalation of exhaled air, a fresh gas flow of >6 l/min needs to be set. With an oxygen mask with a reservoir bag and unidirectional valve, F_iO_2 values of 85 % can be obtained, if the fresh oxygen flow is at 10–15 l/min.

>> With the propofol infusion, Mr. Graham was now very still and tolerated the surgery well. Every now and then, he took an extra deep breath. The hemodynamic parameters were stable, but the S_PO_2 value dropped to 88 % despite the increasing O_2 flow – now at 10 l/min.

20.1.8 What Noninvasive Techniques/Physical Checks Should You Try Now?

The decrease in saturation could have a number of causes. The following points must be checked:

- Fresh gas flow to the mask and correct connections.
- Technical defects of the pulse oximeter should be ruled out.
- An arterial perfusion disorder in the location of S_pO_2 measurement must be ruled out.
- Hypothermia should be ruled out.
- Movement artifacts should be ruled out.
- Sleep apnea with undulating levels of saturation due to partial obstruction of the upper airways, aggravated by propofol administration.
- Auscultation and inspection of the lungs to rule out:
  - Pneumothorax, status post-accident
  - Aspiration of gastric contents
  - Exacerbation of chronic bronchitis
  - Bronchospasm
- If at hand, reevaluate chest X-ray. If not at hand, consider ordering one
- Check hemodynamics.

>> After checking possible causes of the decrease in S_PO_2, Dr. Blue was no smarter than when she began. Everything seemed normal. She reduced the propofol in the meantime to 25 µg/kg/min, and she woke the patient many times to ask him to breathe deeply and cough. Dr. Blue had hoped that there would be an error on the pulse oximeter reading itself, but it remained unchanged after checking everything. The saturation decreased slowly to 85 %, and she became very nervous.

20.1.9 What Invasive Actions Could You Now Take?

The pathophysiological causes of arterial hypoxia have been discussed in Case 1 (see Sect. 1.1.8). If time permits, further diagnostics should be done, such as an arterial blood gas and a chest X-ray. The airway and ventilation – if the patient is in danger of hypoxemia – must be secured by intubation and/or PEEP ventilation. Then, bronchoscopy can be done to rule out aspiration of gastric contents.

>> Dr. Blue decided to intubate and ventilate Mr. Graham. Anesthesia tech Maria had already

prepared everything for a rapid sequence induction. "Hopefully he won't aspirate," thought Dr. Blue, as she began the RSI. The endotracheal tube was just placed as her attending anesthesiologist, Dr. Eldridge, walked through the door. "I thought the times of intubation and plexus anesthesia were over, thanks to ultrasound" was his sinister comment. Silently, Dr. Blue was annoyed by her attending, because she felt that the plexus anesthesia was working very well.

Despite intubation and ventilation with PEEP and a F_iO_2 of 1.0, the S_pO_2 decreased to 84 %. Dr. Blue recruited her attending for assistance and advice. He glanced at the monitor, then on the anesthesia record, and then rolled his eyes. "Draw an arterial blood gas with hemoglobin co-oximetry," he said to Dr. Blue. After a few minutes, the blood gas came back with the following values:

- *P_aCO_2: 34 mmHg (reference 35–46 mmHg)*
- *P_aO_2: 468 mmHg (reference 72–100 mmHg)*
- *pH: 7.43 (reference 7.35–7.45)*
- *BE: 0.7 mEq/l (reference ± 2 mEq/l)*
- *Fractional saturation: 94.7 % (reference 95–99 %)*
- *MetHb: 7.5 % (reference 0.2–1.5 %)*
- *COHb: 0.2 % (reference nonsmoker 0.5–1.5 %, smoker 5–20 %)*

20.1.10 How Do You Interpret These Findings?

The discrepancy between the measured S_PO_2 value of 84 % and the fractional O_2 saturation of 94.7 % is obvious. The high P_aO_2 is the result of ventilation with a F_iO_2 of 1.0. The methemoglobin value of 7.5 % is abnormal.

">> Okay," thought Dr. Blue as she read the arterial blood gas and co-oximetry values, "false measurement – everything is just fine. The intubation wasn't really necessary, the pulse oximeter needs to be sent in for repair."

Attending anesthesiologist Dr. Eldridge took the blood gas printout from her hand and said to himself, "Just as I expected," then left the OR. When he returned, Dr. Blue was quite upset to see that he brought a book with him: ***Complications and Mishaps in Anesthesia****. "You better go home now, think about your errors, and prepare a presentation for tomorrow about methemoglobinemia. I recommend Chapter 20." Dr. Blue left the OR. She felt that Dr. Eldridge had misunderstood all of her actions.*

20.2 Case Analysis/Debriefing

20.2.1 How Does Pulse Oximetry Work?

Every dissolved molecule possesses a specific light absorption behavior, as does oxygenated (O_2Hb) and deoxygenated/reduced hemoglobin (Hb). The Beer–Lambert law shows that a red beam (660 nm) and infrared (940 nm) LED (light-emitting diodes) have a specific spectral analytical behavior. In 660 nm, Hb absorbs almost 10× more light than O_2Hb; in 940 nm, there is an inverse result (see Fig. 20.1).

The capillary bed is subject to pulsating changes so that various amounts of light are absorbed in the transmission through the fingertip. Through several hundred measurements per second for each wavelength, the pulse oximeter differentiates the pulsatile (arterial) part of the transmitted light from the non-pulsatile part (caused by light absorption of venous blood, bones, and soft tissues). It is important to note that the pulse wave can only show changes of the blood volume and movements of the blood vessels walls, but not changes in pressure. Changes in the amplitude of the pulse wave amplitude during general anesthesia do correlate with neurological or hormonal determined changes of the smooth muscle tone of the artery wall.

During systole, the volume of the capillary beds increases so that the amount of absorbed light increases. In the plethysmographical pulse curve shown on the monitor, systole is shown in the trough of the curve (low intensity). The maximum point of the plethysmographical pulse curve represents diastole (higher intensity). Usually, a "swing" of the curve represents inadequate blood volume. The swing is often synchronous with breathing and can be caused by volume deficiency, but not always. Preemptive conclusions with therapeutic consequences should be avoided.

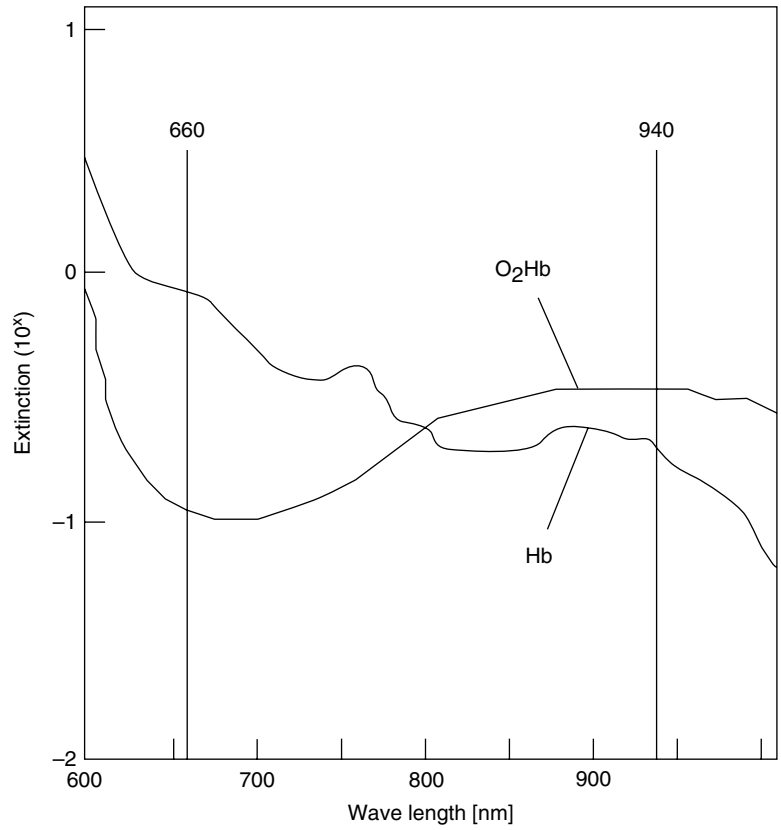

Fig. 20.1 Hemoglobin absorption curve of oxygenated O_2Hb and deoxygenated Hb (From Sinax [2] with permission)

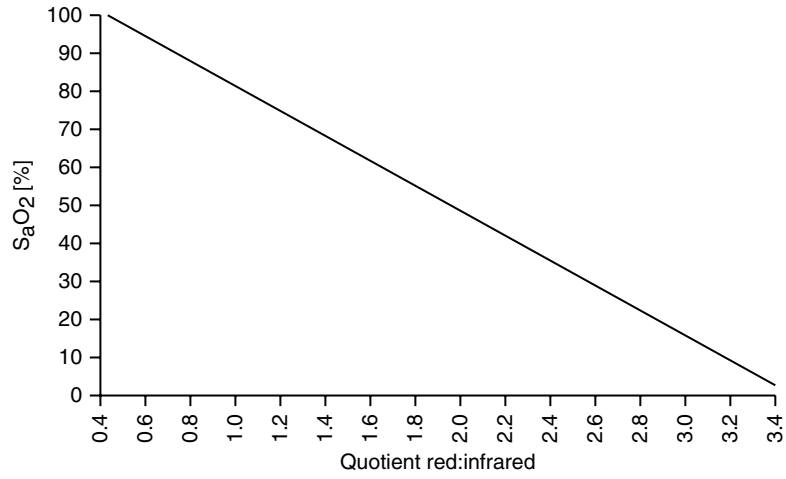

Fig. 20.2 Linear correlation of the relative absorption spectrum relation of red/infrared to oxygen saturation (From Sinax [2] with permission)

The relative absorption ratio of red/infrared reflects the ratio of reduced Hb to O_2Hb and can be calculated into the oxygen saturation (see Fig. 20.2).

Limitations of pulse oximetry are obvious: above all, the measurement is invalidated by optic interference of substances which also absorb red and infrared light. Deoxyhemoglobin is clinically important and is caused by carbon monoxide (COHb) and MetHb binders. Figure 20.3 differentiates from Fig. 20.1 in that the hemoglobin absorption curves of COHb and MetHb are listed.

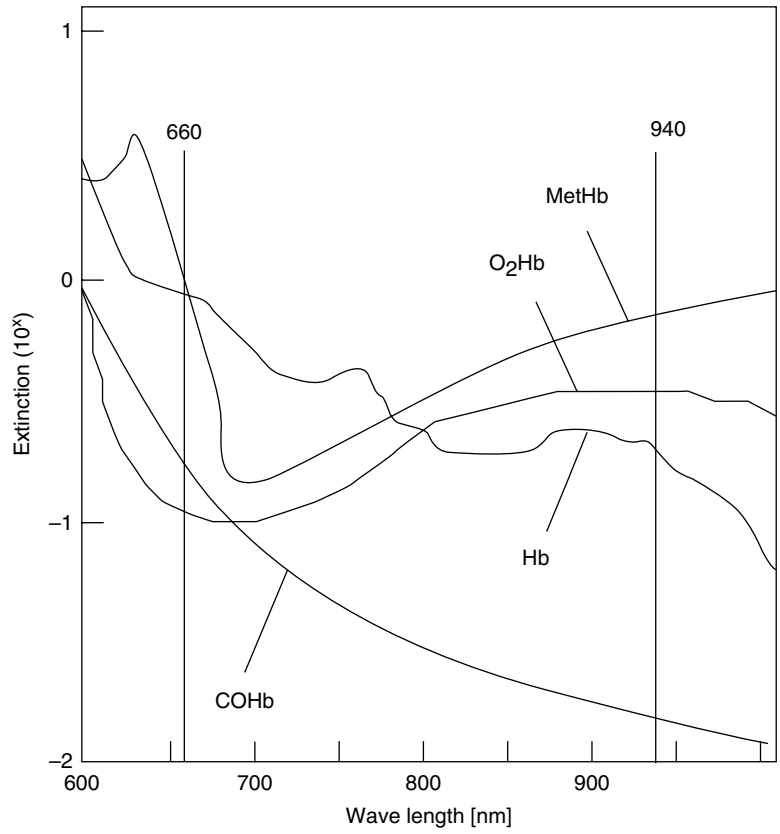

Fig. 20.3 Hemoglobin absorption curve of oxygenated O_2Hb, reduced Hb, carboxy-Hb (COHb), and MetHb (From Sinax [2] with permission)

The two-wave pulse oximeter, most often used in the hospital, can only differentiate between oxygenated and deoxygenated hemoglobin. This gives the partial saturation (S_PO_2), which is also known as functional saturation:

$$\text{Partial saturation } S_PO_2 = \left[O_2Hb / \left(O_2Hb + Hb\right)\right] \cdot 100\,\% \quad (20.1)$$

A two-wave pulse oximeter cannot differentiate between deoxyhemoglobins and oxygenated or reduced hemoglobin. Incorrect values are given into the equation.

- COHb hardly absorbs infrared light but behaves as O_2Hb with red light spectrum. The result is that the S_pO_2 value is falsely elevated.
- MetHb absorbs light in the red spectrum similarly to reduced Hb. In the infrared spectrum, MetHb absorbs more light than is absorbed by O_2Hb and Hb so that the relative absorption ratio of red to infrared approaches 1, and the measured S_PO_2 is about 85 % (Fig. 20.2). This means that with high MetHb (>10 %) the S_PO_2 value of 85 % is falsely high and in low MetHb values (<10 %) the S_PO_2 value of 85 % falsely low.

If a dyshemoglobinemia is suspected, an absorption photometer with at least four wavelengths must be used for measurement. The levels of four forms of hemoglobin are determined, and the real oxygen saturation is calculated with the formula:

$$\text{Fractional saturation } SO_2 = \left[O_2Hb / \left(\begin{matrix} O_2Hb + Hb + \\ COHb + MetHb \end{matrix}\right)\right] \cdot 100\ \% \quad (20.2)$$

Interestingly methylene blue, which is given IV as therapy for MetHb, absorbs the maximum light at 660 nm so that it causes a drastic decrease in the measured S_pO_2 for a few minutes. Apart from causing artifacts when attempting oxygen

saturation measurement, COHb and MetHb cause a left shift of the oxygen binding curve with decreased release of oxygen into tissues [2].

20.2.2 What Causes Methemoglobinemia?

The hemoglobin in the erythrocyte is made up of 4 heme molecules with a central iron ion (Fe^{2+}). During transport, O_2 binds to the heme component, creating oxyhemoglobin (O_2Hb). Apart from oxygenation of the hemoglobin, a true oxygenation of the iron can occur in that the Fe^{2+} enters an Fe^{3+} state. This creates methemoglobin, which is no longer available for oxygen transport.

In erythrocytes, the continuously generated MetHb is reduced by the NADPH (nicotinamide adenine dinucleotide phosphate hydrogenase)-dependent methemoglobin reductase back to hemoglobin.

Usually, human blood has a very little portion of MetHb; however, certain diseases and medications can increase MetHb. Glucose-6-phosphate dehydrogenase (G6PD) is a required enzyme of the pentaphosphate cycle so that the result of an enzyme defect (favism) results in a deficiency of glucose-6-phosphate dehydrogenase and an increase in the formation of MetHb.

Dose-dependent methemoglobinemia occurs after administration of prilocaine. In order to reach a clinically relevant level, a total of 600 mg of prilocaine is needed. However, methemoglobinemia can occur after smaller doses, dependent on individual factors and speed of absorption. Prilocaine is metabolized in the liver to toluidine, which is responsible for the oxidation of MetHb.

20.2.3 Which Medical Errors Do You See in the Presented Case?

20.2.3.1 Placement of Regional Anesthesia

Resident Dr. Blue was inexperienced in the technique. A significant amount of time was needed to facilitate the block. Furthermore, an unusually large amount of prilocaine was injected. This error is often observed in inexperienced anesthesiologists. Her attending should have been present throughout the entire block.

20.2.3.2 Academic Preparation

Dr. Blue obviously was not sufficiently prepared or experienced for regional and local anesthesia. Specifically, she had no knowledge of the typical side effects of prilocaine nor the maximum dose allowed.

20.2.3.3 Checking Success of the Block

After placement of regional anesthesia, there was no check of the block. This must be done before the patient receives the final OK for surgery.

20.2.3.4 Latex Allergy

Dr. Blue, anesthesia tech Maria, and the OR personnel underestimated the potential for a latex allergy. A specific history taking for differentiating between an allergy and sensitization did not occur.

20.2.3.5 Preoperative Evaluation

The injury occurred in a bicycle accident. Dr. Maria didn't question the incident further, so underestimation of possible comorbidities can be assumed.

20.2.4 Which Systems Failures Can You Find in the Presented Case?

20.2.4.1 Information Exchange

The unexpected patient was announced during the morning hours. The information, however, did not get to Dr. Blue. In addition, Dr. Blue did not inform her attending that the patient was in the preoperative holding area.

20.2.4.2 Preoperative Evaluation

The anesthesiologist-in-charge, who was responsible for the organization, neglected to assign someone to perform a preoperative assessment before the patient was brought to the preoperative holding area. As a result, the opportunity to order

preanesthetic studies was limited. Even so, from a patient's point of view, an informative discussion of anesthetic risks/benefit, especially with planned regional anesthesia, best takes place before being transported for surgery.

20.2.4.3 Latex Allergy

Patients with known and unknown latex allergies are quite common. In the OR, there were no specific algorithms clarifying the procedures for such patients.

20.2.4.4 Education and Training

Dr. Blue was new in trauma surgery and inexperienced in regional anesthesia. She began placement of the block without orders or supervision and didn't call her supervisor after difficulties arose. This can be due to an extremely high level of self-confidence on the part of the anesthesiologist or an absence of structured training in the department.

20.2.5 Imagine You Are the Anesthesiologist on a Spaceship to the Andromeda Galaxy. Suddenly Your Beeper Goes Off for Pain Relief in the Labor and Delivery Floor. How Would You Do the Epidural?

Before we answer this question let's get back to the case: Dr. Blue's pharmacological knowledge had space for improvement. She didn't know the side effect of prilocaine – false saturation measurement due to MetHb formation. Therefore, she was unable to completely plan the anesthesia nor was she able to identify the complication. She endangered the patient by injecting large amounts of prilocaine. As the saturation decreased, Dr. Blue attempted to treat an oxygenation disorder, which was not there. Adequate diagnostic studies – such as an arterial blood gas analysis – did not occur. The possibility of an artifact was underestimated since Dr. Blue had never experienced such a situation before. She was subject to a so-called "**availability heuristic**" mistake of judgment [6].

The case shows the connection of contributing incidental factors: knowledge insufficiency together with lack of confirmation ended in a general anesthesia and thereby put the patient at unnecessary risks. The large protective barrier of the team is not capable of preventing the mistake due to lack of supervision by the attending anesthesiologist.

And now to the question: Your knowledge of physiology in zero gravity situations has potential to improve. You don't know the effects of interstellar flights on intrauterine contraction. Accordingly, anticipation of complications is difficult and perhaps impossible for you. You may make an availability heuristic judgment mistake – unless you studied the subject in advance [1] – or you have an experienced co-worker on your team, and you ask that co-worker for help!

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Case 21: Colectomy

21

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Contents

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21.1 Case Introduction

>> This Tuesday morning, Dr. Ferdinand just couldn't get out of bed on time. He had slept so deeply that he didn't hear his alarm clock. He only managed to move into the bathroom after his girlfriend warned him for the second time that he was going to be late. Then everything had to go really quickly – the coffee was too hot to drink, no time for toast. He heaved himself onto his bike and stopped at the baker to pick up some fresh bagels before finally arriving at the hospital somewhat on time. Dr. Ferdinand was an anesthesiology resident in a large city hospital and already in his fourth year. Thank goodness, there were no complicated cases on his schedule for the day. He knew already that the first patient was to have a proctocolectomy with pouch placement.

Just barely on time, he entered the preop holding area. The patient, Mr. Gray, was waiting. Anesthesia tech Denise had already hooked up the basic monitoring and placed him on a

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drip. Dr. Ferdinand didn't know the patient. He reviewed the preoperative evaluation and reviewed the patient's medical record. Mr. Gray was 43 years old and 172 cm tall and weighed 85 k.

For 20 years, he had suffered from ulcerative colitis, which had spread through the entire large intestine early on. Actually, with conservative therapy, the clinical course was satisfactory. The yearly colonoscopy and biopsy had now shown a low-grade dysplasia for the second time. After the 10th year of suffering from ulcerative colitis, the risk of cancer is significantly increased, so the indication for an elective colectomy and ileum pouch was certain.

Dr. Ferdinand also noticed in the medical record that the patient had heart disease, CHF NYHA Class 1, and systemic hypertension. Mr. Gray was a heavy smoker. Dr. Ferdinand silently shook his head disapprovingly. The primary care physician had prescribed sulfasalazine and metoprolol. The patient had given informed consent for combined general and epidural anesthesia, as well as placement of a CVC and an invasive arterial catheter. Lab values:

- *Hb: 13.4 g/dl (reference 12–14 g/dl)*
- *HCT: 42 % (reference 37–47 %)*
- *Platelet count: 167,000 mc/l (reference >150,000 mc/l)*

All other values unremarkable as well.

21.1.1 Which Tests Would You Have Ordered in Addition to the Standard Diagnostic Studies Listed Above?

Mr. Gray is 43 years old and has a positive CHF and smoking history. Even though the patient does not complain of angina, a preoperative ECG should be done (see also Case 5, Sect. 5.1.1).

>> *"Now," Dr. Ferdinand said, stepping to Mr. Gray's bedside, "you don't seem to be too nervous." He looked at the monitor, which showed a blood pressure of 160/80 mmHg, an 80 beats/min sinus rhythm, and a S_PO_2 of 98 %. The patient shrugged his shoulders, "I know what has to happen. I hope that it will all be over as soon as possible; I can't wait to get home again."*

"All right then, let's get started," said Dr. Ferdinand. The attending anesthesiologist stopped by and watched Dr. Ferdinand place the thoracic epidural catheter without incident. Intubation and placement of the CVC and arterial catheter went well, and after a total of 25 min, Mr. Gray was pushed into the OR. The anesthesia was maintained with a mix of desflurane in air and oxygen, supplemented with intermittent regular epidural injections. Further medications were not needed.

Dr. Ferdinand stood by the computer screen, completed the anesthesia record, and mentally prepared himself for a long case. The blood pressure remained stable at 140/70 mmHg; heart rate was between 40 and 50 beats/min. After about 2 h, the blood pressure decreased to 115/55 mmHg, but the heart rate remained unchanged. Dr. Ferdinand got bored. "There is nothing for me to do," he thought. "There is no need to even do a blood gas analysis, everything is so stable. But what does my attending always say to me? A good anesthetic must be boring; if it's not, you're doing something wrong."

Finally, after about 4 h, the surgery came to an end. Dr. Ferdinand noted the fluid balance: infusion of 1.5 l balanced electrolyte solution, minimal blood loss of 300 ml, and, oops, he remembered that he hadn't placed a urinary catheter. "Well, can't change that now," he thought to himself. Mr. Gray's body temperature was 36.0 °C. Promptly after the surgery ended, Mr. Gray's trachea was extubated. Everything went without incident. Before the anesthesia team brought the patient into the PACU – which was 2 long hallways away – Dr. Ferdinand made one last glance at the monitor. Mr. Gray was doing just fine; he could be easily awakened, followed orders, and was hemodynamically stable with S_PO_2 97 %. "Let's go – we're going to the PACU. The patient can be further monitored there," said Dr. Ferdinand to anesthesia tech Denise.

21.1.2 What Must Be Watched for with Bowel: Especially Colorectal – Surgery?

21.1.2.1 Mesenteric Traction Syndrome

In the early surgical phase, mesenteric traction syndrome can occur, with hemodynamic instability. Pathophysiology and therapy are presented in detail in Case 26 (see Sect. 26.1.4).

21.1.2.2 Bleeding

Bleeding occurs rather seldom in colorectal surgery, but that should not affect the watchful eye of the anesthesiologist.

21.1.2.3 Volume and Electrolyte Imbalances

Although modern colon surgery techniques do not require extensive bowel preparation, many hospitals still do extensive cleansing [5]. Typical bowel preps are hypertonic and contain, for example, macrogol, potassium chloride, sodium chloride, and sodium citrate. Preoperative use of bowel preps leads to hypovolemia and changes in osmolality and serum electrolytes. Elderly patients with reduced heart, lung, and renal function are especially at risk.

Even without a bowel prep, bowel surgery leads to intravascular volume and electrolyte changes, since manipulation of the intestines affects its function and permeability. The intraoperative management of fluid and volume therapy has been heavily studied in the past few years. It has been shown that restrictive administration of volume improves postoperative outcome and perioperative morbidity and decreases the hospital stay [3, 9].

21.1.3 How Do You Guide Fluid Therapy?

Reference points for adequate fluid therapy involve several aspects. The preoperative fasting state is rather limited these days, as clear fluids may be consumed for up to 2 h prior to surgery. Also, the fluid requirement per hour in a sedentary state has been significantly overestimated. One further aspect is the intraoperative fluid loss/loss into the so-called third space. This too is often overestimated [4] so that restrictively adapted fluid replacement is generally needed.

Reference points for adequate volume therapy include:

- Hemodynamics
- Urine production
- Course of the central venous pressures (CVP) over time
- Changes of the hematocrit and lactate over time

The iatrogenic sympatholytic effect of the epidural may be counteracted with not only fluids but also catecholamines. There is also no rational reason to replace always the first 1,000 ml blood loss with three to four times as much crystalloid fluid, even though this recommendation is often seen in textbooks. It is wiser to adapt fluid therapy depending on patient's condition and comorbidities. In otherwise healthy patients with normal hematocrit values at the beginning of surgery, the blood loss may be treated with catecholamines up to a certain value. Prerequisites are a very vigilant anesthesiologist who closely evaluates markers for tissue perfusion (e.g., lactate, pH, central venous saturation), intravascular filling (e.g., pulse pressure variation), and heart perfusion (e.g., ST segment analysis).

For many years, the anesthetic literature has debated the proper fluid therapy [8]. Primary indications for crystalloids are to offset the loss through insensible fluid loss (perspiration) and urine production. Overly generous administration worsens tissue oxygenation because isotonic crystalloid solutions diffuse into the entire extracellular room. It may also increase perioperative blood loss. Colloidal solutions remain mostly intravascular, but their administrations are very controversial. Recently, the Pharmacovigilance Risk Assessment Committee (PRAC) of the European Medicines Agency [11] and the FDA [12] issued warnings regarding the use of hydroxyethyl starch solutions (HES). The background is that data analysis showed "increased

Table 21.1 Recommendations from the FDA for the use of HES [12]

Do not use HES solutions in critically ill adult patients including those with sepsis and those admitted to the ICU
Avoid use in patients with preexisting renal dysfunction
Discontinue use of HES at the first sign of renal injury
Need for renal replacement therapy has been reported up to 90 days after HES administration. Continue to monitor renal function for at least 90 days in all patients
Avoid use in patients undergoing open heart surgery in association with cardiopulmonary bypass due to excess bleeding
Discontinue use of HES at the first sign of coagulopathy

mortality and/or renal injury requiring renal replacement therapy, i.e., severe renal injury, when HES was used in critically adult patients including patients with sepsis and those admitted to ICU" [12]. PRAC concluded that "the available data only showed a limited benefit of HES in hypovolemia, which did not justify its use considering the known risks" [11]. Table 21.1 lists the recommendations of the FDA.

>> *It took 5 min for Dr. Ferdinand to get Mr. Gray into the PACU. Anesthesia tech Denise hooked the patient up to the monitor, and Dr. Ferdinand relaxed a bit as he finished the anesthesia record on the computer. The PACU physician was busy with another patient during that time. Just as Dr. Ferdinand wanted to report the uneventful procedure to the now attentive PACU doc, he was dumbstruck by the monitor reading.* "*What is going on*?" *he asked himself out loud. Mr. Gray calmly lay in bed, but his pulse was fluttering between 140 and 150, and his blood pressure was 85/43 mmHg.* "*As we left the OR, everything was fine. Look*! *Just look at the record*!" *He held it up for all to see. The PACU doc shrugged his shoulders.* "*Yeah, well, it's not quite the same now. Can you figure something out*? *I have two new patients that need my help*." "*Yes, of course*," *said Dr. Ferdinand. Silently, he was infuriated with himself for transferring Mr. Gray to the PACU in such condition.*

21.1.4 What Steps Should Dr. Ferdinand Do Next?

The patient has hemodynamically significant acute tachycardia. Further steps to diagnose the tachycardia are necessary, such as questioning the patient about possible causes (e.g., pain) and symptoms, pulmonary and cardiac auscultation, and a 12-lead ECG.

Dr. Ferdinand went to Mr. Gray's bedside, where he was found to be calm and pain-free. He did say that he felt his heart racing, like he had never felt before. Auscultation of the lungs was unremarkable. Anesthesia tech Denise had finished the 12-lead ECG and presented it to Dr. Ferdinand (see Fig. 21.1).

21.1.5 What Is Your Diagnosis?

The diagnosis is tachycardia due to atrial fibrillation (a-fib).

>> "*Man*!" *said Dr. Ferdinand and asked the PACU nurse to measure the CVP stat, then to give 1,000 ml of crystalloid infusion. Afterwards he planned to….*

21.1.6 What Would You Do as Further Diagnostic and Therapeutic Measurements, if You Were Dr. Ferdinand?

An arterial blood gas is of utmost importance, in order to rule out electrolyte imbalance and acidosis as reversible causes for the occurrence of a-fib. A primary concern is a reduction in sympathetic tone. The patient should be questioned about other stress factors besides pain, such as fear. The epidural level should be checked as the patient might be worried about not being able to move his legs.

It is also important to increase the inspiratory oxygen concentration via face mask, to achieve an optimal cardiac oxygen supply.

Hypovolemia as a reversible cause of acute a-fib was considered and correctly treated by Dr. Ferdinand. The CVP should be measured before administrating

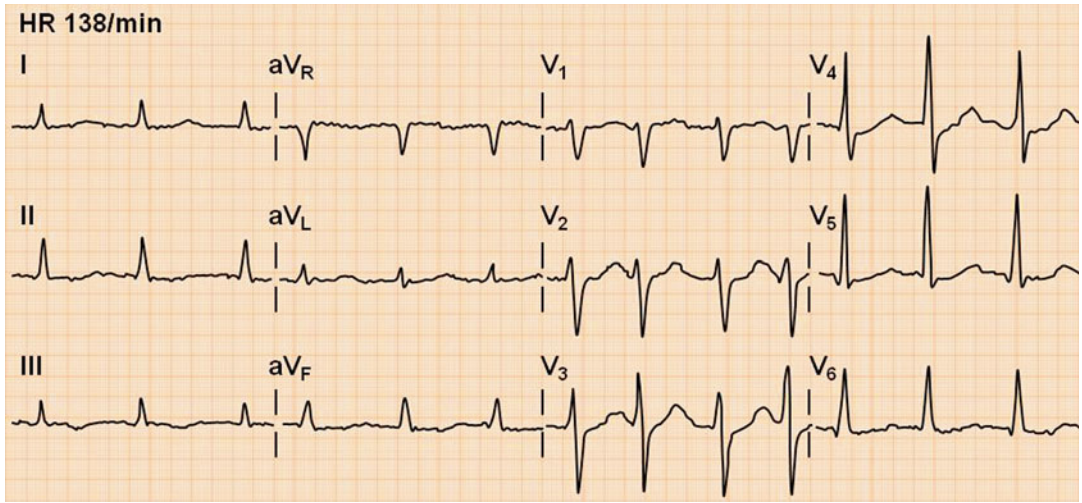

Fig. 21.1 12-lead ECG

fluids is also correct. It should be repeated. Volume overload must be avoided in CHF patients.

>> *Dr. Ferdinand viewed the blood gas and electrolytes – both unremarkable, Hb 15.2 g/dl (reference 12–14 g/dl) and HCT 49% (reference 37–47%). He silently recapped the procedures.* "*Mr. Gray received fluids, without a cardiovascular effect. Acidosis and electrolyte imbalance are ruled out, he has no pain, he is receiving extra oxygen* via *face mask, and the atrial fibrillation is still there. Well, then I have no other choice but to…*"

21.1.7 What Must Now Be Done? How and Why?

Acute a-fib is always an emergency situation. Possible causes are discussed in Sect. 21.2.3 in the Overview and in Table 25.1. After ruling out reversible causes, attention focuses on therapy. In this case, only therapy for acute a-fib will be discussed (therapy of chronic a-fib, see Anderson et al. [2]).

The therapy has three goals:

- Heart rate control
- Correction of the cardiac rhythm
- Protection from thromboemboli

The American Heart Association has developed guidelines which are regularly revised according to new medical knowledge [1, 2, 10]. The guidelines for therapy of a new-onset tachycardia are outlined in the algorithm in Fig. 21.2 (modified according to [1, 10]). An important prerequisite for the use of this algorithm is hemodynamic stability.

The antiarrhythmic therapies are determined from the stability of the patient. The most important symptoms of hemodynamic instability are listed in the Overview.

>> *The most important symptoms of hemodynamic instability are*:

- Hypotension <85 mmHg systolic
- Chest pain
- Acute heart failure
- Cognitive dysfunction, syncope

In the presented case, moderate hemodynamic instability is present. A synchronized electrical cardioversion should be performed if the patient is symptomatic (e.g., chest pain, mental status changes, syncope, signs of shock), which can be repeated up to 3 times. If the arrhythmia is still present, 300 mg amiodarone should be given over 10–20 min. Afterwards, additional cardioversion may be attempted, and, if needed, 900 mg of amiodarone may be given over 24 h. The success rate is high. Up to 95 % of patients convert to a sinus rhythm with an applied energy of 360 J [2, 7].

In hemodynamically stable patients, the therapy is determined by the QRS morphology (broad

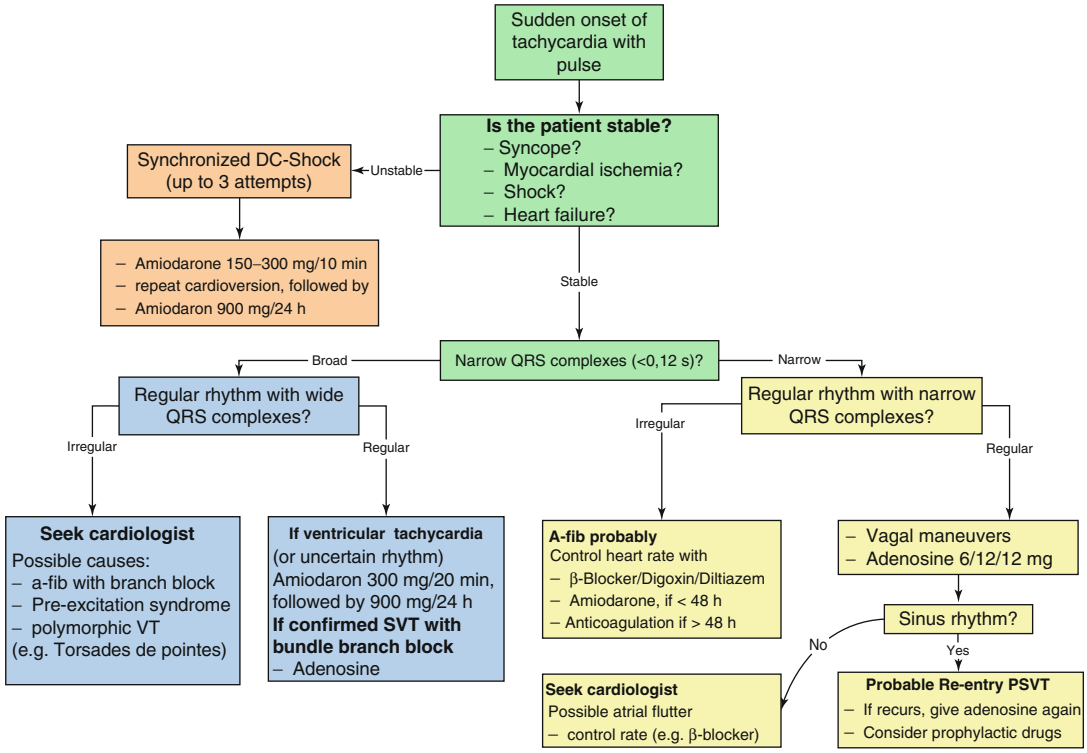

Fig. 21.2 Algorithm for new onset of tachycardia with pulse

or narrow complex). If acute a-fib is confirmed, and the patient has no symptoms, maintaining frequency control is the first priority because there is a high spontaneous conversion rate within the first 24 h. β-blockers or calcium antagonists (diltiazem, verapamil) are recommended. If these measures fail or are contraindicated, amiodarone should be given.

21.1.7.1 Synchronized Electrical Cardioversion

Synchronized electrical cardioversion means that an electric shock is synchronized with the R wave of the ECG. Most defibrillators today have the option to synchronize. Only these defibrillators may be used for cardioversion. There is still discussion as to the best energy choice for cardioversion. Biphasic waveform defibrillators offer a higher success rate and a lower energy level than monophasic. The joules needed are chosen in the following manner [2, 10]:

- In wide complex tachycardia and a-fib, it is recommended to begin with 120–150 J biphasic or 100–200 J monophasic waveforms. If additional shocks are necessary, energy levels should be increased with the third shock at the maximum energy level.
- In atrial flutter or regular narrow complex tachycardia, a lower energy level is chosen.

It is begun with 70–120 J in biphasic or 100 J in monophasic waveform. Possible additional shocks should be given with increased energy levels, again, the third one with escalating energy of the defibrillator.

A basic requirement for defibrillation is analgesia and sedation or general anesthesia.

In hemodynamically stable patients with new onset of a-fib (less than 48 h), pharmacological cardioversion can be attempted. Seek expert help and consider ibutilide, flecainide, dofetilide, or amiodarone. For these patients, electrical cardioversion should always be considered as the rate of success is higher.

21.1.8 What Are the Risks and Side Effects of Cardioversion?

Typical risks of the electric cardioversion are:
- Thromboembolism
- Arrhythmia

Thromboembolism occurs in 1–7 % of patients who do not receive therapeutic anticoagulation. Cerebral infarction is most often encountered. But there are also cases of coronary emboli, myocardial infarction, and stroke after a left atrial thrombus was ruled out by echocardiography [6]. The cause is a transient mechanical dysfunction of the left atrium, also called "stunning", which occurs during the electrical or pharmacological treatment, or spontaneously, and has a thrombogenic effect.

Return of the left atrium to normal function can take weeks and depends on the duration of a-fib. This explains the appearance of thromboembolism in patients with an a-fib duration of <48 h and no evidence of a thrombus in the left atrium. If the duration of an a-fib is over 48 h (or if the duration is unknown), anticoagulation is recommended (initial intravenous bolus followed by a continuous infusion to maintain activated partial thromboplastin time at 1.5–2 times the reference value) [2, 7].

Anticoagulation decisions must be made on an individual basis, especially in the perioperative situation – to weigh the risk of recurrent hemorrhage against the risk of a thromboembolism [2, 7]. The continuation of the therapeutic anticoagulation is not recommended postoperatively, if the a-fib was present for less than 48 h.

During cardioversion, arrhythmias of all forms may occur, which require appropriate therapy. To ensure a safe and effective cardioversion, the serum potassium level must not be below normal. Magnesium substitution does not increase the success of the electrical cardioversion [2], but can help to control rhythm <100/min, in combination with digoxin [16].

>> *In response to Mr. Gray's continuing a-fib, the PACU was prepared for cardioversion. Dr. Ferdinand had called the surgeons, who had no problems with anticoagulation. More than 6 h had passed since placement of the epidural, so Mr. Gray received 5,000 units heparin IV. Dr. Ferdinand spoke with Mr. Gray and explained the situation and the planned procedures.* "*My God*!" *said Mr. Gray*, "*An electric shock? Do we have to? Will I feel something*?" *Dr. Ferdinand calmed him down saying*, "*I will give you something to make you sleep, and you won't feel a thing*."

Everything was prepared, the pads were adhered properly on the chest, and the defibrillator was synchronized. Dr. Ferdinand set 150 J and asked anesthesia tech Denise to give 0.5 mg alfentanil and 150 mg propofol. Mr. Gray slept. "*Everybody back*" *said Dr. Ferdinand loud and clear, then gave the shock. Mr. Gray twitched as the electricity flowed through his body. Dr. Ferdinand ventilated him for a short time* via *a face mask.*

The PACU physician came to the bedside and asked, "*Fixed it*?" *A deep humiliation came over Dr. Ferdinand as he now realized that he had forgotten to check the rhythm. He looked now*; *Mr. Gray had a sinus rhythm of 70 beats/min* (Fig. 21.3). *Blood pressure was 120/55 mmHg.*

"*Yep, we've got it now*," *he said with a thankful breath. After all of this, Dr. Ferdinand really needed to take a break and eat his bagels.*

21.2 Case Analysis/Debriefing

21.2.1 Define Atrial Fibrillation!

Atrial fibrillation (a-fib) is the most common arrhythmia requiring treatment. A-fib is defined as a supraventricular tachyarrhythmia, caused by fast irregular chaotic atrial activity and an obligatory worsening of atrial function. The pathogenesis is complex. Two requirements must be met:
- First, an initiating incident
- Second, an anatomical substrate

There are various theories about the mechanism of a-fib. Some blame an aberrant pathway; some blame multiple small waves causing reentry. Neither mechanism completely rules out the other, but can arise simultaneously in patients. The theory of an aberrant pathway is supported by various experiments and success of ablation therapy [2]. In ECG, the a-fib is characterized by oscillating

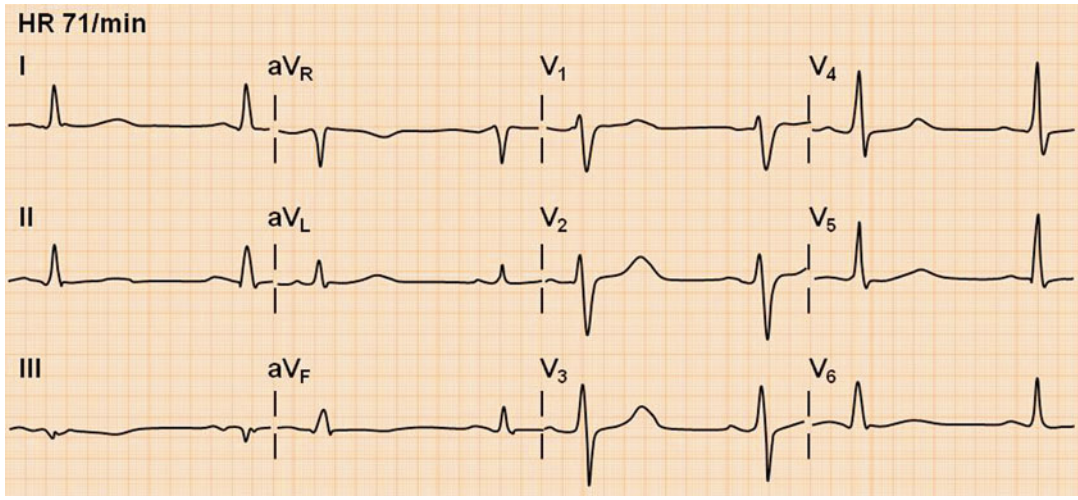

Fig. 21.3 12-lead ECG of the patient after successful cardioversion

and fibrillating waves with varying forms, amplitudes, and rates. The atrial frequency is between 350 and 600/min, resulting in irregular, rapid ventricular transfer. The ventricular rate is dependent on the electrophysiological conditions of the AV node, which should provide a filter function:

- From the level of vagal and sympathetic tone
- From the presence of accessory pathways
- From the effects of medications [2]

The QRS complexes are usually narrow. Rapid atrial discharges cause loss of atrial contraction and therefore reduce ventricular filling. Cardiac output decreases up to 20 %.

The Wolff–Parkinson–White syndrome – a special form of supraventricular tachyarrhythmia – requires special therapy and a watchful eye and will not be discussed in detail here.

The classification of a-fib depends on the clinical occurrence. A differentiation is made between:

- Acute a-fib: first appearance/detection
- Chronic a-fib, which is further divided into:
  - Paroxysmal a-fib: self-limiting episodes
  - Persisting a-fib: occurrences which can be converted to a sinus rhythm by cardioversion
  - Permanent a-fib: occurrences which cannot be permanently converted into a sinus rhythm

Since this classification cannot completely cover the new treatment methods – for example, chronic a-fib in existence for several years may be converted to a sinus rhythm by new complex ablation therapies – there is also differentiation between the following [15]:

- Acute, paroxysmal a-fib
- A-fib of short duration (<1 year)
- Extended duration persisting a-fib (>1 year)

The prevalence of a-fib in the general population is 0.4–1 %. It increases with age, so that after age 65, the incidence is 5 % and after age 80, the incidence is about 10 % [13].

21.2.2 What Are the Clinical Signs and Symptoms, and What Complications May Arise Due to A-fib?

Typical symptoms of a-fib include palpations, dizziness, dyspnea with possible chest pain, and cough. Due to the hemodynamic compromise, decrease of cardiac output, and secondary decrease of blood pressures, episodes of syncope can occur, especially under physical stress.

The absent productive atrial function detrimentally affects ventricular filling and thereby reduces stroke volume. The ventricular tachycardia aggravates the reduction of stroke volume, by shortening diastole. The acute-onset a-fib especially decreases the cardiac output, up to

20 %, and can lead to acute heart failure with a poor prognosis, angina pectoris, and acute pulmonary edema. A-fib causes changes in the myocardium and a progressive dilation of the left atrium. When a-fib progresses uncontrolled, left ventricular dilation with reduced systolic pump function can result.

Apart from the above described cardiac and hemodynamic effects, a-fib also raises the danger of arterial emboli, especially the risk of stroke. The reduced flow through the left atrium causes thrombotic material to collect, usually in the area of the left auricular appendage. The longer the a-fib is present, the higher the risk of emboli. In patients with a new-onset a-fib for less than 48 h, about 15 % had an atrial thrombus that was detected by transesophageal echocardiography [14].

21.2.3 What Are the Predisposing Factors for the Occurrence of A-fib in the Perioperative Setting?

The main risk factors for the occurrence of cardiac arrhythmia in the perioperative setting are (according to Butte et al. [7]) listed in the Overview.

21.2.3.1 Risk Factors for Occurrence of Cardiac Arrhythmia

- Statistical factors:
  - Age
  - Male gender
  - History of arrhythmia
  - Coronary heart disease
  - Heart failure
  - Congenital heart defect
  - Valvular heart disease
  - Cardiomyopathy
  - COPD
  - Heart/thoracic surgery
- Reversible factors:
  - Stress, adrenergic stimulation, fear
  - Hypoxia/hypercapnia
  - Acidosis
  - Electrolyte disorders
  - Hypothermia/hyperthermia
  - Hypovolemia/shock
  - Perioperative myocardial ischemia
  - Mechanical irritation
  - SIRS, sepsis
  - Pro-arrhythmogenic medications (e.g., atropine or glycopyrrolate)

The incidence of perioperative cardiac arrhythmia is highly variable, dependent on the type of surgical procedure, the types of patients and their comorbidities, the definition of arrhythmia, and so on. Arrhythmias are reported for about 10–40 % of the cardiac and thoracic patients and about 4–20 % for the vascular and abdominal surgery patients. Atrial fibrillation is the most common perioperative arrhythmia, with occurrence in 5 % of the noncardiac, non-thoracic procedures and 30–50 % in heart/thoracic surgery [7].

21.2.4 Which Medical Errors Do You See in the Presented Case?

21.2.4.1 Preop Checkup

The missing ECG was already mentioned in Sect. 21.1.1.

21.2.4.2 Neglected Blood Gas Analysis

In Sects. 21.1.2 and 21.1.3, the problems of perioperative fluid balance in colon surgery were discussed. The fluid restriction with fast-track surgery requires an especially watchful eye and adequate monitoring including:

- Regular hematocrit checks to estimate insensible fluid loss
- Regular checks of the lactate level and pH for monitoring tissue perfusion
- Electrolyte checks (see below)

21.2.4.3 Urinary Catheter

It was careless to conduct the operation without a urinary catheter. The catheter is critically necessary to monitor volume therapy, especially when fluid restriction is planned.

21.2.4.4 Fluid Therapy

Mr. Gray did not receive enough fluid during the operation. Therefore, his hematocrit increased from 42 % preoperatively to 49 % postoperatively – even after 1,000 ml of a crystalloid infusion was given in the PACU. One must assume

that the volume deficit was more pronounced at the end of surgery.

21.2.4.5 Electrolyte Balance Checks

In the presented case, at least one intraoperative arterial blood gas should have been drawn in order to monitor the electrolyte status. Electrolyte or volume abnormalities could have arisen due to the preoperative bowel prep and intraoperative manipulation of the intestine. The potassium value is a decisive factor in cardioversion success.

21.2.4.6 Monitoring

The transport of newly extubated patients into the PACU without monitoring and oxygen is reckless practice, unless the PACU is directly adjoining the operating room.

21.2.4.7 Preparation for Cardioversion

As discussed above, Mr. Gray received too little intraoperative fluid replacement and was hypovolemic. The blood gas immediately after the cardioversion showed a hemoglobin value of 49 %. Therefore, one of the reversible causes of a-fib – hypovolemia – was not yet treated. The cardioversion was successful despite the volume deficiency, indicating that Mr. Gray has a healthy heart. On the other hand, cardioversion may have been avoided if the hypovolemia was properly treated (spontaneous conversion is often observed).

21.2.5 Which Systems Failures Can You Find in the Presented Case?

21.2.5.1 Lack of Information Sharing with the Attending Anesthesiologist

All players in this case neglected to inform the attending physician about the a-fib. On the other hand, the attending is responsible for supervising the residents.

21.2.5.2 Availability of Monitoring

All patients who have received anesthesia should be transported to the PACU with supplemental oxygen. Even if the PACU is just a little further away from the OR, a portable monitor to measure vital signs should be used at all times for all patients. Every member of the anesthesiology team should be trained and required to use the monitoring without exception.

21.2.5.3 Monitoring the Fluid Therapy

As discussed in Sect. 21.2.4, the intraoperative monitoring of fluid balance was lacking. Setting standards and communicating the standard procedures can avoid this.

21.2.6 What Could Dr. Ferdinand Have Done to Avoid His Mistakes?

Dr. Ferdinand was watching over a general anesthetic, which presented no big anesthesiology challenge for him. Mr. Gray was relatively healthy, and Dr. Ferdinand didn't expect any surprises from the surgical side. The induction was the standard routine, which he quickly carried out, and then he moved into a phase with less workload. Accordingly, his activation level decreased, and his concentration faltered. He forgot the preoperative placement of a urinary catheter. Later he forgot to adequately monitor the fluid balance, a mistake which eventually led to the development of a-fib.

The process described above is typical for an anesthesiologist's work environment, as well as for many other professions. One achieves competence over the scope of tasks and then feels a lack of stimulation due to the lack of challenge with daily practice. This correlation between procedural difficulties (arousal for the challenge) and personal performance capability is described in the **Yerkes–Dodson law** (Fig. 21.4).

How can one maintain high performance during periods of low workload?

One strategy to actively resist a slack in vigilance during low workload is to cognitively anticipate problems or challenge yourself with solving fictitious problems: What would I do now if the oxygen were suddenly cut off? How much blood loss could my patient tolerate? Is it true that the surgeon had an affair with the nurse?

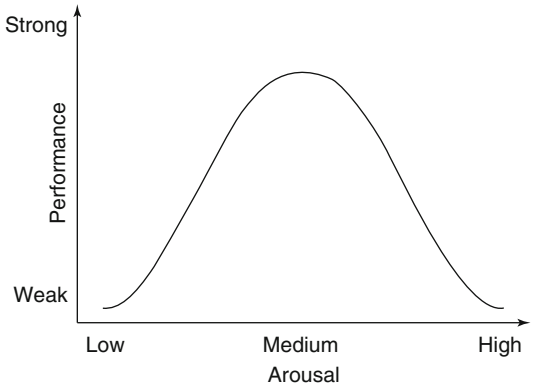

Fig. 21.4 Yerkes–Dodson law in the modification of Hebb [13]. An inverted U curve describes the correlation between different levels of arousal for the challenge and level of performance. During stressfully overwhelming periods and static boring periods, performance is not optimal. Optimal performance is achieved in tasks which are neither too demanding nor too routine

Apart from these personal strategies for maintaining vigilance, the employer also plays a role. The employer is responsible for creating a good working environment – like proper lighting and alarms and giving breaks – and is responsible to assign each worker tasks according to his or her capabilities.

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Case 22: Knee Joint Replacement

22

Katharina Martin, Mike Hänsel, Karen B. Domino, and Matthias Hübler

Contents

22.1 Case Introduction

>> Anesthesiology resident Dr. Sofia returned to work rejuvenated from her summer vacation. She was in her third year and recently rotated to orthopedics. Finally she could learn regional anesthesia techniques from the ground up. This is what she had been missing from her residency training. She entered the OR ready to get to work!

The first patient on this day was Ms. Kelly, who came for a hip replacement under spinal anesthesia. Dr. Sofia was confident in her spinals, and she promptly achieved a good block with bupivacaine 0.5 %. The surgical team began with preparing the patient. Dr. Sofia quickly filled out her anesthetic record because she knew that a complicated regional for a knee joint replacement was coming next, and she wanted to have enough time to prepare for it. Ms. Kelly received a sedative, because she didn't want to hear any saw or drill noises. She slept promptly after midazolam was given. Immediately after the first incision for Ms. Kelly's hip replacement,

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Dr. Sofia called for the next patient to be brought into the preoperative holding area.

The experienced CRNA Kathleen promised to stay in the OR to care for Ms. Kelly, whose vital signs were stable.

Dr. Sofia left the OR to care for the next patient, even though she was always nervous about leaving her patients with other health-care professionals. It just so happened that this orthopedic hospital placed great importance on quick patient turnover, so this was standard procedure to hand off the patient to another anesthesia provider.

If something were to happen, who would be responsible?

22.1.1 What Is the Legal Situation in the USA? In Other Countries?

In the USA, anesthesia is performed by specially trained personnel, including physicians (anesthesiologists) and nonphysician personnel, including nurses (nurse anesthetists or CRNAs) and anesthesia assistants (similar to physician assistants) [1–3]. The type of practice varies throughout the USA, depending upon state licensure requirements, hospital practice requirements, and local workforce issues. Anesthesia practice in most of the USA uses an anesthesia care team model, in which a physician anesthesiologist medically directs anesthesiology residents, nurse anesthetists, and/or anesthesia assistants. For medical direction, the anesthesiologist needs to be present at induction, emergence, all critical phases, and immediately available throughout the anesthetic, as well as be responsible for preoperative evaluation and recovery room care. Residents are generally supervised on a maximum of 2:1 ratio due to training requirements, whereas an anesthesiologist may medically direct up to 4 CRNAs. In about a third of states, CRNAs may work independently and do not require supervision or medical direction by an anesthesiologist, whereas anesthesia assistants generally work under the medical direction or supervision of an anesthesiologist. In some states, a surgeon may supervise a CRNA, often in rural areas where there are few anesthesiologists. In some urban areas of the USA, especially in the west, anesthesiologists may provide physician-only anesthesia. However, with changing reimbursement and health-care reform emphasizing the importance of nonphysician team members, this form of practice is continuing to diminish.

The type of practice differs significantly in other countries. For instance, in Germany, the BDA and the DGAI have issued guidelines for parallel anesthesia with delegation of monitoring [7, 9]. Germany has ruled against allowing anesthesia to be carried out by nonphysicians. The anesthesiologist should be responsible for all aspects. Simultaneously, the delegation allows limited monitoring to be done by specially trained nonphysicians, during the following circumstances:

- In patients without risk factors involving their health, the surgery, or the anesthesia.
- The nonphysicians must be trained and experienced.
- The nonphysicians may not perform any other tasks during this time.
- The nonphysicians may only act within standing orders. They may not decide therapeutic actions.
- The responsible anesthesiologist must be close by in order to take over the anesthesia if needed.
- During induction and emergence, the anesthesiologist must be present.

Dr. Sofia's thoughts were on the right track – she was responsible for signing out to an appropriate anesthesia provider. However, the attending anesthesiologist, in this case, Dr. Eldridge was the person ultimately responsible for the patient's anesthetic care and must adequately supervise residents and CRNAs.

>> *Ms. Pine was in the preoperative holding area – a typical ortho patient. Anesthesia tech Julie had already started an IV and hooked up the routine monitors. Dr. Sofia greeted the patient and glanced through her medical record. The preoperative evaluation listed the following*:

- *69 years old, obese, BMI 39.8 kg/m², and 160 cm tall.*
- *Diabetes mellitus type 2, controlled with glyburide 2–1–0, held this morning.*
- *Systemic hypertension, sufficiently controlled with a combination of hydrochlorothiazide*

12.5 mg, ramipril 5 mg, and metoprolol 100 mg. She had taken one of each tablet this morning, as usual.

- *Hyperlipidemia, treated with a statin, 20 mg, mornings.*
- *Dermatomyositis, diagnosed 3 months ago, currently treated with 7.5 mg prednisone.*
- *The lab values were within reference range, apart from anemia with an Hb of 9.2 mg/dl (reference 12–14 mg/dl) and a hypokalemia of 3.4 mEq/l (reference 3.5–5.0 mEq/l). ECG was unremarkable. Ms. Pine had donated two bags of RBCs for this operation. This explained the anemia.*
- *Premedication with 10 mg midazolam PO.*
- *There were no additional orders noted for the preoperative holding area.*

22.1.2 What Was Not Noted in the Preoperative Evaluation?

22.1.2.1 Diabetes Mellitus

The patient has a non-insulin-dependent diabetes mellitus. As recommended, she did not take her oral diabetic medication on the day of the surgery. However, the blood glucose must be checked! A fasting level in the morning should be done, followed by further checks every 4 h.

>> *Knee joint replacements were done in this hospital under regional anesthesia, unless there is a contraindication. The standard technique consists of a combination of a psoas compartment block (PCB) and a continuous sciatic nerve block (posterior approach). Dr. Sofia began with the preparations. She positioned Ms. Pine on her side, decided upon the needle insertion point, and prepared the skin and draped the area. "Please call attending anesthesiologist Dr. Eldridge now and tell him we are ready to begin," she said to anesthesia tech Julie. Dr. Sofia had already done a few of these blocks, but did not feel confident enough to attempt it alone.*

Dr. Eldridge always had a lot to do. Anesthesia tech Julie reported back, "We should begin with the sciatic catheter alone; he will come later for the psoas compartment block (PCB)." Before beginning, Ms. Pine received 100 µg fentanyl IV. Dr. Sofia visualized the nerve using an ultrasound device, and she used a nerve stimulator to better localize the nerve and quickly received the response she was looking for. The anesthesia tech injected 20 ml of mepivacaine 1%, and afterwards, Dr. Sofia inserted the catheter and secured it with a dressing. Suddenly Dr. Eldridge appeared, and the PCB was placed according to his instructions. Not surprisingly with this patient's body mass index (BMI), the lumbar plexus was not stimulated before the needle was introduced 15 cm deep. Ms. Pine received 10 ml of mepivacaine 1% and 20 ml ropivacaine 0.5% for the block. Finally, she was repositioned supine, and after some time, the success of the nerve block was tested. Since motor control of the adductors was active, Dr. Sofia blocked the obturator nerve with 10 ml of ropivacaine 0.5%. After a repeat of the testing, movement was no longer possible. Dr. Sofia was proud of herself and felt awesome! Anesthesia tech Julie stayed with Ms. Pine, as Dr. Sofia returned to the OR to care for Ms. Kelly.

Ms. Kelly's hip surgery finished without complication. Ms. Pine was then rolled into the OR. As the orthopedic surgeon positioned the leg and affixed a tourniquet with 300 mmHg, Ms. Pine complained that she could feel something. "That's totally normal," Dr. Sofia reassured her. "What's important is that you don't feel any pain." Just to be safe, she called her attending. "Sedate the patient so that she does not consciously realize what is happening" was Dr. Eldridge's recommendation. Ms. Pine received 100 µg fentanyl and a propofol bolus of 40 mg, followed by a continuous infusion of 20 µg/kg/min. She slept through the beginning of the procedure; 45 min had passed since placing the regional anesthetic and the block seemed to have been a success!

The surgery had been in progress for 15 min when Ms. Pine woke up. She moaned and threw her head from side to side to free herself from the oxygen mask. "Maybe she has pain?" thought Dr. Sofia and gave another 100 µg fentanyl and 50 mg propofol. Ms. Pine slept again. Shortly thereafter, she woke up again – more agitated than before! "What is wrong? Are you in pain?" asked Dr. Sofia. Ms. Pine said something that Dr. Sofia couldn't make out. The situation was very strange. She called her attending again and asked him to come to the OR.

22.1.3 What's on Your Differential Diagnosis List? What Would You Do Now?

Acute perioperative cognitive disorders are discussed in Case 18 (see Sects. 18.1.2 and 18.1.7). The following overview lists possible differentials:

Differential diagnosis of the acute perioperative cerebral dysfunction includes:

- Decreased cerebral perfusion/low cerebral dissolved oxygen concentration
- Neurological diseases like dementia
- Electrolyte or metabolic disorders
- Toxic drug reactions
- Medication side effects (also paradoxical reactions)

In acute situations, it is important to rule out or confirm a diagnosis as soon as possible. Recommended first actions include:

- Discontinuing the propofol infusion, followed by
- A general neurological check
- An arterial blood gas including electrolytes and glucose

>> *Upon arriving in the OR, Dr. Eldridge, the attending anesthesiologist, shut off the propofol at once. Ms. Pine could not or would not give him an understandable answer to any of his questions. The monitor revealed a blood pressure of 160/90 mmHg, pulse 63 beats/min, and S_PO_2 of 92%. The ECG showed isolated supraventricular and ventricular extra systoles.*

"*Draw a blood gas*" *he said, turning to Dr. Sofia. Shortly afterwards, anesthesia tech Julie gave them the results. The only abnormality was anemia with a hemoglobin of 9.9 g/dl (reference 12–14 g/dl). Blood glucose and electrolytes were normal. Dr. Eldridge turned to the resident,* "*What is your working hypothesis*?"

22.1.4 What Is Your Diagnosis?

Ms. Pine displayed signs of a mental status disorder and an arrhythmia. Even if the alteration in mental status can be explained by the analgesia and sedation, local anesthetic toxicity should also be considered. Sixty minutes have passed since placement of the block so that absorption could have now caused an elevated plasma level [13]. Ms. Pine received a total of 150 mg ropivacaine and 300 mg mepivacaine for the sciatic, obturator, and lumbar block. She weighs a little over 100 kg. Even when the dose of each individual anesthetic is calculated to be well within a safe dose, it is not known whether or not a combination of individual local anesthetics can competitively bind plasma protein and cause toxic effects. In addition, Ms. Pine is anemic, so one must assume that there are higher levels of unbound plasma anesthetics.

22.1.5 How Would You Now Treat Ms. Pine?

Local anesthetic toxicity is very difficult to treat; therefore, prevention assumes an even more important role. *Prevention* includes the following:

- Administration of the smallest amount possible.
- Intermittent aspiration during slow injection.
- Addition of vasoconstrictors, such as epinephrine.
- Ultrasound guidance can reduce the rate of intravascular injection (however, it was used for the block in this case).

In the presented case, it is too late for most of these measures. The therapeutic options now are:

Assure sufficient oxygenation
Avoid hypoventilation

Compromised oxygenation increases the chance of neuro- and cardiotoxic effects. Hypoventilation leads to respiratory acidosis, thereby increasing the plasma level of the unbound anesthetic. Many authors therefore recommend deliberate hyperventilation. The hyperventilation, via cerebral vasoconstriction, theoretically also causes attenuation of the central neurotoxic effects. However, pronounced hyperventilation can also simulate central nervous system toxicity [14].

Increase the seizure threshold

A local anesthetic-induced seizure can cause increased toxicity due to hypoxemia and

hypercapnia. Apart from the abovementioned hyperventilation, an anticonvulsive medication should be considered, such as a benzodiazepine in order to prevent a seizure.

Administer a lipid infusion

In some case examples and experiments, lipid infusions have been successful in alleviating neuro- and cardiotoxic symptoms [8, 12, 16]. A good overview of the therapeutic approach and of up-to-date knowledge can be found at www.lipidrescue.org. There, an initial bolus of 1.5 ml/kg/body weight of a 20 % lipid solution is recommended, followed by a continuous infusion of 25 ml/kg/min for 30–60 min. In persistent asystole, the bolus can be repeated 1–2 times. The mechanism of action is not completely known, but lipid therapy has very few side effects, so the application is justified [5]. Most ORs now have LipidRescue Carts for emergencies, and it is now becoming standard of care.

>> Dr. Sofia didn't know the correct answer to Dr. Eldridge's inquiry, but he didn't expect her to know it. He gave Ms. Pine 2 mg midazolam IV and called the intensive care unit to request a lipid infusion. After administration of 150 ml 20% lipid infusion, the arrhythmia was gone. The benzodiazepine worked well, and Ms. Pine slept soundly. Her breathing was better, despite the sedation, and the S_PO_2 was 97%. The suspected diagnosis of attending anesthesiologist Dr. Eldridge, as always, was right on!

22.1.6 What Are the Causes of Local Anesthetic Toxicity, and What Are the Clinical Signs?

Local anesthetic toxicity is the result of an overdose, an accidental intravascular injection or especially rapid absorption at the site of injection. The local anesthetic is distributed by the bloodstream to other areas where it can block ion channels, preferentially sodium channels – especially sodium channels of nerves and the heart. Clinically, signs appear as central nervous system disorders or cardiac toxicity.

22.1.6.1 Signs of Central Nervous System Toxicity

The clinical signs of central nervous system toxicity appear as the plasma concentration slowly increases, often in a typical order:

- Restlessness, uneasiness
- Muscle tremor
- Sensory disorders
- Generalized seizures
- Coma and respiratory arrest

22.1.6.2 Signs of Cardiac Toxicity

Cardiac toxicity signs most often appear with higher plasma concentrations than symptoms of the central nervous system. Exceptions seem to be the rule, especially when there is a rapid increase in the plasma concentration in accidental intravascular injection.

The effects on the cardiovascular system are complex and span from a negative inotropic effect and a reduction in the speed of conduction to an increase in abnormal automaticity and vasodilation. Accordingly, the clinical picture varies, from presenting as a rhythm disorder with hypotension to a cardiac arrest.

The toxicity increases with the potency of the local anesthetic. Furthermore, the danger of toxic effects directly correlates with the plasma concentration and speed of its increase. Early symptoms may not be exhibited, and the first symptom may be a generalized seizure or cardiac arrest.

>> Dr. Sofia was relieved that her attending Dr. Eldridge had so quickly alleviated the crisis. "Wow, everything that can happen did!" she thought to herself, "I would have never thought about local anesthetic toxicity. Quite some time had passed since I placed the blocks – that was a long symptom-free interval." Dr. Eldridge asked her if she needed anything else before leaving the OR. Anesthesia tech Julie was called into another OR and left her telephone number as she said goodbye.

Dr. Sofia was in the process of documenting the procedures and interventions on the computer as the monitor alarm sounded. "An elevated systolic blood pressure" she said to herself, "170/90 mmHg. That's strange." Ms. Pine

rested peacefully on the table. Dr. Sofia cancelled the alarm and continued her documentation. After 3 min, the alarm sounded again, and Dr. Sofia measured blood pressure again just to confirm. Sure enough, it was 185/92 mmHg. The heart rate was 92 beats/min and the S_PO_2 was 97%. "Maybe she's in pain," thought Dr. Sofia. "The oxygen saturation is good. I should give her another bolus of fentanyl." She injected another 100 μg, but the desired effect did not appear. The only calming effect was that the opioid did not have an adverse impact on Ms. Pine's breathing.

Dr. Sofia thought about it: "*Pain would be the most probable cause. The local anesthetic toxicity has been successfully treated and therefore is not a possible cause. Ms. Pine is sleeping soundly and does not appear to be stressed. Probably it is simply hypertension.*" *With this diagnosis, she thought to herself*, "*I'll try labetalol.*"

22.1.7 Do You Agree with Your Colleague's Conclusions? What Possible Causes Did She Forget?

22.1.7.1 Local Anesthetic Toxicity

The local anesthetic toxicity is not yet treated; only the possible effects have been attenuated or prevented. Metabolism of the amide occurs in the liver and is dependent on plasma protein binding and hepatic perfusion. Even so, in this case it is unlikely that the local anesthetic is responsible for the increase in blood pressure.

22.1.7.2 Stress

Even a sedated patient can be in a stressful situation, such as during a paradoxical reaction to a medication. Despite her incorrect reasoning, Dr. Sofia was correct not to deepen the sedation.

22.1.7.3 Systemic Hypertension

Ms. Pine did have a diagnosis of hypertension. The morning of the surgery, she took her medications as prescribed. The blood pressure was normal until now. An acute hypertensive crisis is very unlikely.

22.1.7.4 Pain

Ms. Pine received several sedatives. The fact that she is asleep does not rule out the possibility that she is in pain. The conclusion that pain wasn't an issue was incorrect. Specific questioning of the patient was unfortunately no longer possible.

22.1.7.5 Tourniquet Pain

Dr. Sofia didn't think of tourniquet pain as a cause for the hypertension. Tourniquet pain is a regularly seen phenomenon, especially when the tourniquet is placed on a leg. The pain appears after various intervals and is usually treated by deepening the anesthesia. The mechanical pressure leads to reversible nerve damage. Usually, myelinated A delta-fibers are affected so that inhibition of the pain from the conducting non-myelinated C fibers diminishes [6]. The increased activity of the C fibers is correlated with an increase of the size of the affected area. The resulting circulatory changes are induced by a humeral response.

>> *Dr. Sofia was unhappy with herself. In the meantime, Ms. Pine had received another dose of labetalol IV, but the systolic blood pressure was still over 160 mmHg. "She has really got a resistant hypertension," she thought. "Later, I better let my attending know."*

In the meantime, the surgery was almost finished, and the orthopedic surgeon gave the signal to open the tourniquet, in order to control any bleeding before closing the wound. Dr. Sofia was tentatively documenting everything as the monitor alarm sounded again. "Somehow this is a very annoying blood pressure. I set the alarm levels extra high, and still…" she stopped in mid-thought, as she caught a glimpse of the screen: 65/35 mmHg.

22.1.8 What Are the Possible Causes of the Decrease in Blood Pressure?

22.1.8.1 Tourniquet Pain Relief

Removal of the pneumatic tourniquet leads to relief of the pain with a decrease in the humeral stimulation.

22.1.8.2 Pulmonary Embolism

One possibility is, after the tourniquet was deflated, a fat embolism or thrombus was released and caused a pulmonary embolism (see Chap. 11). With the help of a transesophageal echo, pulmonary embolism can be visualized in up to 70 % of patients who received a cemented knee arthroplasty with tourniquet hemostasis [4].

22.1.8.3 Volume Shift

The opening of the leg vessels led to a volume shift to the affected limb, as a result of reactive hyperemia and regional decrease of peripheral resistance. In the presented situation, the tourniquet was opened to perform surgical hemostasis. Even the loss of blood could be hemodynamically significant.

22.1.8.4 Release of Acidic Metabolites into the Systemic Circulation

The acidic metabolites formed during the ischemia and released after reperfusion lead to a short (15–30 min) change in pH. As a result, the pulmonary arterial pressure increases and the peripheral vascular resistance decreases. Both contribute to a decrease in blood pressure.

22.1.8.5 Deflating the Tourniquet Too Quickly

The negative effects of lung emboli, volume shift, and release of acidic metabolites are more pronounced when the tourniquet is opened too quickly. A slow release of the pressure over a period of at least 60 s is recommended.

>> After Ms. Pine received 500 µg of phenylephrine and 500 ml of a crystalloid infusion, the blood pressure improved, and Dr. Sofia relaxed a little. The surgical wound was sutured shut, an X-ray was done to check positioning, and Ms. Pine was brought to the PACU. The orthopedic surgeon had placed two drains in the knee and requested that these be opened after 15 min.

22.1.9 Why Wait 15 min?

Wound and tourniquet pain cause a surge in catecholamines which leads to an increase in platelet aggregation and to hypercoagulability. In conjunction with the reduced mobility, and the comorbidity of obesity, the patients have an increased risk of thrombosis. On the other hand, ischemia during tourniquet use leads to local activation of tissue plasminogen activators. The result is local hyperfibrinolysis. This lasts for about 30 min after removal of the tourniquet. This is the reason why a delayed opening of the wound drainage was advised.

>> Dr. Sofia transferred Ms. Pine to the PACU resident Dr. Cedric, a colleague who had been working in the PACU for the last 2 months. Ms. Pine had hemodynamic and respiratory stability at the time of handoff. The sedation had worn off. She looked a little clammy and complained of mild pain in the area of the sciatic nerve. The orthopedic surgeon checked motor function of the nerve and left the PACU relieved as Ms. Pine had lifted her foot. Dr. Sofia told the PACU resident about the suspected local anesthetic toxicity and blood pressure difficulties, before heading back in the OR.

Dr. Cedric ordered 1 g of acetaminophen IV, and the PACU nurse Carolyn prepared it. Dr. Cedric wanted to wait awhile before hooking up the sciatic catheter to a pump, just to be safe. As instructed, Dr. Cedric opened the vacuum drainage after 15 min. The collection bottle filled in no time at all and needed to be changed. Dr. Cedric was not alarmed "Happens all the time with knee replacements," he thought. "It hardly bleeds at all during the operation, only afterwards!" A check of some basic lab values showed a hematocrit of 25 % (reference 37–47 %), and Ms. Pine received the blood she had donated before the surgery. She slept through most everything.

One hour later, PACU nurse Carolyn called Dr. Sofia to see Ms. Pine. "Something about her is not right" she said. "She keeps getting sleepier and sleepier the longer she is here. Her blood pressure is pretty low, and she just vomited." As if Ms. Pine wanted to confirm the nurse's words, she vomited yet again, but she remained lying flat as if she were too exhausted to lift her head. "We'll do another blood gas, maybe she needs more blood" added Dr. Cedric. Then he called

the attending anesthesiologist Dr. Eldridge, who also thought the whole situation was very strange. As Dr. Eldridge entered the PACU, the blood gas was ready:

- *Hemoglobin*: *9.7 g/dl* (*reference 12–14 g/dl*)
- *Hematocrit*: *29%* (*reference 37–47%*)
- *pH*: *7.36* (*reference 7.35–7.45*)
- P_aO_2: *105 mmHg* (*reference 70–100 mmHg*)
- P_aCO_2: *40 mmHg* (*reference 36–44 mmHg*)
- HCO_3: *21.9 mEq/l* (*reference 22–26 mEq/l*)
- *BE*: *–1 mEq/l* (*reference ±2 mEq/l*)
- SO_2: *96%* (*reference 95–98%*)
- *Lactate*: *1.2 mmol/l* (*reference 0.5–2.2 mmol/l*)
- Na^+: *143 mEq/l* (*reference 135–150 mEq/l*)
- K^+: *3.2 mEq/l* (*reference 3.5–5.0 mEq/l*)
- *Glucose*: *52 mg/dl* (*reference 70–120 mg/dl*)

Dr. Eldridge listened again to the events and reviewed the anesthetic record and the patient's medical record on the computer. He then shook his head and at last spoke up: "*I think I know what the problem is*."

22.1.10 Do You Know Too?

Not just anyone can identify Ms. Pine's problem. The symptoms, which she displayed, are very unspecific – hypotension, nausea, vomiting, and decreased level of consciousness. The lab shows mild hypokalemia and hypoglycemia. The final clue was found in the patient's medical record. There he saw that Ms. Pine was recently treated with prednisone for dermatomyositis.

22.1.10.1 Dermatomyositis

Dermatomyositis is inflammation of the muscle with dermatological involvement. Clinically there is symmetrical, often painful muscle weakness with edema and erythema, and the typical butterfly rash on the face. Apart from that, vasculitis, perimyocarditis (leading to heart failure and arrhythmias), interstitial lung disease, alveolitis, and hepatic involvement may occur. Sometimes mobility disorders of the esophagus with dysphagia can also occur. The therapy of dermatomyositis is primarily highly dosed corticosteroids, which must be slowly reduced.

Ms. Pine had been in treatment with steroids for 3 months. Her current dose caused iatrogenic Cushing's syndrome, so secondary adrenal insufficiency must be assumed. Such patients need steroids perioperatively, although there is uncertainty as to how the regime should be dosed [10]. Figure 22.1 shows a possible treatment algorithm, which was created for the Clinic for Anesthesiology of the University Hospital in Dresden.

The anesthesiologist who performed Ms. Pine's preoperative evaluation, as well as Dr. Sofia, neglected to order perioperative glucocorticoids, and the patient developed an Addisonian crisis. An Addisonian crisis is caused by a rapid loss of adrenal gland function, as may occur with infarction or progression of chronic loss or through inadequate adrenal function in stressful situations – as in trauma or infection, with preexisting adrenal insufficiency. The symptoms of an Addisonian crisis are nonspecific.

22.1.10.2 Symptoms of an Addisonian crisis:

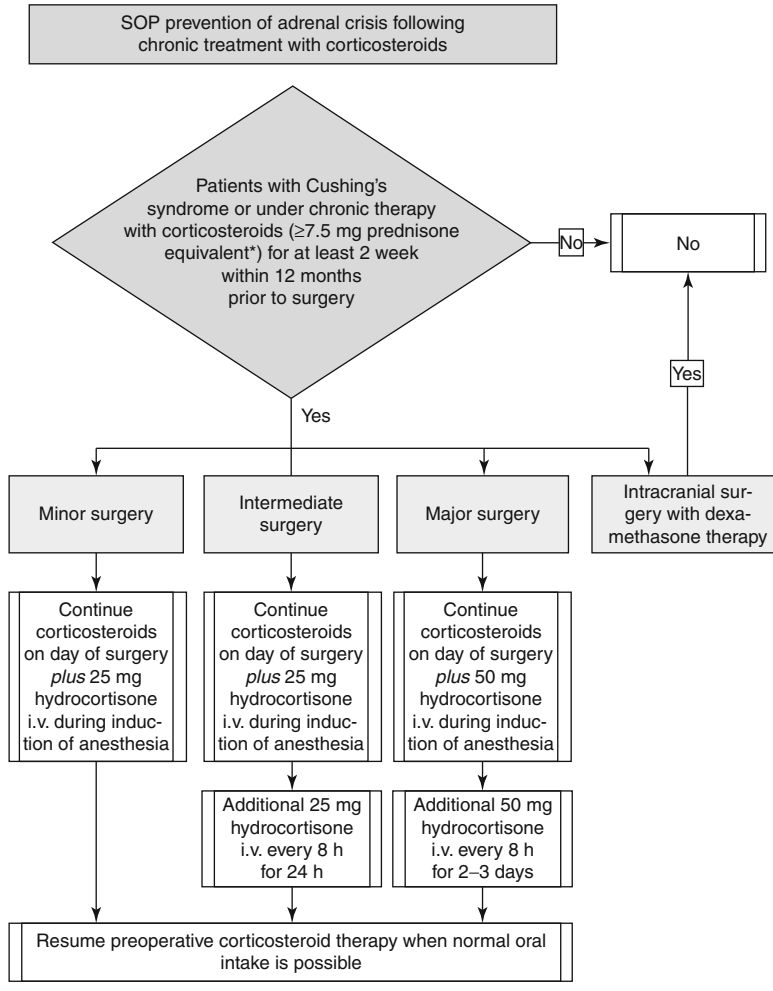
- Weakness
- Nausea and vomiting
- Hypotension
- Decreased vigilance
- Dehydration
- Fever
- Hypoglycemia

In secondary causes, such as with Ms. Pine, electrolyte disorders are usually not present.

Due to the decreased blood pressure regulation, severe shock can occur. The therapy consists of:

- Glucocorticoids
- Glucose infusion
- Fluids

>> *Following orders from attending anesthesiologist Dr. Eldridge, Ms. Pine received 100 mg hydrocortisone IV, 40 ml dextrose 40%, and an electrolyte infusion. She improved immediately and bombarded all the care takers around her with questions. Dr. Eldridge left the answers up to the PACU doc. He himself set off to find Dr. Sofia to discuss the case.*



Corticosteroids	Prednisone equivalent*
Betamethasone	1 mg
Dexamethasone	1 mg
Hydrocortisone	20 mg
Cortisone	25 mg

Corticosteroids	Prednisone equivalent*
Methylprednisolone	4 mg
Prednisolone	5 mg
Prednisol	5 mg
Fluticasone	0.5 mg

Fig. 22.1 Algorithm of the Department of Anesthesiology of the University Hospital Dresden for perioperative glucocorticoid substitution in patients with Cushing's syndrome or long-term corticosteroid therapy

22.2 Case Analysis/Debriefing

22.2.1 Can Tourniquet Pain Appear in Other Forms of Anesthesia? Can It Be Prevented?

Tourniquet pain can occur with all types of anesthesia [11]. In a retrospective analysis of 699 orthopedic patients, cardiovascular changes were experienced by 67 % of patients during general anesthesia, 18.6 % of patients with intravenous anesthesia, 2.7 % of patients with spinal anesthesia, and 2.5 % of patients with upper limb plexus anesthesia. The occurrence of tourniquet pain correlates with age of the patient, duration of the ischemia, type of procedure, and leg procedures [15].

Tourniquet pain cannot be entirely prevented. An important factor is a complete as possible

sensory block. Applying EMLA to the skin before the tourniquet sometimes helps. Many additives for IV regional anesthesia have been tested in small studies with partial success and include clonidine, morphine, melatonin, magnesium, and gabapentin.

However, therapy is often not successful. Opioids often do not achieve the desired effect. Ketamine or a nonsteroidal analgesic can be administered. It is important to limit the duration of the tourniquet because of the pain. Tourniquets are relatively contraindicated in sickle cell anemia patients due to the erythrocyte malformation.

22.2.2 Which Medical Errors Do You See in the Presented Case?

22.2.2.1 Hydrocortisone

This point was already discussed in Sect. 22.1.10.

22.2.2.2 Blood Glucose

In addition to not considering a glucose check preoperatively, (see Sect. 22.1.2) the resident physicians neglected to order a postoperative glucose check.

22.2.2.3 Steroid and Implantation of Foreign Material

Due to her long-term steroid therapy, Ms. Pine was at an increased risk for postoperative (wound) infections. The implantation of a knee replacement must be viewed critically.

22.2.2.4 Therapy of the Systemic Hypertension

Administration of antihypertensive medications shortly before the tourniquet was opened was incorrect. The ensuing blood pressure decrease was amplified and endangered the patient.

22.2.2.5 Alarm Pause

An undesired monitor alarm should be cancelled for a short interval, not completely turned off or reset (see Case 9, Sect. 9.1.6).

22.2.3 Which Systems Failures Can You Find in the Presented Case?

22.2.3.1 Operating Room Scheduling

Patients with diabetes should be highlighted on the OR schedule and ideally obtain an early slot. A preset arrangement is needed between the anesthesia and surgical teams.

22.2.3.2 Rescue Kit

Considering the limited therapeutic possibilities and the low reported risk of administrating IV lipids, a LipidRescue Kit (see also www.lipidrescue.org) should be present in the OR and wherever regional blocks are performed (including labor and delivery and the preoperative holding areas), in order to treat emergencies without delay.

22.2.3.3 Supervision and Training

Attending anesthesiologist Dr. Eldridge was the responsible anesthesiologist, and Dr. Sofia was not yet very experienced with caring for patients with knee replacement surgeries. Theoretical knowledge, such as the effects of tourniquets, must be acquired by self-study. A check of the resident's knowledge base by the attending anesthesiologist is essential.

22.2.4 On a Purely Rational Level, How Do People Make Quick Decisions?

As Ms. Pine's blood pressure increased, Dr. Sofia began treatment with pain medication. When this didn't help, she came to the conclusion that Ms. Pine's preexisting systemic hypertension was responsible, so Dr. Sofia administered antihypertensive medication. Dr. Sofia's attention was controlled by the characteristics of the situation which were recognizable to her, such as the causes of high blood pressure for which she had knowledge. She paid no attention to other causes, such as the tourniquet, and did not include them in her analysis of the cause of hypertension. For a timely appraisal of the situation, despite uncertain

information, Dr. Sofia used the rule of **representativeness heuristic**.

Representativeness heuristic is an oversimplification of decision-making. The advantage is that time-sensitive judgments can be quickly made about the general situation by concentrating on a few less representative characteristics.

In this case, it would mean that a hypertensive patient fresh out of surgery has pain. Very few characteristics, which correspond to the expected prototypic scheme, were perceived. With uncertainty and very few clues, the conclusion was drawn that this was again a "typical hypertension."

Representativeness heuristic is always dangerous when too few situational characteristics are considered in the diagnosis and clues that negate the accuracy of the judgment are ignored. Incorrect decisions are thereby fostered.

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Case 23: Laser Surgery

23

Angelika Eichner, Mike Hänsel, Karen B. Domino, and Matthias Hübler

Contents

23.1 Case Introduction

>> Lisa was a lively, friendly 3-year-old. She loved to go to her preschool. Even though she had been in a hospital before, she was still open and approachable by strangers in scrubs. Four months ago, she developed hoarseness with dysphonia. An ENT physician in another hospital diagnosed a papilloma and removed it under general anesthesia. Everything went well. In order to rule out a recurrent laryngeal papilloma, Lisa received regular ENT check up visits.

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Unfortunately, the hoarseness and mild inspiratory stridor had returned again, and the parents now brought Lisa to the university hospital. Here it was decided that the new papilloma must be removed. Apart from that, Lisa was a happy, healthy child, growing very well. The parents had noted on the informed consent form that Lisa had slept for a very long time after her last anesthetic. In addition, she weighed 14 kg, had no other medical disorders, and no respiratory infection. For premedication, Lisa received a 7 mg midazolam suppository and entered the OR sound asleep.

23.1.1 What Is Recurrent Laryngeal Papillomatosis?

Recurrent laryngeal papillomatosis, also known as recurrent respiratory papillomatosis (RRP), is a chronic viral infection of the respiratory tract and affects children and adults. It is caused by the human papillomavirus (HPV) types 6 and 8 and is the most common neoplasm of the larynx in children [1]. The incidence is about 4/100,000 in children and 2/100,000 in adults. The symptoms usually begin as hoarseness, inspiratory biphasic stridor, chronic cough, dyspnea, dysphagia, recurrent pneumonias, and even respiratory failure.

Interestingly, HPV 6 and 11 cause 90 % of genital condylomata, so vertical transmission during vaginal childbirth is likely. Other HPV types, such as HPV 16, 18, 31, and 33, are associated with cervical, vaginal, vulvar, and oropharyngeal cancer. Although vaccination to prevent RRP is recommended in Germany, in the USA, the Centers for Disease Control only recommends vaccination of preteens and teens to prevent cervical, genital, and oropharyngeal cancers, not infants or pregnant women to prevent RRP (www.cdc.gov/vaccines/vpd-vac/hpv). When children with RRP must undergo operative intervention more often than four times a year, adjuvant pharmacological therapy is recommended.

There is no cure. The current therapy consists of surgical "no touch" removal of the papilloma, taking care to preserve the anatomical structures, especially the airway.

>> Resident Dr. Andrew read through the file, as anesthesia technician Steve skillfully placed an IV on the EMLA-numbed skin of Lisa's right hand.

23.1.2 What Are the Apparent Problems to Watch Out for, and What Form of Anesthesia Would You Choose?

Typically, the main problems of ENT procedures stem from the intersecting focus of the anesthesiologist and the ENT surgeon, which is the airway. The spatial proximity and narrow opening require good communication and teamwork between the two disciplines concerned about this small space, and knowledge and understanding of the planned procedures and the sequence of events, all the while keeping in mind that there is a danger of losing the secured airway. Before surgery commences, the planned procedures must be agreed upon between the ENT and anesthesiology physicians.

In the presented case, general anesthesia with TIVA is indicated.

>> Since Dr. Andrew was on call this week, he wasn't in ENT the day before. He was in his last year of residency and had already done several anesthetics for ENT. He called his attending anesthesiologist Dr. Eldridge to ask what was planned. Over the phone, Dr. Eldridge explained that surgical removal of a papilloma was planned, possibly with a laser. "The decision will be made intraoperatively," said Dr. Eldridge. "Last time they didn't use a laser." After the phone call ended, an unfamiliar young woman presented herself to Dr. Andrew. "May I introduce myself? My name is Imogen Smith. Please call me Imo. I am a med student doing a rotation here." Dr. Andrew thought to himself, "On top of everything else, some kid wants to follow me around?" With all the friendliness he could muster, he said, "Nice to have you here, Imo! Please let me know if you have any questions!"

Table 23.1 Medical lasers

Laser type	Wavelength [nm]		Absorption according to [type of tissue]	Application area
CO_2	10.600	Invisible – infrared	Various tissues	General
			Water	Precise incision
Nd:YAG (neodymium yttrium aluminum garnet)	1,064	Invisible – infrared dark	Pigmented tissue	Coagulation (via fiberoptic)
				Tumor debulking
Nd:YAG-KTP (Nd:YAG potassium titanyl phosphate)	532	Visible – emerald green	Blood	General
				Pigmented lesions
Argon	488–514	Visible – blue green	Melanin	Vessel
			Hemoglobin	Pigmented lesion
Krypton	400–700	Visible – blue red	Melanin	General
				Pigmented lesion

23.1.3 What Is a Laser, and What Types of Lasers Are You Familiar With?

LASER stands for **l**ight **a**mplification by **s**timulated **e**mission of **r**adiation. Atoms, ions, or molecules can be excited at a certain energy level and produce energy in the form of light which, via amplification and bundling, is sent out as a laser beam. The longer the wave, the more energy is absorbed in the superficial layers as heat. The shorter the waves, the deeper the laser beams infiltrate the tissue before absorption.

The CO_2 laser has been in use since the mid-1970s. Due to its long waves of 10,600 nm, the coherent light is nearly completely absorbed in the tissue surface and converted to thermal energy. The thermal energy is absorbed by intracellular water and leads to vaporization of the cell. The various types of lasers and their areas of use are listed in Table 23.1.

23.1.4 What Are the Specific Dangers of Lasers?

The risks of laser surgery are the following:

- Injuries to surrounding structures caused by reflected or indirect lasers.
- In oxygen-rich areas, the heat can cause burns, fire, or explosions.
- There is evidence of active viral DNA in the laser fumes, which could potentially infect health-care personnel.

A new technique without thermal injury is the endoscopic microdebrider. A survey of ENT surgeons expressed consensus that this technique replaces "shaving with the CO_2 laser" [5]. The cold microdebrider removal is more often used in adult patients.

23.1.5 What Are the Three Requirements for Creation of a Fire in the OR?

Fire needs three things: flammable material, ignition, and oxidizing agent [3]. Flammable material could be tubes, sutures, topical sprays, dry tamponades, tonsil tissue, dry connective tissue, muscle, and fat.

Flammable anesthetics such as ether and cyclopropane are no longer in use. Source of ignition could be electric cautery or laser. Oxidizing agents include oxygen (O_2) and nitrous oxide (N_2O).

>> Dr. Andrew considered which tube would be best to use. Assuming there would be no laser, he decided on the Magill tube. "What is this tube made of?" asked Imo, bubbling over with curiosity.

23.1.6 Do You Know?

Endotracheal tubes are either made of polyvinyl chloride (PVC), silicon, rubber, or metal. These days, most tubes are PVC, which are free of latex.

Table 23.2 Oxygen index of flammability (OI) and N_2O index for endotracheal tubes

Material	(Shown in Fig. 23.1)	OI (%)	N_2O index (%)
Polyvinyl chloride (PVC)	(Solid line)	26.3	45.6
Silicon	(Dashed line)	18.9	41.4
Rubber	(Dotted line)	17.6	37.4

>> Dr. Andrew felt trapped by his own ignorance and sneaked a peak at the package, "It's made of PVC." Ever-energetic Imo took another from the drawer: "This one is silicon. Which is better suited for laser surgery?" she asked. While Dr. Andrew was silently cursing the question, anesthesia tech Steve came to the rescue and interrupted the two. "Can we get to work? The coffee I just made will be gone without me if I don't make it to the break room soon!"

23.1.7 What Determines the Flammability of a Tube?

All endotracheal tubes, regardless of their composition, can catch fire if burned by a laser. The flammability of a material is described by its limiting oxygen index (LOI), critical oxygen index (COI), or oxygen index of flammability (OI). To determine the OI, various mixtures of oxygen and nitrogen are placed in the tube and then lit with a hydrogen flame. The oxygen concentration is increased, until the burning continues after removal of the flame. The OI describes the minimal concentration of O_2 in an O_2/N_2 mixture, which is still capable of supporting the flame. High OI values indicate high flammability protection/low flammability. The formula is

$$OI = O_2 / (O_2 + N_2) \qquad (23.1)$$

and is given in percent. Since nitrous oxide is also an oxidizing agent, there is a N_2O index on every tube.

Air contains 20.95 % oxygen. Every material which has an OI value <20.95 % is ignitable in room air. A material with an OI value of >20.95 % would burn poorly in room air. The value can approach zero, after the igniting flame is removed [4, 6].

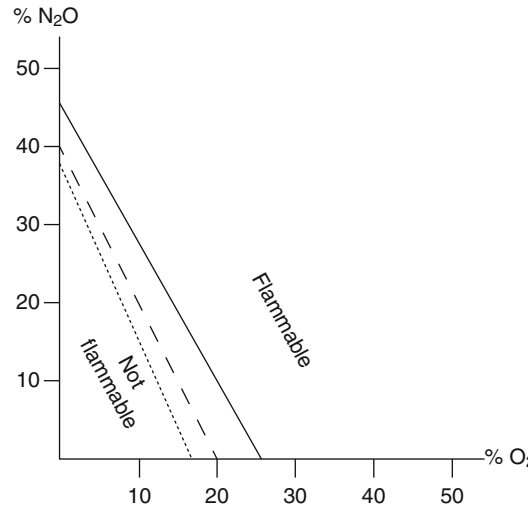

Fig. 23.1 Flammability of endotracheal tubes. Presented are OI values of rubber (*dotted line*), silicon (*dashed line*), and PVC (*solid line*) in mixture of nitrous oxide and O_2. Mixtures which are above and to the right of the straight line are flammable. The graph illustrates the use of a mixture of nitrous oxide and O_2 in ENT surgery is not safe and no longer used since either ignition is greatly increased or it is necessary to work with a hypoxic gas mixture (From Wolf and Simpson [6] with permission)

Table 23.2 shows the OI value of various endotracheal tubes, which are discussed in Fig. 23.1 (according to [6]).

>> Dr. Andrew gave Lisa 75 µg fentanyl IV. She participated wonderfully in preoxygenation with the "astronaut mask," and after 100 mg of propofol, she was fast asleep. "The mask ventilation is going well" said Dr. Andrew to anesthesia tech Steve. "Give her 4.5 mg mivacurium." During the laryngoscopy, Dr. Andrew had a full view of the vocal cords. The papilloma was on the right.

The size 5 unblocked tube slid safely through the glottis. Afterwards, remifentanil at 0.3 µg/kg/min and propofol at 150 mcg/kg/min were started. Together with the ENT surgeons, Dr. Andrew positioned Lisa. Vital signs were unremarkable; however, after hyperextending the neck, a small leak appeared.

23.1.8 Evaluate the Use of a Cuffed Compared to a Noncuffed Endotracheal Tube!

Repeated tracheal intubation and extubation increases the chance of bringing infectious material into the trachea and increases the danger of causing mucosal irritation. The ENT procedure in and of itself has the potential to cause mucosal edema, so additional intubation injuries should be avoided. An inflatable cuff can compensate for a tube that is too small and help to avoid changing the tube. Also, cuffed tubes help to prevent fresh gas leaks (e.g., leaks which arise by positioning the head) and an unmeasurable amount of O_2 and nitrous oxide leaked into the surgical area. Cuffed tubes can also prevent pieces of removed tissue from being carried into the trachea.

>> "*Should we reinsert a larger tube*?" *asked anesthesia tech Steve. Before Dr. Andrew could answer, the ENT surgeon said* "*I'll put some sponges around the entry to the larynx. That should seal the leak*." *And then the surgery began. Dr. Andrew filled out his anesthesia record, and the ENT surgeon operated in silence. After a while, he glanced up from his surgery.* "*I think I'm going to have to use the laser. The papilloma is just too extensive to handle without it*."

23.1.9 What Options Does Dr. Andrew Have Now?

- **Extubation and reintubation with a laser-compatible tube**

Laser-safe tubes are cuffed and made of metal or metal-covered plastic (PVC, silicon, rubber). They are available starting at size 4.0. The non-laser-safe cuff is filled with methylene blue and is blocked with NaCl instead of air. Therefore, an accidental cuff tear allows immediate visual identification. The NaCl solution absorbs thermal energy and simultaneously extinguishes any possible ignition. Laser-safe tubes are a bit more expensive.

- **Intermittent extubation/intubation** of the PVC tube currently in use, with lasering during the apnea phases.
- **Jet ventilation** (supra-/subglottic).
- **Extubation and lasering during spontaneous breathing**.

In pediatric otolaryngology, there has been an interesting trend during the past 15 years in operative and anesthesia techniques. In the 1990s, a CO_2 laser was used in 90 % of the surgeries and a laser-resistant endotracheal tube in 50 %. Today, the microdebrider has taken the place of the CO_2 laser; the anesthesia techniques changed with the surgical variations. Two-thirds of papilloma surgeries are done with spontaneous breathing or periodic ventilation. Controlled ventilation occurs in ¼ of the cases via jet ventilation, and a laser-resistant endotracheal tube is used in 10 % of procedures [5]. The choice of method to use depends on:

- Localization and accessibility of the tumor
- Extent and duration of the lasering
- Size and age of the child
- Cooperation and experience of the anesthesiologists and ENT colleagues
- Technical capabilities

>> "*Steve, please get me one of those laser tubes. We need to reintubate*" *said Dr. Andrew*. "*Are you sure about that*?"*asked tech Steve*. "*I only have one more 5.0 tube, as I try not to order them until I have to because they are so expensive*." *The ENT surgeon suggested that he could work during apnea, with intermittent extubation and intubation with the PVC tube*. "*No, I don't feel comfortable with that*," *said Dr. Andrew. Lisa received 1.5 mg mivacurium and a propofol bolus of 70 mg IV. The exchange of endotracheal tubes went without incident*.

In the OR, special mouth and eye protections were distributed for the use of the laser, and the warning light above the outside door was turned on. Dr. Andrew set the inspiratory oxygen concentration (F_iO_2) to 30 %, and the ENT surgeon positioned Lisa's head. Then he inserted the metal tube and fixed it. Dr. Andrew noticed that the S_PO_2 slowly dropped from 99 to 91 %. All other parameters remained within reference range.

23.1.10 What Are the Pediatric Differential Diagnoses for a Slow Decrease in Oxygen Saturation?

See also Case 15, Sect. 15.2.1.

23.1.10.1 Ventilatory Causes

These causes include tube dislocation, one-sided intubation, bronchospasm, atelectasis, or hypoventilation.

23.1.10.2 Cardiovascular Causes

For example, hypotension or hypovolemia can influence saturation, as well as pulmonary perfusion, and perfusion of the extremity where the measurement is being taken.

23.1.10.3 Artifact

Basically, clarification of a S_pO_2 decrease must begin with a check of the signal quality, in order to rule out an artifact.

23.1.10.4 Technical/Mechanical Malfunctions

Mechanical malfunctions are infrequent, but must also be excluded. Included in this category are problems with the ventilator, tube system, or gas mixture.

>> Dr. Andrew auscultated the lungs but could not detect any abnormal findings. The S_PO_2 decreased to 90%, so he increased the $F_iO_2$to 40%. When the ENT surgeon began with the lasering, the S_PO_2 increased to 91%. "Why is the saturation so poor?" asked the curious Imo. "In room air, she had a saturation of 99%." Before Dr. Andrew could answer, he was interrupted by the ENT surgeon, "Can I have some peace and quiet in here? Could you continue your conversation later, and shut off the alarm now? Or have you got a problem?"

Dr. Andrew denied any problems, shut off the alarm, and increased the F_iO_2 to 45%. The S_pO_2 increased to 92%. Dr. Andrew injected more mivacurium into Lisa's IV. Imo was confused by that and asked "What does muscle relaxation have to do with saturation?" Dr. Andrew put a finger to his lips and whispered, "Later."

23.1.11 You Can Answer Imo's Question, Can't You?

Muscle relaxation has no influence on the S_PO_2, unless artifacts from shivering are present. However, it influences the oxygen requirement since a relaxed muscle has a lower rate of metabolism. The relaxation in the presented case is necessary, in order to prevent sudden movement, thereby minimizing the danger of laser injury to other structures.

>> Dr. Andrew filled out the anesthetic record, and the OR became quiet. After 15 min, the S_PO_2 had improved (100%), and Dr. Andrew reduced the F_iO_2 to 35%, without a change in the saturation. Simultaneously, he set a PEEP of 3 mmHg on the ventilator.

23.1.12 What Was the Most Probable Cause of the Decrease in Saturation?

In the supine position, the intestines push the diaphragm cranially. Atelectasis may form in the dorso-basal lung segments, which causes a decrease in saturation. The loss of PEEP during the reintubation and the insufficient ventilation increase the formation of atelectasis.

23.1.13 Which Physiological Compensatory Mechanisms Do You Know Of?

Hypoxic pulmonary vasoconstriction (HPV) describes a paradoxical physiological compensation mechanism to improve the ventilation and perfusion ratio when regional ventilation (oxygen) deficits are present. With unchanged perfusion, a right left shunt develops in this area and ensuing hypoxia. Through direct (hypoxia) and indirect (catecholaminergic) stimulation, pulmo-

nary vasoconstriction results in the areas with a poor ventilation–perfusion ratio. HPV reaches maximum after 15 min.

>> After a few minutes of working with the laser, the ENT surgeon informed Dr. Andrew that he was almost finished. "I only lasered three lesions; I don't think there's a risk of swelling." Dr. Andrew saw his afternoon break within reach. He asked anesthesia tech Steve to give 1 mg morphine IV so that Lisa would not wake up in pain. He turned off the remifentanil and propofol infusion pumps and increased the FiO_2 to 100%. Suddenly the ENT physician yelled "Dang! I hit the cuff!" and the leak was clearly heard. The ventilator sounded the alarms for pressure and volume. "Should I?" asked the ENT surgeon. "What? Do what?" responded Dr. Andrew, visibly shocked.

Tech Steve quietly asked Imo to go get Dr. Eldridge immediately. Dr. Andrew glanced around confused, shutting off alarms and increasing fresh gas flow. The ENT surgeon interrupted again – this time with a much harsher tone of voice. "Should I remove the tube? I am finished with the surgery." Dr. Andrew disagreed and was aghast and unable to think. At this moment, Dr. Eldridge entered the room. He had caught the last sentence. "Yes, of course, please remove the tube," he said calmly.

23.1.14 Why Must the Tube Be Removed?

The tube was accidentally hit by a laser and damaged. Due to the high F_iO_2, there is no assurance that the tube is not smoldering. The algorithm for (suspected) tube fire is shown below:

>> Algorithm in (suspected) endotracheal tube fire

1. Abort ventilation.
2. Cease oxygen administration, remove tube, and flood surgical area with NaCl.
3. Perform non-flexible laryngoscopy and bronchoscopy, in order to assess the extent of injury and remove debris.
4. Monitor for 24 h.
5. Administer short-acting corticosteroid therapy.

Easier to memorize is the **4 E's rule**.

4 E's rule should direct actions in a (suspected) tube fire

1. **Extract** (flammable material)
2. **Eliminate** (oxygen administration)
3. **Extinguish** (burning material with NaCl)
4. **Evaluate** (extent of injury)

The tube appeared undamaged, apart from the blown cuff. The ENT surgeon inspected the larynx, "I don't see any injuries here." Attending anesthesiologist Dr. Eldridge nodded, apparently satisfied. "Finish the anesthesia with a face mask," he instructed Dr. Andrew, as he left the OR. The resident felt like he had just been jumped on, but he carefully ventilated by hand. It went easily. All in all, the procedure had taken less than 30 min. Slowly, Dr. Andrew eased up on his ventilation so that Lisa could begin to breathe for herself again. Fifteen minutes later she was ready: her spontaneous breathing was sufficient, although she took shallow breaths and had a high respiratory rate.

After another 5 min, she became restless with unpredictable jerky movements, and her closed eyelids twitched. The breathing pattern remained the same.

23.1.15 What Is Your Suspected Diagnosis? What Is the Cause? What Is the Therapy?

The clinical symptoms described indicate a postoperative residual neuromuscular blockade. Mivacurium is a benzylisoquinoline muscle relaxant with duration of action of 15–25 min. As succinylcholine, it is metabolized quickly in plasma via nonspecific pseudocholinesterase (pseudo-ChE) and in very small amounts by cholinesterase. The duration of action is affected by two variations in pseudo-ChE:

- Quantitative pseudo-ChE deficiency: A quantitative pseudo-ChE deficiency becomes clinically apparent only if the enzyme levels are very low.

- Qualitative pseudo-ChE deficiency: Genetic variation results in decreased enzyme activity [2].

The diagnosis of qualitative pseudo-ChE deficiency is made using the in vitro dibucaine test. Dibucaine is a local anesthetic, which inhibits about 80 % of the activity of genetically normal pseudo-ChE (dibucaine number = 80). Atypical pseudo-ChE in heterozygous patients has a dibucaine number of 40–60 and homozygotes of 20. Reduced quantitative activity is observed in newborns, pregnant women, patients with neoplasms, liver or kidney disease, burns, collagenases, hypothyroidism, and in conjunction with certain medications (cyclophosphamide, bambuterol).

Prolonged muscle relaxation after the administration of mivacurium can be treated with ChE inhibitors, even in the presence of atypical pseudo-ChE disorders. The ChE inhibitor therapy does not only inhibit acetyl cholinesterase in the synaptic space but also the plasma cholinesterase – an effect which is especially pronounced after administration of neostigmine. It is therefore safer to continue ventilation of the patients. Administration of human plasma ChE concentrate or plasma itself is no longer recommended.

>> *Dr. Andrew had a bad feeling about what was happening. Just a few days ago, he had a similar case. He had extubated, because he thought the tube had some sort of resistance which was causing breathing problems. The patient then had a seizure and looked like a ladybug on its back fighting to right itself. The patient was cyanotic and panicked and fighting for breath without success. Dr. Andrew had assisted with a face mask and finally antagonized the muscle relaxant. It was a very bad memory for everyone involved. "Can't happen again. This time I used a short-acting muscle relaxant," he thought. Despite this, he asked tech Steve to administer 0.15 mg glycopyrrolate and 1.5 mg pyridostigmine to little Lisa. The clinical situation improved promptly, and after a few minutes, the child was breathing again peacefully and regularly. Upon arrival in the PACU, Lisa was already smiling and asking for her mommy.*

23.2 Case Analysis/Debriefing

23.2.1 Which Medical Errors Do You See in the Presented Case?

23.2.1.1 Preoperative Evaluation

On the informed consent sheet, the parents noted that Lisa had "slept very, very long" after the last anesthetic. It is possible that mivacurium was given as a muscle relaxant. The anesthesiologist who performed the preoperative evaluation neglected to investigate further.

23.2.1.2 Choice of Airway

In supraglottic procedures, securing the airway with an endotracheal tube is a safe technique. Noncuffed tubes may be used in pediatrics. In ENT procedures, however, it is advisable to use small, cuffed tubes, in order to give the ENT surgeon a better view of the lesions, while safely securing the airway.

23.2.1.3 Intraoperative Ventilation

This aspect was already discussed in Sect. 23.1.12. A PEEP of 3–5 mmHg is recommended for atelectasis prophylaxis and replacement of the auto-PEEP. Dr. Andrew was unfamiliar with the physiology of the human papillomavirus (HPV). He reacted to a drop in saturation by dangerously increasing the F_iO_2.

23.2.1.4 Extubation

With the working diagnosis of postoperative residual neuromuscular blockade, Dr. Andrew antagonized mivacurium without the recommended neuromuscular monitoring. His reliance on the clinical criteria alone was severely flawed.

23.2.2 Which Systems Failures Can You Find in the Presented Case?

23.2.2.1 On-Call Week

In many clinics, on-call week means that a resident covers for vacant slots in the roster for a week. Changing working areas daily leads to

deficiencies in preparedness for the work of the day. This systems failure might not be avoidable, but the anesthesiologist-in-charge needs to offer intensive supervision to the rotating resident (an act of negligence can lead to organizational culpability).

23.2.2.2 Emergence from Anesthesia

Emergence from pediatric anesthesia, especially after procedures involving the larynx, generally requires the presence of an experienced anesthesiologist or specialist. The attending anesthesiologist usually will be present in the OR at all times in these sorts of cases.

23.2.2.3 Medical School Student

A medical school student who is assigned to a resident on a specialty rotation in the OR results in a bad training situation for both of them. Medical school students should only be assigned to routine cases with experienced residents and attendings who are available and interested in teaching them.

23.2.2.4 Communication Between Surgical and Anesthetic Teams

The ENT service missed a key ingredient – preoperative communication directly with the anesthesiologist about the planned procedure and the sequence of events, especially the possibility of laser use. Dr. Andrew had spoken on the phone with his attending Dr. Eldridge about the surgical plan, and he came away with the impression that laser would probably not be used. Dr. Andrew therefore chose a Magill tube. When laser was required later in the procedure, Dr. Andrew chose to extubate the patient and reintubate with a laser-safe tube. The necessary repeated intubation caused an added risk for the child. A preoperative checklist would force communication between the surgical and anesthetic teams and reduce the unnecessary risk to the patient.

23.2.2.5 Provisions

Laser-resistant tubes may be expensive, but that is no reason not to keep enough of them in stock. A cuff injury can occur anytime and require an exchange of the tube.

23.2.3 Do You Always Increase the Inspiratory Oxygen Concentration When S_pO_2 Decreases?

When Dr. Andrew realized that the child's S_pO_2 value was decreasing, he promptly increased the O_2. This exemplifies a typical algorithm which many anesthesiologists carry out by instinct, when oxygen saturation should be increased. Unfortunately, the risk of a tube fire was not considered. Why not?

Realization of critical parameters leads to the need to find a solution quickly. In the process, one will often reach for the learned or experienced maneuvers because emotion is dominating the situation. A purely affective–emotional-based decision-making process does not leave any space for analytic thought processes. In this case, the obvious physical correlation between fire and oxygen was lost in the mix.

Decisions made from a gut feeling can be correct decisions, and colleagues who often reach correct decisions enjoy prestige and are seen as especially competent. But emotions like fear, annoyance, and euphoria can suddenly appear and, if they have enough influence, can drastically impair rational handling of an emergency. Cases 7 and 16 (see Sects. 7.2.3 and 16.2.4) mention the step-back technique. This technique could have helped Dr. Andrew when he needed to act on the ENT surgeon's directive to remove the tube. The step-back technique could have been helpful in making decisions about further ventilation for Lisa.

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Case 24: Tibia Fracture

24

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24.1 Case Introduction

>> It was a summer that wouldn't soon be forgotten: beautiful, warm, long days, the whole country caught up in soccer fever, and almost everyone seemed to be on vacation. It was awesome for those colleagues who did get vacation time, but for those left behind in the hospital, it was very stressful indeed. The emergency room (ER) and admissions were exploding at the seams, the ORs were overflowing, and the surgical schedule was packed. One multiple trauma after another arrived in the ER; the blood bank had to call for emergency donations. Dr. Conall, a resident near the end of his training, savored the enormous workload in the ER as he was busy collecting procedures to fill up his procedure book.

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In the OR, however, he wasn't quite so excited. This was Dr. Conall's last year of training before taking the boards, and he had come to know every corner of the department very well. Today, he was in trauma surgery, with a long list of procedures for the day: ORIFs of fractures of various extremities, removal of metal devices, revision of external fixation, and a dorsal stabilization. There was no way they were going to get through that OR schedule today, regardless of how quickly he got the patients in and out.

After a short breakfast break at about 11:00 a.m., the next patient was Mr. Scott, a 21-year-old whom Dr. Conall had met a week earlier in the ER. Mr. Scott had been riding his motorcycle, most likely not within the speed limit, when he lost control in a curve and hit a tree. It was a serious accident; the bike and the man were totaled. It was amazing that the young man survived his multiple trauma. He was brought to the hospital with a cerebral contusion and a Glasgow Coma Scale (GCS) of 3, blunt chest trauma with bilateral chest instability and bilateral pneumothoraxes, blunt abdominal trauma with parenchymal injury, a third-degree open limb fracture with arterial vessel damage, hemorrhagic shock, and a suspected vertebral column injury. Upon arrival in the OR, Dr. Conall was part of the trauma team and had immediately placed the first chest tube. Now he was anxious to see the patient again and see how he was recovering.

24.1.1 What Is Multiple Trauma?

Multiple trauma is defined as simultaneous injury of multiple organ systems and regions; at least one injury, or a combination of several, must be life-threatening. The Injury Severity Score (ISS) in multiple trauma is by definition ≥16. The primary care of trauma patients follows Advanced Trauma Life Support (ATLS) guidelines.

Multiple trauma is differentiated from a trauma patient with multiple noncritical injuries and also from a patient with one life-threatening injury.

>> The following was noted on the preoperative evaluation form:

- *S/P anterior basal intracerebral hematoma, traumatic SAH (subarachnoid hemorrhage) multiple, cerebral contusions*
  Therapy: observation, supportive care, ICU
- *Awake, obeys simple commands, not orientated, spastic paresis of the upper limbs, vegetative state*
  Therapy: physical therapy, antiepileptics
- *S/P multiple rib fractures, right, with hemopneumothorax, bilateral pulmonary contusions, and acute lung injury*
  Therapy: S/P chest tube, respiratory therapy
- *S/P cardiac contusion*
  Therapy: conservative
- *Subcapsular liver hematoma*
  Therapy: conservative
- *Pelvic fracture*
  Therapy: conservative, therapeutic administration of low molecular weight heparin for deep venous thrombosis prophylaxis
- *S/P 3° open tibia comminuted fracture with injury of the right popliteal artery*
  Therapy: S/P suture, external fixation
- *Ulna and radius fracture, right*
  Therapy: conservative with cast
- *Cervical acceleration–deceleration*

Today, final ORIF of the right tibia was planned.

24.1.2 Which Form of Anesthesia Would You Choose?

24.1.2.1 Spinal/Epidural Anesthesia

A neuraxial blockade could not be performed because of the anticoagulation, traumatic SAH, lack of patient cooperation, and prolonged surgical duration.

24.1.2.2 Peripheral Regional Anesthesia

Individual or combined peripheral catheter regional anesthesia is possible but technically difficult due to positioning with a pelvis fracture, motor response with external fixation, and possible nerve injury. The lack of patient cooperation, the long duration of surgery, and the

tourniquet placement add to the difficulty. The possibility of an iliac crest bone graft must also be considered. Anticoagulation therapy, by itself, is not a contraindication.

24.1.2.3 General Anesthesia

Due to the multiple injuries, general anesthesia is the best option. It can be performed as total intravenous or balanced anesthesia. Nitrous oxide is contraindicated, due to the hemopneumothorax and severe traumatic brain injury.

>> As Dr. Conall read the medical record, anesthesia technician Cindy prepared everything for general anesthesia. A few minutes earlier, she had asked if an endotracheal tube or laryngeal mask airway would be used. Dr. Conall waved his hand without comment. Then a bright idea occurred to him: "We could use the laryngeal tube in order to protect the C-spine."

24.1.3 Which Advantages Do You See in Using a Laryngeal Mask Airway Compared to a Laryngeal Tube?

The laryngeal tube was developed in Germany and is used in Europe, but is rarely used in the USA. Both the laryngeal tube and laryngeal mask airway effectively maintain a patent airway. In addition to the classical one-lumen model, there are also double-lumen models in which the second lumen drains the esophagus (e.g., ProSeal™ LMA, laryngeal tube suction LTS). Placement of a laryngeal tube is done with the head in a neutral position and may be easier and quicker than placing a conventional laryngeal mask airway. Neither device offers definitive protection against aspiration of gastric contents.

An advantage of the laryngeal tube is that it forms a good seal compared to a conventional laryngeal mask airway. Relatively high ventilation pressures are tolerated before a leak occurs (30 mbar in laryngeal tube vs. 20 mbar a conventional laryngeal mask airway). However, the ProSeal™ LMA maintains similar seal pressures and is more effective than the laryngeal tube with ventilation [3]. In addition, it allows a better view of the larynx with a fiberoptic bronchoscope. Oropharyngeal pressure damage occurs in both techniques and is dependent on the duration of use [6].

>> Conscientiously, Dr. Conall noted the respiratory and hemodynamic values (HR 105 beats/min, blood pressure 140/85 mmHg, S_PO_2 96%), auscultated the lungs, and inspected the spot where he had placed the intercostal chest drain after the accident. The lab values were within reference range, apart from an increased creatinine [1.24 mg/dl (Reference 0.70–1.20 mg/dl)] and anemia [9.8 g/dl (Reference 12–14 g/dl)].

The ECG monitor showed the rhythm presented in Fig. 24.1.

24.1.4 Which Rhythm Disorder Is Present? What Causes It?

There is an irregular rhythm with a heart rate of >100 beats/min, in other words, irregular supraventricular tachycardia. The cycles deviate in length, so that the difference between the shortest and longest PP interval is >120 ms. Causes include a sinus node dysfunction, age, and digitalis intoxication.

Small variations of the PP interval (<120 ms) are normal and referred to as heart rate variability (HRV). A reduction in HRV is a sign of autonomic nervous system damage, as caused by

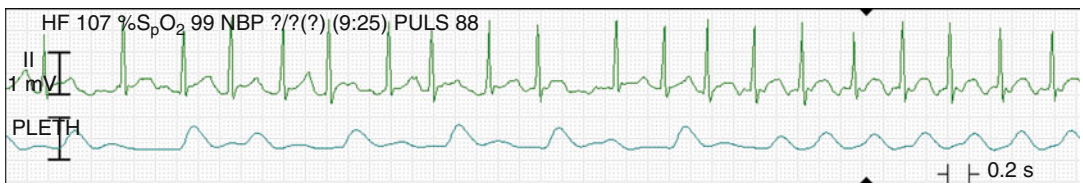

Fig. 24.1 ECG

diabetes, for example. A special form of sinus arrhythmia is respiratory arrhythmia, which is often found as a sign of vegetative dystonia in children and young adults.

24.1.5 Define Myocardial Contusion! What Are the Implications of a Myocardial Contusion?

Myocardial contusion is a traumatic damage to myocardial tissue. Five to fifty percent of blunt chest traumas result in myocardial contusion. It is often seen after motor vehicle and motorcycle accidents, deceleration accidents (such as falls), and certain high-velocity sports, such as baseball and lacrosse. Often additional injuries such as rib or sternum fractures or pulmonary contusions are present. Signs and symptoms are diverse and varied during the first 24 h and include:

- Asymptomatic
- Arrhythmia (usually sinus arrhythmias, extra systoles, ventricular tachycardias, even ventricular fibrillation)
- Conduction disorders (right bundle branch block and AV block, because usually the right ventricle and septum are affected)
- Repolarization disorders (unspecific ST- and T-wave changes)
- Structural myocardial injuries with cardiac dysfunction (papillary muscle and valve injury, coronary dissection of the right coronary artery)
- Cardiogenic shock

A serious cardiac contusion is more likely to be diagnosed than a mild one, since hemodynamic instability is usually the predominating sign [9]. However, nonspecific symptoms such as hypotension and tachycardia in trauma patients have countless other causes and can make the diagnosis more difficult.

The clinical diagnosis is confirmed by a 12-lead ECG, rhythm monitoring, echocardiography, cardiac enzyme tests, and possibly coronary angiography or myocardial scintigraphy. Of the biochemical markers, troponin is most helpful: initially and after 4–6 h, troponin T or troponin I values can confirm or rule out a diagnosis of cardiac contusion [8]. Continuous rhythm monitoring must be done for at least 48 h. Therapy with inotropes or an intra-aortic balloon pump may be indicated in severe cases. The most important differential diagnosis is a peri-traumatic myocardial infarction, which is a difficult diagnosis to make. Currently, a cardiac MRI with gadolinium is recommended for work-up [5].

>> Dr. Conall began with preoxygenation in order to get the induction done as soon as possible. Mr. Scott was agitated and uncooperative. By chance, anesthesia tech Cindy noticed the last line of the informed consent page; instead of the patient's signature, there was only a wiry scribble.

24.1.6 Define Vegetative State!

The key requirement for diagnosis of a vegetative state is that there must be no evidence of awareness of self or environment at any time. This includes:

- No response to visual stimuli
- No response to auditory stimuli
- No response to tactile stimuli
- No response to noxious stimuli
- No evidence of language comprehension or meaningful expression

If the vegetative state lasts for >4 weeks, it is called persistent vegetative state. After 1 year it is called permanent vegetative state.

Mr. Scott was agitated and uncooperative, meaning that he clearly reacted to different stimuli. The diagnosis noted on the preoperative evaluation was therefore incorrect. Mr. Scott was not in a vegetative state.

24.1.7 Evaluate the Patient's Capacity to Give Informed Consent!

"Capacity" means that a patient has the ability to make a specific decision at a specific time [7]. To have capacity, patients must be able to understand their medical condition and understand the indications, risks, benefits, and alternatives to

proposed treatments [7]. Due to Mr. Scott's abnormal mental status from his traumatic brain injury, he is not capable of giving informed consent [7]. The patient's next-of-kin, such as a parent, spouse, or sibling if spouse or parent is not alive, can provide informed consent for both surgery and anesthesia.

In emergency situations, two-physician consent is adequate, taking into account the assumed wishes of the patient. In contrast, elective procedures should be postponed until the next-of-kin can provide informed consent. If there is no next-of-kin, a court-appointed guardian can be obtained if the patient is not mentally competent.

In some cases, psychiatric consultation may need to be performed to determine if the patient possesses the competency to give informed consent.

"*>> Did Mr. Scott sign this himself*?" *she asked Dr. Conall. His only thought was* "*Which of our anesthesiologists got that signature from him*?" *He shrugged his shoulders and said*,"*Let's get started. Time is running out*."

Dr. Conall asked anesthesia tech Cindy to give 300 μg fentanyl and 200 mg propofol IV. As the laryngeal tube size 4 was placed, Mr. Scott bit Dr. Conall's finger – he was definitely not in a vegetative state. "*Ouch*! *Don't bite me*! *Cindy*, *give him another 100 mg propofol*!" *The second try went well. Dr. Conall checked the placement, and anesthesia tech Cindy secured the laryngeal tube. As Dr. Conall set the mechanical ventilation* (*IPPV*), *the peak inspiratory pressures* (*PIP*) *were over 30 mmHg. A significant leak could be heard.*

24.1.8 What Caused the Ventilation Problems?

Many causes could explain the ventilation problems:

24.1.8.1 Possible Causes of Ventilation Problems

- Insufficient anesthesia depth.
- Choice of the wrong size laryngeal tube (a size too small).
- Tube misplacement. If the tube is not placed deep enough (ignoring the recommended and marked insertion lines), both cuffs can cause a partial obstruction.
- Incorrect ventilator settings.
- Individual patient causes such as:
  - Restrictive ventilation disorders, such as reduced pulmonary compliance as a result of the pneumothorax, pleura effusions, hemothorax, pulmonary contusion, rib cage fractures, and interstitial pulmonary fibrosis
  - Limited thorax movement as a result of kyphoscoliosis

>> Dr. Conall gave another 100 mg propofol IV and repositioned the laryngeal tube. The ventilation pressures were still high and the leak still present. Dr. Conall felt the reduced lung compliance when he attempted manual ventilation.

24.1.9 What Would You Do Now?

The airway is not secure. The underlying cause is the chest trauma, which is not expected to improve in time to correct the ventilation problems for this procedure. In addition, the duration of surgery is not clear. High peak pressures caused by pulmonary or other anatomical changes are a contraindication for the use of a laryngeal tube. Since the risk of undesired stomach inflation with regurgitation and possible aspiration must be avoided, there is clear indication for securing the airway via endotracheal intubation.

>> Dr. Conall let anesthesia tech Cindy give 45 mg atracurium and 200 μg fentanyl IV. After 3 min, he intubated the trachea of Mr. Scott without incident. The ventilation was currently being performed in the BIPAP ("*biphasic positive airway pressure*") *mode. It remained stable and unremarkable throughout the entire procedure. The external fixation was removed, and after 2 h the tibia fracture was treated with an intramedullary rod. Finally, an arm cast was scheduled to be changed under general anesthesia. Dr. Conall reduced the depth of anesthesia and asked the circulating nurse to call for the next patient as he was almost ready to begin emergence.*

Anesthesia tech Cindy's phone rang. "*I've got to go to an emergency in Admissions*!" *she*

reported and then promptly left Dr. Conall alone. "*I'll be fine without you*!" *he called after her. Dr. Conall shut off the anesthesia as the cast was being placed. The janitors began removing garbage bags and used equipment to quickly ready the room for the next patient. Shortly thereafter*, *Mr. Scott woke up and began flailing around*.

"*You still need to practice a soft landing*," *said a surgeon furiously to Dr. Conall*. "*The cast isn't dry yet*!" *Nasty comments from the surgeons were nothing unusual. Mr. Scott threw his head from side to side*, *but he did not react to commands and he fought the tube*. "*Should I extubate or deepen the anesthesia*?" *wondered Dr. Conall as he held the tube and the head with both hands. The propofol syringe was not within reach*, *and no new pain stimulus was expected*, *so he decided to extubate immediately. He forgot to check for the oxygen mask*, *which had been removed by the janitor with the garbage bags*.

Even after extubation, *the patient was still agitated and uncooperative*, *flailing back and forth. Dr. Conall noticed that Mr. Scott had not yet taken a good breath*, *and shortly thereafter the peripheral saturation fell to 85%*. *He looked in the anesthesia cart for another mask and couldn't find one. Nurse Ruth happened to walk into the OR*, *and Dr. Conall immediately asked her to get a new face mask from the anesthesia workroom*. "*He's gonna start breathing any second*," *he thought to himself while the* SpO_2 *continued to plummet. Nurse Ruth returned with the mask and said*, "*He's breathing strangely. Could he have postoperative residual neuromuscular blockade*?"*Dr. Conall had also noticed the paradoxical breathing. He held the mask tightly to the patient's face and pulled the lower jaw forwards. The* S_PO_2 *continued its crash course*, *now 56%*, *and Dr. Conall was nervous*.

24.1.10 Which Diagnosis Is Most Likely with This Clinical Picture?

The symptoms and the time point of the occurrence indicate laryngospasm. The cause could be patient manipulation during emergence, extubation, excitation, or obstruction of the larynx by saliva or secretions. Residual neuromuscular blockade is unlikely since Mr. Scott was strong and moving constantly.

>> Dr. Conall tried unsuccessfully to mask ventilate the deeply cyanotic patient. The alarms were going off like crazy, *and Mr. Scott was wincing in agony. Dr. Conall frantically gave 150 mg propofol and 100 mg succinylcholine IV. Within a few seconds*, *effective ventilation was possible. Everyone present stood around like statues*, *staring at the* S_PO_2 *as it slowly began to rise. Dr. Conall shrugged his shoulders nonchalantly*, "*These young guys are tough*, *and they can take it. I already know this guy pretty well from the night he came into our ER. Guess I just saved his life for the second time*."

After a S_PO_2 *of 97% was achieved*, *the peacefully sleeping patient began to breathe on his own. Dr. Conall stopped bagging and gave the OK to transfer Mr. Scott to the PACU. All of a sudden*, *the ECG showed a heart rate twice as fast as that of the pulse oximeter. At 224 beats/min*, *the monitor's tachycardia alarm went off. Dr. Conall shut it off*, *pulled out the plug on the ECG cable*, *and pushed Mr. Scott into the PACU*.

24.1.11 What Do You Think Is the Likely Cause of the Tachycardia Alarm?

An elevated T wave on the ECG is an early sign of hyperkalemia (>6 mEq/l). ECG software often interprets tall T waves as Rs and counts them into the heart rate. Succinylcholine was given, which often leads to a short-term increase in potassium. The potassium released from the skeletal muscle cells increases the serum potassium usually by 0.5 mEq/l.

De-innervation of skeletal muscle, as occurs in immobile patients, increases the number of extrajunctional nicotinic acetylcholine receptors (nAChR). Usually there is only a small number of extrajunctional nAChR, because their synthesis is inhibited by neuronal activity. Immobility is accompanied by a decrease in neuronal activity. The absence of the inhibition quickly – within 48–72 h – induces extrajunctional nAChR on the

surface of the myocytes. These receptors are typically very sensitive to agonists like acetylcholine and succinylcholine. Stimulation of the receptors tends to lead to excessive potassium release, with the observed T-wave changes. If potassium levels increase further, then the decreased gradient of the transmembrane potassium concentration can lead to PR broadening and QRS prolongation. During this time, there is increased risk of an impulse conduction block, which may even lead to ventricular fibrillation or asystole.

Therefore, succinylcholine may only be used if absolutely necessary in muscular dystrophy, de-innervated muscles, serious skeletal muscle trauma with immobility, lesions of the first motor neuron, as well as burns [4].

>> *Upon arrival in the PACU, vital signs were*:

- *Blood pressure 135/85 mmHg*
- *HR 90 beats/min*
- *S_pO_2 93% in room air*

The T waves were not quite so tall anymore, but still visibly abnormal. Mr. Scott lay in bed calm and breathing adequately. Dr. Conall handed over the patient to nurse Elisabeth and ordered nasal oxygen administration until the S_PO_2 reached 97%.

Despite the additional oxygen, the S_PO_2 remained at 93% and then decreased to 90%. Mr. Scott was breathing with increased effort and frequency. Nurse Elisabeth called the PACU attending Dr. Sage. He yanked his stethoscope out of his pocket and auscultated the lungs – detecting fine crackles bilaterally.

24.1.12 What Differential Diagnoses Are You Thinking About Now?

Acute postoperative dyspnea may have many causes:

24.1.12.1 Differential Diagnoses of Postoperative Dyspnea

- Pain
- Prolonged neuromuscular blockade
- Aspiration
- Bronchospasm
- Asthma
- Laryngeal edema
- Pulmonary embolism
- Pneumothorax
- Pulmonary edema

After the bilateral fine crackles appeared, the diagnosis is more limited.

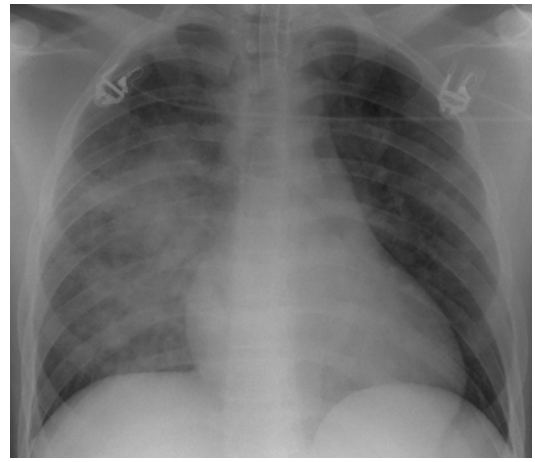

Fig. 24.2 Supine chest X-ray

>> *Dr. Sage read in the anesthesia record that the last opioid administration was 2 h ago. To exclude a pain-induced dyspnea, he gave morphine IV. The desired effect was not achieved. He called Dr. Conall who, somewhat unwillingly, returned to the PACU. Dr. Conall told his colleague about the increased intraoperative airway pressures, the past history of chest trauma, and the laryngospasm during emergence. Both suspected that a pneumothorax had formed again. "I better place a chest tube. I've done it before, I know how to go in," suggested Dr. Conall without delay. Dr. Sage didn't like the immediate action and ordered, just to be safe, a supine chest X-ray* (Fig. 24.2).

24.1.13 What Do You Notice? What's Your Diagnosis?

The radiologist noted the following in her report: heart size normal, new-onset diffuse interstitial and possible alveolar edema, no pneumothorax, and fractured ribs.

The following symptoms and signs are present:

- Desaturation
- Crackles
- Dyspnea, tachypnea
- Agitation
- X-ray: interstitial pulmonary edema
- S/P laryngospasm

The most likely diagnosis, based on the symptoms and the history, is negative pressure pulmonary edema (NPPE) (see Sect. 14.1.7). The incidence of NPPE is 0.05–0.1 %. It occurs in young, healthy, muscular patients. It is usually seen after the following:

- Laryngospasm (50 % of all cases)
- Upper airway obstruction
- Aspiration
- Endotracheal suctioning
- Biting the endotracheal tube or laryngeal mask airway
- Snoring

Pathophysiologically, forced inspiration against resistance, such as a closed glottis, leads to extensive negative intrapleural pressure. Depending on muscle strength, pressures of up to –140 cmH_2O can be reached. This is 10–20 times more negative than in normal breathing. As a result, the venous return to the heart increases, causing excessive transcapillary hydrostatic pressure in the pulmonary circulation. Pulmonary edema develops within minutes or hours.

The therapy is symptomatic, consisting of continual O_2 application. The symptoms usually subside spontaneously within 24 h. Supportive CPAP or mechanical ventilation with PEEP can be indicated for a brief period of time. Administration of diuretics is debated [1, 2].

"*>> Thank goodness I didn't agree putting in a chest tube*," *said Dr. Sage*. "*Sometimes one must take the time to be careful in emergency medical care*." *He had heard of low pressure pulmonary edema*, *but had never seen a patient with it and was impressed by this case. With a CPAP mask and a tight seal*, *Mr. Scott's oxygen saturation improved dramatically. Dr. Sage transferred him to the ICU*, *explained the patient's case and current diagnosis*, *and recommended therapy to the team there*.

24.2 Case Analysis/Debriefing

24.2.1 Which Medical Errors Do You See in the Presented Case?

24.2.1.1 Preoperative Evaluation and Informed Consent

Legally speaking, there was no informed consent obtained for this procedure. Without such, it is physical injury, and the elective procedure and anesthesia should never have been started without it.

In our day-to-day work, this problem is often avoided as we tend to classify questionable procedures as emergency procedures. Legally, the anesthesiologist must also check the classification of the procedure as "emergency." Surgical misjudgment is not accepted in court as grounds for the lack of informed consent.

24.2.1.2 Choice of Method to Secure the Airway

Due to the short time period between the chest trauma and the current pulmonary disease, the choice of the laryngeal tube was incorrect. The thoughts about caring for the cervical spine were appropriate; however, a C-spine whiplash injury does not increase the risk for spinal injury during a properly carried out endotracheal intubation with manual in-line stabilization.

24.2.1.3 Monitoring

Due to the severity of the multiple trauma, intraoperative cardiopulmonary complications should be expected. These include ventilation and oxygenation problems, abnormal ratios of dissolved oxygen and requirements, tachycardia due to anemia, increased intraoperative bleeding due to the anticoagulation therapy, danger of malignant arrhythmia due to the cardiac contusion, and acute fat or thrombotic lung embolism. A 5-lead ECG is obligatory for rhythm and ischemia monitoring. The transport of the patient into the PACU without monitoring was grossly negligent.

24.2.1.4 Emergence

The emergence occurred without the required calm and careful handling by the anesthesiologist.

The laryngospasm may have been prevented if the following were given attention: punctual pain therapy at the end of the operation, standardized procedures for oropharyngeal suctioning, check of equipment needed for emergence, personnel available within earshot to assist, and consideration of extubation criteria. Used equipment should never be removed from the OR until after emergence when the patient leaves the OR.

24.2.1.5 Therapy of the Laryngospasm

Due to the fact that the face mask had been removed with the garbage while the patient was emerging, the recommended therapy for the laryngospasm – assisted high pressure ventilation – could not begin. The recommended succinylcholine dose to relax the vocal cords in emergency is 0.15–0.3 mg/kg body weight. The unnecessarily high succinylcholine dose, together with the patient's increased sensitivity, leads to temporary hyperkalemia which was not expected or recognized by the anesthesiologist. Ignoring the ECG alarm and disconnecting the cable during patient transport into the PACU was grossly negligent and could have had fatal consequences.

24.2.2 Which Systems Failures Can You Find in the Presented Case?

24.2.2.1 Absence of Standards for Emergence

The resident should have called his attending prior to extubation. In addition, an assistant, such as an anesthesia tech or a circulating nurse, should be nearby. The anesthesiologist assumes responsibility.

24.2.2.2 PACU Transfer

The report given to the PACU nurse was incomplete. Specific intraoperative anesthetic problems, such as laryngospasm and ECG changes, were not mentioned. Further treatment of the patient was therefore delayed and made more difficult.

Physician-to-physician patient reporting is mandatory in critically ill patients and in cases of extremely difficult anesthesia.

24.2.2.3 Personnel Assignments

Dr. Conall had informed anesthesia tech Cindy of the planned extubation. Cindy was called off to another task. The call came from a person who probably didn't know what Cindy was doing at that moment. The person should at least have asked if Cindy was available to assist.

24.2.3 Take Action – Think? Think – Take Action?

As an anesthesiologist-in-training with a special interest in trauma and emergency care, Dr. Conall is accustomed to making quick decisions. He naturally assumes the role of a leader and distributes tasks and information to the team members. His bustle of activity is an obstacle to considering several therapy options.

The immediate extubation without presence of an assistant and the premature offer to insert a chest tube indicate a lack of critical analysis of the problem and possible solutions. Also, Dr. Conall does not feel compelled to recruit additional resources, such as his attending physician.

How can such a frenzy of decision and action be avoided, even in time-critical situations?

The positive example of the PACU attending Dr. Sage makes it clear: he tries to obtain as much information as possible about the patient in order to discuss and then decide on the optimal therapy options. During his search for more information, he did not let himself be pushed into action by his colleague. He thereby prevented placement of an unnecessary chest tube.

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Case 25: Inguinal Abscess

25

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25.1 Case Introduction

>> It was 7:15 in the evening, only 45 more minutes till Dr. Conner got off. He was on the late shift and had been doing preoperative evaluations the first part of his long day. After that, he took over the anesthesia for a general surgery procedure, to relieve a colleague. He enjoyed his work, but today he was looking forward to his after-work plans. Just as he had brought the patient to the PACU and given the receiving nurses the rundown, his phone rang. Dr. Eldridge, the anesthesiologist-in-charge, assigned him an urgent procedure, debridement of an abscess. "You'll finish before it's time for you to get off," Dr. Eldridge said. "I don't think so," thought Dr. Conner, "I might as well forget about catching the movie after work tonight."

Dr. Conner had worked in the hospital a little more than 4 years. He had almost completed all the procedures he needed for his board certification, and he was scheduled to rotate to the ICU.

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He was already excited about the medical challenges, although not so thrilled about shift work. When Dr. Conner reached the OR, the patient, Mr. Walter, was waiting. Timothy, the anesthesia technician, was busy getting the patient hooked up to the monitor. Dr. Conner introduced himself and began to review the patient's medical record. He copied the following information from the preoperative form to the electronic anesthesia record:

- *76-year-old male, 175 cm, 70 kg*
- *Diabetes controlled with metformin*
- *Renal insufficiency, in the stage of compensated retention*
- *Advanced prostate cancer, first diagnosis 2 years ago*
- *Chronic pelvic pain from bone metastasis, treated with diclofenac and pregabalin*
- *Inpatient treatment for UTI with fever, 4 weeks ago*

Shortly after his last discharge, Mr. Walter noticed swelling in his right inguinal region. The swelling slowly got bigger and has been red and painful since yesterday; therefore, he came to the urgent care center this afternoon. Mr. Walter denied any further illnesses or complaints. Only the pain in his pelvis hindered him from being as active as he would like. "In the past 2–3 days, though, I have felt increasingly weak. The urgent care doc thought my weakness is coming from the abscess and my fever. I am pleased the abscess can now be treated," said Mr. Walter. During Dr. Conner's conversation with Mr. Walter, he noticed the patient's tachypnea. He glanced at the monitor and saw:

- *Blood pressure 105/60 mmHg*
- *Heart rate 115 beats/min*
- *Sinus rhythm*
- *S_PO_2: 95%*

25.1.1 What Could Be Causing the Tachycardia?

Preoperative sinus tachycardia can have many causes, such as:

- Psychological stress, e.g., anxiety or excitement
- Volume depletion
- Anemia
- Electrolyte imbalance, e.g., hypokalemia, hypomagnesemia, hypocalcemia
- Hypercapnia
- Hypoxia
- Hyperthermia/fever
- Infection
- Hypothyroidism
- Pheochromocytoma
- Medications, e.g., tricyclic antidepressants, theophylline, caffeine
- Drugs, such as cocaine, amphetamines
- Withdrawal symptoms

In a non-sinus supraventricular tachycardia, causes such as reentry tachycardia or atrial fibrillation/flutter must be considered.

>> Dr. Conner's quick physical exam did not reveal any further abnormalities. The ECG from 4 weeks ago showed a normal sinus rhythm. The following laboratory values were out of normal range:

- *Creatinine: 2.9 mg/dl (reference value: 0.5–1.0 mg/dl)*
- *Urea: 10.5 mmol/l (reference value: 3.0–9.2 mmol/l)*
- *Cystatin C: 4.33 mg/l (reference value: 0.93–2.68 mg/l for patients aged 60–79 years)*
- *Hemoglobin: 7.9 g/dl (reference value: 12–14 g/dl)*
- *Hematocrit: 25% (reference range: 37–47%)*
- *Leukocytes: 20,900 (reference value: 3,600–9,800 cells/mcl)*

During Mr. Walter's last hospitalization, the creatinine was also elevated: 2.3 mg/dl. He also was transfused 2 units of RBC during his last visit, due to anemia.

The anesthesia technician, Timothy, called the blood bank. "The surgeons ordered 2 units crossmatched. The lab technician says she needs 15 more minutes until the tests are done and the blood is ready." Dr. Conner replied, "Tell her to send the bags as soon as possible."

25.1.2 What Is the Significance of Cystatin C to Assess Kidney Function?

The best index to determine kidney function is the glomerular filtration rate (GFR). Calculating GFR with the help of external filtration markers such as inulin is time-consuming, so in practice, endogenous filtration markers are usually used

to estimate GFR. The most common surrogate parameter used is creatinine concentration, or the reciprocal creatinine concentration. Ideally, the 24-h urine creatinine concentration should be measured along with the serum creatinine concentration in order to properly calculate the GFR.

Many factors influence the accuracy of this calculation, such as failure to collect all urine excreted in 24 h and the patient's age, diet, physical activity, and muscle mass.

In the search for a more reliable marker, cystatin C has recently come into focus [5, 6]. Cystatin C is a cysteine proteinase with a low molecular weight of 13.359 kD. Cystatin C is produced in all nucleated cells, and its plasma concentration is unrelated to physical activity and muscle mass. The reciprocal value of cystatin C correlates especially accurately with the GFR, allowing slight decreases in renal function to be better detected [11].

25.1.3 Would You Transfuse?

An in-depth discussion of transfusion can be found in Case 4 (Sect. 4.1.11). The decision to transfuse should be made individually. Induction of anesthesia can lead to a reduction in cardiac output; volume therapy can then further lower the hematocrit. The cause of Mr. Walter's tachycardia is not yet understood; his tachypnea could be an indication of low available oxygen ($\dot{D}O_2$). Therefore, a transfusion is most likely necessary.

>> *Dr. Conner called his attending, Dr. Eldridge, because he wasn't quite sure whether he should transfuse before inducing anesthesia.* "*You want to get out of here on time tonight, don't you? Start the anesthesia and transfuse the blood as soon as you get it,*" *was the answer he received.*

"*Let's use a laryngeal mask airway,*" *Dr. Conner said to Timothy, after hanging up the phone. Dr. Conner discussed the possible complications of the anesthesia and blood transfusion with Mr. Walter and obtained informed consent. Then he began preoxygenation, which didn't actually work so well, because Mr. Walter didn't have a single tooth left. The measured end-tidal CO_2 was 19 mmHg.*

25.1.4 What Could Be the Cause of the Low End-Tidal CO_2?

The most likely cause of the low end-tidal PCO_2 during preoxygenation is a leaky mask. Low end-tidal CO_2 is also found with an elevated respiratory minute volume or increased dead space ventilation – often seen in the case of a pulmonary embolism or serious pulmonary hypertension.

25.1.5 What Is Preoxygenation? What Is the Purpose of Preoxygenating Patients?

Preoxygenation refers to ventilation with 100 % O_2 before inducing respiratory arrest. In spontaneously breathing patients, preoxygenation is done by placing a mask with 100 % oxygen over the patient's face, instructing the patient to breathe normally (tidal volume respiration) for 3–4 min or by having the patient take 4–8 very deep breaths from the mask (vital capacity respiration). The preoxygenation should achieve an end-expiratory oxygen concentration greater 90 %.

Oxygen is stored in blood, in tissues – especially muscle – and in the lungs. The oxygen stores in tissue and in blood are already almost completely saturated by normal room air ventilation, so that the lungs are most affected by preoxygenation. During preoxygenation, the lung's nitrogen will be washed out and replaced with oxygen. Through this denitrogenation, the oxygen level in the lungs increases fourfold as it replaces the nitrogen. Altogether, the preoxygenation triples the oxygen content of the body. Correspondingly, the apnea tolerance is extended, delaying the occurrence of hypoxemia, so that more time is available to secure the airway.

25.1.6 When Should a Dramatic Drop in Oxygen Saturation (S_PO_2) After Induction of Anesthesia Be Expected?

A dramatic decrease in S_PO_2 after induction can have several causes:

25.1.6.1 Reduced Oxygen Storage Capacity

The oxygen storage of the lung is actually the functional residual capacity (FRC). A reduced FRC, as is usually the case with children, pregnant, and adipose patients, shortens the period of apnea tolerance.

The oxygen storage capacity of the body is also reduced with a reduction in the hemoglobin concentration. S_PO_2 does not reflect oxygen content (C_aO_2).

25.1.6.2 Increased Oxygen Utilization

The normal oxygen utilization ($\dot{V}O_2$) at rest is 250–300 ml/min. VO_2 increases with increasing metabolism, often drastically; for example, children or patients with fever or hyperthyroidism can have very short apnea tolerance times.

25.1.6.3 Severe Circulatory Depression

Induction of general anesthesia can cause a dramatic decrease in cardiac output; the peripheral perfusion can be so decreased that the pulse oximeter can no longer measure a valid value. With simultaneous respiration, a drastically reduced expiratory PCO_2 is the clue for an invalid S_PO_2 measurement and an inadequate cardiac output.

>> *Dr. Conner fiddled with the mask, but was unable to get a tight seal. Finally he asked Timothy to administer 200 µg fentanyl and 120 mg propofol IV. The laryngeal mask airway was then placed uneventfully. As expected, the blood pressure dropped to 82/40 mmHg following anesthetic induction. The heart rate remained at 120 beats/min.* "*Give Mr. Walter some phenylephrine and turn up the infusion rate*," *Dr. Conner said to Timothy. Because the second half of the amp of phenylephrine didn't have much effect on the pressure, a vasopressin infusion was started.* "*Come on, let's get the surgeons to scrub. I don't want to be here all night*!" *said Dr. Conner.*

He turned up the desflurane to maintain the anesthesia, and the surgeons began their preparations. Mr. Walter's systolic pressure stayed around 90 mmHg, even though the vasopressin infusion was running. Timothy hung a new bag of normal saline solution. The pulse stayed at 120/min. The packed red blood cells were delivered shortly after the procedure had begun; the bedside tests checked out, so Timothy began the transfusion.

25.1.7 What Are the Determinants of Systemic Arterial Blood Pressure?

The mean arterial blood pressure is proportional to cardiac output (CO) and systemic vascular resistance (SVR):

$$\text{Arterial pressure}\left(\text{mmHg}\right) = \text{CO}\left(\text{l}/\text{min}\right)\times\text{SVR}\left(\text{dynes}\times\text{s}\times\text{cm}^{-5}\right) \quad (25.1)$$

A drop in CO can be compensated by an increase in SVR, and vice versa. Therefore, blood pressure alone does not allow for accurate determination of CO or SVR (see Sect. 3.2.1).

25.1.8 What Causes Intraoperative Hypotension?

25.1.8.1 Vasodilation

Vasodilation leads to relative hypovolemia. It is triggered by IV and inhalational anesthetics or neuraxial anesthesia. Furthermore, vasodilatation can be caused by anaphylactic reactions, or the release of dilating agents/mediators such as during opening of the abdomen and moving the intestines, or after opening a tourniquet, or by septic bacteremia.

25.1.8.2 Reduced Venous Return

Venous return can be reduced by many mechanisms, such as patient positioning, pneumoperitoneum, high respiratory pressures, tension pneumothorax, or intraoperative accidental compression of the inferior vena cava.

25.1.8.3 Absolute Volume Deficiency

Apart from blood loss, other causes of dehydration must be considered, such as preoperative

fasting, perspiration, diuretics, or preoperative bowel preps.

25.1.8.4 Acute Decrease in Afterload

Opening tourniquet or blood vessel clamps results in a sudden decrease in afterload. A sudden drop in blood pressure may be enhanced by the release of vasoactive mediators from the previously ischemic area.

25.1.8.5 Arrhythmia

Arrhythmias can lead to hypotension through a variety of mechanisms. One example is limited left ventricular filling as a result of atrial fibrillation.

25.1.8.6 Abnormal Contractility

Abnormal contraction is a typical result of myocardial ischemia. Administration of IV fluids and negatively inotropic solutions further reduce the contraction capacity.

25.1.8.7 Pulmonary Embolism

An acute embolism of air/CO_2, methyl methacrylate bone cement, or a thrombus can lead to acute right heart failure. See Case 11 (Sect. 11.1.5) for complete pathophysiology.

>> All vital signs were stable. "We can stop the vasopressin infusion as soon as the RBCs are in," thought Dr. Conner as he charted on the computer. Suddenly the respiratory alarm sounded. Dr. Conner looked up and realized that the expiratory CO_2 was too low. One glance at the CO_2 curve and he knew the cause: Mr. Wilson was trying to breathe. "Alright, go for it!" thought Dr. Conner, "Spontaneous respiration is great with laryngeal mask airways." So he turned the respirator to "spontaneous." He didn't want to give additional opioids because the surgery wouldn't take much longer.

The total surgery time was 25 min. As the abscess was opened, a large amount of smelly pus oozed out. After the second RBC bag, the vasopressin infusion rate could be cut in half. A hemoglobin check showed 10.4 g/l. "Wow, if I hurry, I'll make it to my movie tonight!" thought Dr. Conner as he shut off the desflurane vaporizer. Mr. Walter was breathing adequately through the laryngeal mask airway. The end-tidal PCO_2 was 21 mmHg. Just as Dr. Conner was removing the laryngeal mask airway, he noticed the heart rate jump to 140 beats/min. The rhythm was no longer regular, and atrial fibrillation was present. The blood pressure remained unchanged.

25.1.9 What Should You Do Now?

Management of new-onset fibrillation depends on the patient's hemodynamic status. Atrial fibrillation (a-fib) leads to loss of atrial contraction and ensuing atrial dilation. In combination with hypovolemia, the ventricular filling is especially affected, which can lead to a drop in cardiac output. Unstable patients with symptoms and signs of low cardiac output or cardiac ischemia should undergo urgent cardioversion [3, 8].

If cardiac function is unaltered, i.e., no hemodynamic instability, diagnosis may be confirmed with a 12-lead ECG. Rhythm and rate should be monitored and treated pharmacologically. Suitable medications include β-blockers, digitalis, or amiodarone. Anticoagulation therapy is indicated to prevent thromboembolytic complications. A cardiology consultation is recommended (for more information on this topic, please see Case 21, Sects. 21.1.6 and 21.1.7, as well as Fig. 21.2).

25.1.10 What Causes Atrial Fibrillation?

There are many causes of a-fib (Table 25.1 [10]; and Sect. 21.2.3). Clarification of the cause is important, in order to give the proper therapy for rhythm and rate control. Especially important are the reversible causes, for example, hypothyroidism or electrolyte imbalances.

>> Now Dr. Conner was getting nervous. He called his attending, Dr. Eldridge, to ask what to do. "We should give Mr. Walter 2 mg metoprolol and a bolus of IV fluids, then do an arterial blood gas." Dr. Conner asked Timothy to increase the IV fluids, as he opened the desflurane vaporizer again.

Table 25.1 Causes of a-fib [10]

Primary		Idiopathic
Secondary	Cardiac causes	Rheumatic or inflammatory heart disease
		Cardiomyopathy
		Hypertensive heart disease
		Coronary artery disease
		Postcardiotomy syndrome
		Valvular heart disease
		Pericarditis
		Cardiac contusion
		Cardiac tumors
		Lysosomal storage diseases
		Mechanical irritation (e.g., by catheters or wires)
	Noncardiac causes	Hyperthyroidism
		Electrolyte imbalances
		Alcohol-toxic heart disease
		Fever/infections/sepsis
		Noncardiac shock
		Chronic renal insufficiency
		Chest trauma
		Subarachnoid hemorrhage (see Sect. 2.1.5)
		Side effects of various drugs

After administration of the β-blocker, Mr. Walter's heart rate dropped to 130 beats/min. The blood pressure was unchanged at 105/60 mmHg. With the vasopressin infusion still running, Dr. Conner drew blood from the radial artery and gave the blood sample to Timothy. Timothy soon returned and handed the printout to Dr. Conner saying, "We haven't seen anythin' like this in a long time!"

- *pH: 7.18 (reference value 7.35–7.45)*
- *P_aO_2: 280 mmHg (reference value 70–100 mmHg)*
- *P_aCO_2: 25 mmHg (reference value 36–44 mmHg)*
- *HCO_3^-: 10.9 mEq/l (reference value 22–26 mEq/l)*
- *BE: – 18 mEq/l (reference value ±2 mEq/l)*
- *S_aO_2: 99.5 % (reference value 95–98 %)*
- *Lactate: 1.2 mmol/l (reference value 0.5–2.2 mmol/l)*
- *Na^+: 143 mEq/l (reference value 135–150 mEq/l)*
- *K^+: 5.4 mEq/l (reference value 3.5–5.0 mEq/l)*

25.1.11 How Do You Interpret the ABG?

The patient has a metabolic acidosis, with partial respiratory compensation. The hyperkalemia is probably a result of the H^+ ion excess.

25.1.12 How Can Metformin Contribute to Metabolic Acidosis?

Metformin is a biguanide and is employed in the oral therapy of diabetes. It decreases hepatic gluconeogenesis and increases glucose uptake by skeletal muscle and fatty tissue. Furthermore, metformin inhibits lipolysis and reduces the release of free fatty acids. The inhibition of gluconeogenesis is shown in Fig. 25.1.

Metformin is not metabolized and is almost completely eliminated renally. In the case of renal failure, metformin can accumulate and result in lactic acidosis [7].

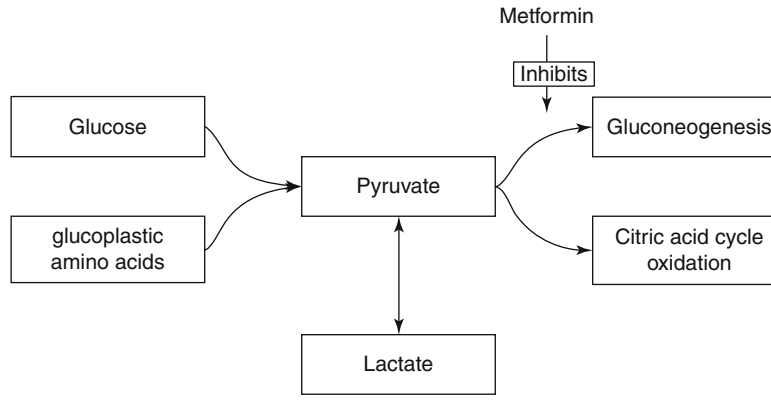

Fig. 25.1 Effects of metformin on lactate metabolism. Metformin inhibits gluconeogenesis from pyruvate. In case of metformin overdose, accumulation of lactate may occur

25.1.13 What Causes Metabolic Acidosis?

Metabolic acidosis can occur through increase of acids or loss of bases.

25.1.13.1 Increase of Acids

An accumulation of acids occurs in the following situations:

- Increased endogenous acid formation:

In the case of ketoacidosis (diabetic coma) or in lactate acidosis, an acid excess is created.

Increased lactate levels arise from anaerobic metabolism in hypoxic situations such as shock, seizures, or after intake of certain medications such as biguanides or xylitol infusions.

- Exogenous acid intake:

Excessive intake of acids is usually only seen in suicide attempts or accidental ingestion of acids such as acetylsalicylic acid, methanol, acetaminophen, and an excess of chloride.

- Inhibition of acid excretion/breakdown:

In the case of drastically reduced renal function, especially with acute renal failure, the renal buffering capacity is restricted due to limited capability to excrete acids. In addition, liver failure decreases the lactate breakdown and leads to acid accumulation.

25.1.13.2 Bicarbonate Loss

Metabolic acidosis due to bicarbonate loss is rare. It can be observed in the following situations:

- Acid loss via the gastrointestinal tract, such as with diarrhea, gallbladder–pancreas fistulas, or ureteroenterostomy
- Renal loss, such as in proximal tubular acidosis or through carboanhydrase inhibition

In this presented case, the metabolic acidosis is probably due to renal failure. In addition, infectious diseases can exacerbate renal insufficiency, which can set off a systemic inflammatory reaction.

>> *Dr. Conner thought about it. Why Mr. Walter had constantly breathed against the ventilator was now obvious to him. Dr. Conner thought he vaguely remembered that a pH under 7.20 requires treatment*. "*I'll put in a central venous catheter and an arterial line*," *he said to anesthesia technician Timothy*. "*We've got to buffer*."

25.1.14 Should You Buffer?

Administering bicarbonate has been debated for years and should only be done after careful consideration. First, the cause of the acidosis must be treated. The goal of buffering is to restore normal physiological balance, thereby improving hemodynamics. Simultaneously, buffering causes a left shift of the oxygen binding curve and reduces tissue perfusion.

>> *As Timothy was preparing for the central venous line, Dr. Conner phoned the ICU and arranged a bed for Mr. Walter. With quick teamwork, the central and arterial lines were in within minutes. Dr. Conner immediately infused 100 ml sodium bicarbonate* via *the central line and shut off the desflurane.*

Suddenly Mr. Walter sat up, looked around very confused, and tried to speak through the laryngeal mask airway. The door opened and Dr. Eldridge, the attending anesthesiologist, walked in. When he saw the central and arterial lines, he raised his eyebrows and said to Dr. Conner, "*Don't you want to free the patient of the mask?*" *Then he glanced at the anesthesia record and shook his head.* "*I think we are going to need to have a long discussion about this. After all, you're going to the ICU before long.*" *After Dr. Conner had delivered Mr. Walter to the ICU without further complications, he went into the OR staff room where Dr. Eldridge was waiting.*

25.2 Case Analysis/Debriefing

25.2.1 What Is SIRS?

According to the consensus conference of the American College of Chest Physicians/Society of Critical Care Medicine (ACCP/SCCM), severe inflammatory response syndrome (SIRS) can occur as a reaction to many different types of injury. Per definition, at least two of the criteria listed below must be fulfilled before SIRS can be diagnosed.

25.2.1.1 SIRS Criteria (≥2 Must Be Fulfilled)

- Fever >38 °C or hypothermia under 36 °C
- Tachycardia >90 beats/min
- Respiratory insufficiency with a frequency of >20/min or spontaneous hyperventilation with a P_aCO_2 <32 mmHg or P_aCO_2 <70 mmHg with spontaneous ventilation or P_aCO_2/F_iO_2 <175 under controlled ventilation and without preexisting pulmonary disease
- Leukocytosis >10,000 cells/mcl or leukopenia <4,000 cells/mcl or >10 % immature neutrophil granulocytes

→ In Mr. Walter's case, the SIRS criteria were fulfilled.

25.2.2 What Do You Know About Sepsis, Severe Sepsis, and Septic Shock?

25.2.2.1 Sepsis

A diagnosis of sepsis can be made if SIRS criteria are fulfilled and an infection can be detected or is clinically suspected [2].

→ In Mr. Walter's case, the sepsis criteria were fulfilled.

25.2.2.2 Severe Sepsis

Severe sepsis [2] is present when, additionally, one or more of the following organ dysfunctions are present:

- Acute encephalopathy
- Relative or absolute thrombocytopenia (decrease of >30 % within 24 h or thrombocyte count ≤100,000/mcl)
- Arterial hypoxemia with a P_aCO_2 <75 mmHg breathing room air or a P_aCO_2/F_iO_2 smaller than 250 mmHg under oxygen application
- Renal dysfunction with diuresis less than 0.5 ml per kg body weight per hour for at least 2 h or a doubled increase in serum creatinine above the reference value
- Metabolic acidosis with a base excess smaller than 5 mEq/l or lactate concentration greater than 1.5 times the reference value

→ Because of the metabolic acidosis, Mr. Walter fulfilled the criteria for severe sepsis. Renal dysfunction was present.

25.2.2.3 Septic Shock

Septic shock is present when [2], additionally, for at least one hour, the systolic arterial blood pressure remains lower than 90 mmHg or vasopressor therapy is necessary to maintain blood pressure. The hypotension exists despite adequate volume and cannot be explained by other causes.

Mr. Walter received vasopressors, but only after the commencement of general anesthesia. An adequate fluid therapy was not (yet) carried out.

25.2.3 What Are the Mainstays of Therapy for Sepsis?

The mainstays of sepsis therapy are:
- Causal treatment
- Supportive care
- Adjunctive therapy

More information can be found in the Surviving Sepsis Campaign international guidelines for management of severe sepsis and septic shock [4].

25.2.3.1 Causal Therapy

Most importantly, treat the cause through elimination of the focus of infection. If the cause is unknown, a search should be immediately undertaken to identify the focus.

Early, calculated broad-spectrum antibiotic therapy should be initiated to narrow the microbial spectrum as soon as possible.

25.2.3.2 Supportive Therapy

Supportive therapy assures an adequate oxygen supply. Early hemodynamic stabilization through fluid resuscitation should be achieved, with a targeted median arterial pressure of 65 mmHg. Depending upon the patient's comorbidities, a higher mean arterial pressure may be desired. Extended hemodynamic monitoring may be necessary. Central venous oxygen saturation of ≥70 % should be maintained in patients with severe sepsis or septic shock, as part of the "early goal-directed therapy" plan [9].

To assure adequate oxygen supply, intubation and mechanical ventilation may be necessary, with a targeted S_PO_2 of >90 %. If mechanical ventilation is required, the recommendations of the Acute Respiratory Distress Syndrome Network (ARDSNET) should be followed [1].

25.2.3.3 Adjunctive Therapy

Adjunctive therapy includes, e.g., administration of glucocorticosteroids, antithrombin III, immunoglobulins, thrombosis prophylaxis, and early enteral nutrition.

25.2.4 What Are the Indications for the Placement of a Central Venous Catheter (CVC)?

The main indications for central venous catheterization are listed below.
- Hemodynamic monitoring with measurements of central venous pressure (CVP) and central venous saturation
- Administration of circulatory and cardiac medications with a short half-life, e.g., catecholamines such as norepinephrine
- Administration of fluids with an osmolarity of >600 mOsmol/l, especially involving parenteral nutrition
- Administration of venous irritants, such as sodium bicarbonate and potassium chloride
- No possibility of placing a peripheral line, as may be the case in:
  - Shock
  - Extensive burns
  - Poor venous access, as a result of obesity, long-term infusion therapy, or intravenous drug abuse
- Neurosurgical operations in a sitting and half-sitting position, in order to measure the CVP and for air aspiration in the case of venous air embolism

For treatment of sepsis, a central venous catheter is needed for the administration of cardiovascular medications (norepinephrine, dobutamine) and to allow extended hemodynamic monitoring.

25.2.5 Which Medical Errors Do You See in the Presented Case?

25.2.5.1 Preoperative Evaluation

The preoperative evaluation and informed consent obtained in the OR were only superficially "informed." The tachypnea was noticed by Dr. Conner, but he paid no attention to it. He also ignored Mr. Walter's fever. Both were clues that the local infection had already become systemic. SIRS or sepsis was not considered. Consequently, an ICU bed was not arranged in advance.

In addition, the changes in Mr. Walter's renal function since the last hospital visit were ignored. The possibility of an acute septic renal injury in the context of existing renal insufficiency was not considered.

25.2.5.2 Note

The same issues apply to the admitting surgeon, who did not recognize the severity of the illness. The surgeon and anesthesiologist show joint responsibility for the poor preoperative assessment of the patient's risk.

25.2.5.3 Induction of General Anesthesia

Before inducing general anesthesia, the indication for a blood transfusion was noted. The induction was carried out, even though the RBCs were not yet approved by the transfusion lab. Mr. Walter had already received a blood transfusion, so there was a possibility that irregular antibody formation were already triggered; therefore, a significant chance existed that the blood could not be crossmatched. A transfusion was immediately indicated, due to the hemoglobin. The action taken put the patient in danger.

25.2.5.4 Controlling Blood Values

Mr. Walter was diabetic. When metabolic acidosis was diagnosed, at the least blood sugar should have been measured. In addition, there was no post-buffer ABG, which leads one to question the necessity of the preinduction ABG.

25.2.5.5 Monitoring

Mr. Walter fulfilled the criteria for severe sepsis. The fact that this did not occur to Dr. Conner explains why he did not initiate proper monitoring. At the very least, invasive blood pressure monitoring, a CVC or CVP-controlled volume administration, central venous saturation measurement, and a urine catheter to quantify output would have been sensible. Temperature, as well, should have been measured. In addition, a patient with sepsis should be intubated and not just managed via laryngeal mask airway.

25.2.6 What Systems Failures Can Be Found in This Case?

25.2.6.1 Obtaining Informed Consent

The consent was not obtained until the patient was already in the OR, putting the patient under unnecessary time pressure and stress.

25.2.6.2 Permission for Anesthesia

The attending anesthesiologist gave the go-ahead over the telephone without having seen the patient, even though Dr. Conner's recap signaled that this was not a routine case. This behavior does not meet ACGME standards for training of residents, and it does not meet billing requirements that require the attending to be present for induction, emergence, and all critical phases, and immediately available throughout.

25.2.6.3 Choice of Anesthesiologist

Dr. Eldridge asked Dr. Conner to carry out the anesthesia and informally indicated that working late might be necessary.

25.2.7 Still Interested, or Would You Rather Get Off to the Movies?

Read a little more; the commercials are still playing.... Dr. Conner was mentally already at the movies. His main goal was getting off work on time, which was looking pretty iffy. Extra work, unpredictable complications, too much thinking – all that was no longer on his schedule for the day. As Mr. Walter spontaneously attempted to breathe, Dr. Conner anticipated a quick reversal of anesthesia and a low aspiration risk and was pleased. Other reasons for spontaneous breathing, such as insufficient analgesia, insufficient hypnotics, or in this case, relative hypoventilation, were simply not considered.

Dr. Conner's attitude was: "Nothing will go wrong, everything is normal, I have it all under control." Information which indicated otherwise was blotted out. He didn't pick up on the clues that he needed to reevaluate the situation objectively.

How could this problem have been avoided?

In Case 28 (Sect. 28.2.3) a "worst-case scenario" strategy is described. Imagining the worst possible reason for a symptom increases the activation level by mobilizing thought processes. This problem-solving strategy only works in the presence of motivation, which Dr. Conner lacked. In such a case, team resources are essential. Dr. Conner should have been replaced by another anesthesiologist or at least informed how much longer he needed to work. For this to work, the team culture must permit such open communication. Dr. Conner didn't clearly state that he absolutely had to leave at a certain time, nor was he told how much longer he had to stay.

Alright, that's enough – off to the movies with you!

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Case 26: Aortic Aneurysm

26

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Contents

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26.1 Case Introduction

>> The last few days, Doug Berg had become more and more uneasy. He was 65 years old and had retired 2 years ago. He enjoyed his free days and the time he spent with his wife. Many afternoons he was with his grandchildren, whom he picked up from kindergarten. This morning he woke up very early and had hardly slept at all. He knew from the suitcase packed in the corner that this was not a dream. He had to go in for his surgery today, and he couldn't postpone the appointment again. After a light breakfast with his wife and after taking his daily medications, he arrived at the hospital on time. His room wasn't yet ready for him, but the nurses said they had a lot of appointments planned for him anyway. With a heavy heart, Doug Berg said goodbye to his wife, trying to look as optimistic as he could for her sake. Then his hospital stay began.

Nurse Rose drew blood and measured vital signs; blood pressure was 150/85 mmHg. "Fairly well-controlled hypertension," she commented.

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Doug Berg sighed discontentedly. About 6 years ago, he had begun having pain in his legs after walking long distances. The diagnosis was peripheral vascular disease. Because the pain intensified over time, various vascular surgeries had been necessary: a percutaneous thromboangioplasty, stent placement in the left superficial femoral artery and in the right common iliac artery, as well as a patch angioplasty in the right femoral bifurcation.

Mr. Berg could not even remember all the operations with strange names. For this reason, he had made himself a long list with everything medically important that had happened over the past few years. He kept the list with him at all times, together with his medication list.

About 3 years ago, his family physician noticed that his abdominal aorta had enlarged. He sent Mr. Berg to a vascular surgeon, and Mr. Berg had been going to see him every other year since then. At the last visit, the aneurysm had expanded. "It could burst any time; there would be nothing anyone could do to help you," he was told. Mr. Berg knew that it was a risky operation, but he couldn't live with the daily fear of sudden death.

26.1.1 What Are the Symptoms and Signs of a Ruptured Aneurysm, and How High Is the Probability that an Aortic Aneurysm Will Rupture?

An aneurysm is a concentric or eccentric dilation of an artery, localized and often in conjunction with thinning of the vessel wall. There are three different types of aneurysms: true, false, and dissecting.

- In a true aneurysm, the arterial wall remains intact. Depending on the form of the bulge, it can be termed saccular or fusiform.
- In a dissecting aneurysm, the individual layers of the wall around the media are split so that two vessel lumens are formed. Principally, the ubiquitous structures of the vessel separate the two lumens.
- The pseudoaneurysm (aneurysm spurium) is most often caused by trauma and forms outside the arterial wall. It does not have complete communication with the artery.

An aneurysm could occur in any artery, but the distribution varies. Abdominal aortic aneurysms are the most common type of aneurysms of the aorta.

The most common cause for a true aneurysm in the area of the abdominal aorta is degeneration of the elastic and collagen fibers in the media of the vessel; an imbalance of collagen breakdown and regeneration is suspected. Furthermore, inflammatory processes play a role. The mechanisms of vessel degeneration in aneurysm differ, at least partly from classic atherosclerotic processes. The identical risk factors apply to both conditions, and there is a strong relationship between aneurysms and atherosclerosis [4]. The cause of the peripheral vessel aneurysm is not arteriosclerosis, apart from the cerebral vessel pathologies. Significantly lower on the list are ascending aortic and aortic arch aneurysms, mycotic infectious arteritis (peripheral artery) aneurysms, as well as inborn and poststenotic aneurysms in patients with peripheral vascular disease.

Many of the aortic wall changes exhibit no symptoms. If they become symptomatic, the symptoms arise from irritation of the surrounding structures and diffuse abdominal pain, local thrombosis, or passage of emboli into the periphery.

The most dangerous complication of an aneurysm is life-threatening rupture. The rupture is usually accompanied by severe pain, beginning in the abdomen or thorax and radiating to the back. The pain is often confused with an acute myocardial infarction or an acute abdomen. Regardless of whether or not the perforation is open or covered, the symptoms include dizziness, loss of consciousness, and acute or prolonged shock. Aneurysm rupture has a high risk of mortality.

The most important risk factor for aortic rupture is the maximum diameter of the aneurysm. As a definition, an aneurysm is defined as an increase of the aortic diameter of more than 3 cm. According to the law of Laplace, the arterial wall stress increases proportionately with the aneurysmal dilation and the vessel's internal pressure. This explains why aortic aneurysms with wide lumens are most in danger of rupture. With a diameter of <4 cm, the yearly risk of rupture is minimal but increases significantly after a diameter of 5 cm. According to current literature, the chance of rupture in an aneurysm with a diameter of >5 cm is

4 %, of >6 cm is 7 %, and of >7 cm is 20 % [8]. Diameters above 5.5 cm have a rupture risk that exceeds the surgical intervention risks [10].

Additional risk factors include a rapid increase of the aneurysm diameter of >1 cm per year, female gender, hypertension, and smoking. It has been determined that an aneurysm 5.5 cm in men or 4.5–5 cm in women, which occurs in conjunction with clinical symptoms, is an indication for surgery. Aneurysms between 4.0 and 5.4 cm are followed up every 6–12 months; the therapy can be modified after individual risk factors have been considered [13].

The mortality of elective abdominal aortic surgery (open surgery) is 4.6–9.6 % [11].

>> After Doug Berg settled into his room, the surgical hospitalist Dr. Valentine arrived. He did a quick checkup and explained that an arteriogram would be done that afternoon in order to localize the aneurysm and verify the dilation. Tomorrow he would explain the surgery, and an anesthesiologist would come to explain the anesthesia.

As planned, Dr. Valentine came the next day to discuss the surgical procedures. The aneurysm was infrarenal, distal to the kidneys, and involved the branching of the aorta into smaller arteries. The plan was to insert an aortic bifurcation graft. Afterwards, Mr. Berg would go to the ICU for at least 1–2 days. Mr. Berg listened carefully and thought he had understood everything. He didn't have any more questions as he had already done a lot of research about the topic at home. He sighed after the surgeon had left the room: "Why does this have to happen to me? If only I hadn't smoked!" In order to keep from worrying, he watched TV until the anesthesiologist arrived.

Dr. Elen was an anesthesiologist in her third year of training. She had already done her ICU time, which was required for her rotation. The only things missing in her procedure book were the large abdominal and intrathoracic procedures. She was currently assigned to vascular surgery and was excited about the possibility of doing the preoperative evaluation of patients whose anesthesia she would take care of tomorrow. These were the first aortic procedures that Dr. Elen was allowed to do.

On the surgical ward, Dr. Elen first reviewed Mr. Berg's medical record on the computer. There was also a paper file of other records, and some pages fell out when she picked it up. She read that Mr. Berg had an infrarenal aortic aneurysm extending to the bifurcation. Angiography had estimated the size at 5.8 cm. In addition, the angiography report mentioned that Mr. Berg had only one kidney. Dr. Elen was surprised and quickly scrolled through the record until she found a sonography report which also noted only one kidney. However, she couldn't find any explanation for the missing kidney. "I'll ask Mr. Berg later," she thought to herself.

Mr. Berg also suffers from hypertension, peripheral vascular disease, non-insulin-dependent diabetes with peripheral polyneuropathy, and hyperlipidemia. The echocardiography was unremarkable. The following medications were on the list from the family physician.

- *Micardis plus (80 mg telmisartan, 12.5 mg hydrochlorothiazide) PO QD*
- *Clopidogrel 75 mg PO QD*
- *Glimepiride 0.5 mg PO QD*
- *Simvastatin 20 mg PO QD in evening*

The labs and the ECG were unremarkable. Dr. Elen hung the chest film on the viewer; it was also unremarkable. Satisfied with the quick review of the patient's medical records, she asked the nurse for the room number. On the way to see Mr. Berg, she started thinking about her anesthesia.

26.1.2 Which Types of Anesthesia Could Be Used for This Surgery? What Would You Choose? Explain Your Answer, According to the Current Guidelines for Neuraxial Blockade in Patients Receiving Antithrombotic Therapy!

The planned surgery requires balanced general anesthesia with extended hemodynamic monitoring, placement of an arterial line for constant blood pressure measurement, and central venous access. It must be decided on an individual basis whether or not this monitoring is sufficient for the patient or if other intraoperative monitoring is necessary. Other possible measures include intraoperative transesophageal echocardiography, a pulmonary arterial catheter, or pulse-induced contour cardiac output (PiCCO) monitoring.

Mr. Berg's PiCCO would require placement in an axillary, brachial, or radial artery, but PiCCO is beginning to be used more than pulmonary artery catheters especially for noncardiac surgery [12].

The combination of a balanced general anesthesia together with an epidural is controversial. Benefits include postoperative pain therapy and increased intraoperative perfusion of the abdomen. Disadvantages include the danger of intraoperative abdominal bleeding and hypotension, which is greatly enhanced with an epidural. This is due to sympathetic nervous system blockade and the fact that the patient's compensatory mechanisms are limited. On the other hand, this undesired effect can be partially counteracted by the constant administration of phenylephrine or vasopressin.

In Mr. Berg's case, taking clopidogrel is a contraindication for placement of an epidural. Clopidogrel acts by reducing the ADP-dependent activation of glycoprotein IIb/IIIa, thereby noncompetitively and irreversibly inhibiting platelet aggregation. This leads to a decrease in fibrin formation and platelet cross-linking.

Platelet inhibition can be demonstrated 2 h after a single oral dose of 75 mg clopidogrel. The maximum platelet inhibition effect occurs after 3–7 days, since clopidogrel is a prodrug, activated in the liver by the cytochrome P450 enzyme. Initial administration of a bolus of 300–600 mg achieves maximum effect after 12–24 h. The active metabolites are eliminated in the urine. After achieving a stable plasma level, the elimination half-life is 30–50 h, and normal platelet function returns about 6–7 days after discontinuation of clopidogrel therapy. Due to the increased risk of epidural/spinal hematoma formation during placement as well as after removal, an interval of 7 days is required between the last clopidogrel dose and placement of the block [5, 9].

The preoperative discontinuation of antiplatelet drugs must be carefully and individually assessed, especially for patients with cerebral and cardiovascular risk factors. Patients with an acute coronary syndrome, percutaneous coronary intervention, and stent placement profit substantially from dual antiplatelet therapy with acetylsalicylic acid (aspirin) and clopidogrel. In patients with coronary stents, the risk of acute stent thrombosis, myocardial infarction, and concurrent (consecutive) mortality increases with the discontinuation of dual therapy. The increased risk may even be present when the antiplatelet therapy is discontinued less than 6 days before surgery and also when the patients receive bridging therapy with heparin. Actually, patients with drug-eluting stents are at an increased risk due to the delayed and incomplete endothelial formation over the stent. The American Heart Association currently recommends placement of drug-eluting stents only when no elective surgeries are planned within the next 12 months [6]. The dual therapy should be continued for 12 months without interruption. With non-eluting stents (bare metal stents), combined therapy with clopidogrel and aspirin is recommended for at least 4 weeks. In both groups, aspirin should be given lifelong and not paused for surgery. A cardiologist should be consulted every time a question arises about platelet inhibition therapy in one of your patients.

Table 26.1 shows the recommended intervals between neuraxial regional anesthesia and antiplatelet/anticoagulation medications [5, 9]. European [5] and American [9] guidelines are mostly similar, although American guidelines do not recommend any delays in performing a block or removing a catheter for subcutaneous unfractionated heparin doses of less than 5,000 U q12h [9].

>> *When Dr. Elen arrived in the room, Mr. Berg had dosed off. She didn't want to wake him up but needed to do so to stay somewhat on schedule. Mr. Berg appeared much younger than 65 years old; he was kind and had done a good job of filling out the preoperative information form. She asked him about his physical condition and other acute illnesses, and she asked why he was missing a kidney.* "*Well*," *responded Mr. Berg*, "*that was discovered by chance during an ultrasound I had for pain in my legs. I never noticed the missing kidney, and I don't have any problems because of it. I've been told I was just born that way*." *Dr. Elen was satisfied and discussed the planned general anesthesia with extensive hemodynamic monitoring. She explained that he would go to the ICU after the surgery, but he was aware of that already.*

Table 26.1 Recommended interval between neuraxial regional anesthesia and antiplatelet/anticoagulation medications [5]

	Before puncture/catheter removal[a]	After puncture/ catheter removal[a]	Check lab values
Unfractionated heparins (prophylaxis ≤15,000 IE/day)	4 h	1 h	Platelets, if therapy >5 days
Unfractionated heparins (therapy)	IV 4–6 h SC 8–12 h	1 h	PTT (ACT), platelets
Low molecular weight heparin (prophylaxis)	12 h	4 h	Platelets if therapy >5 days
Low molecular weight heparin (therapy)	24 h	4 h	Platelets if therapy >5 days (anti-Xa)
Fondaparinux (prophylaxis, 2.5 mg/day)	36–42 h	6–12 h	(anti-Xa)
Rivaroxaban (prophylaxis, 10 mg/day)	26–30 h	4–6 h	PT
Apixaban (prophylaxis, 2.5 mg BID)	26–30 h	4–6 h	
Dabigatran (prophylaxis, 15–220 mg)	Contraindicated by the manufacturer	6 h	
Vitamin K antagonist	INR <1.4	After removal	INR
Hirudin (lepirudin, desirudin)	8–10 h	2–4 h	PTT, ECT[b]
Argatroban[c]	4 h	2 h	PTT, ECT, ACT
Acetylsalicylic acid	None	None	
Clopidogrel	7 days	After removal	
Ticlopidine	10 days	After removal	
Prasugrel	7–10 days	6 h	
Ticagrelor	5 days	6 h	
Cilostazol[c]	42 h	5 h	
NSAID	None	None	

[a]All durations listed are for patients with normal renal function
[b]Ecarin clotting time
[c]Extended interval in patients with hepatic insufficiency

26.1.3 For What Else Would You Obtain Informed Consent?

Since this type of surgery often has extensive blood loss, transfusion and related risks must be discussed. Also, informed consent should also be obtained for the urinary catheter.

Mr. Berg is scheduled to be transferred to the ICU after surgery, possibly intubated and ventilated. Furthermore, perioperative cerebral dysfunction could develop (see Sect. 18.1.2) causing him to accidentally remove various catheters or drains. Restraining devices might be needed on the ICU in order to prevent self-harm. Restraints used in acute medical care need to be ordered by a physician or another licensed health-care provider (e.g., a physician's assistant or a nurse practitioner, depending upon state law) and should be for the shortest time frame necessary. As part of informed consent, it is always best to forewarn the patient and family of this possibility.

>> *Mr. Berg shook his head in agreement, had no further questions, and signed the informed consent form.* "*We'll see each other tomorrow morning*!" *said Dr. Elen and then added as she departed,* "*Everything will go well*; *I'm sure of it*!" *Mr. Berg watched as she left*; *for the rest of the day, he watched TV without much interest.*

Dr. Elen prepared the anesthesia preoperative evaluation and orders. She ordered 4 units of packed red blood cells to be held on standby in the blood bank. For the morning of the surgery, she ordered Mr. Berg's standard medications, intentionally omitting the glimepiride. She ordered a glucose check in the morning and 1 mg of lorazepam 1 h before the scheduled surgery. After one last check, Dr. Elen was satisfied. She went to see

two other patients before reporting back to her attending anesthesiologist Dr. Eldridge.

On the morning of surgery, Mr. Berg woke up at about 4 a.m. He became quite annoying to the floor nurses. Every half hour he rang the nurses to ask if he could finally receive his sedative and to ask how much longer he would have to wait to be taken to the OR. Shortly after 6:30 a.m., he was finally in the preoperative area. Dr. Elen greeted him with a warm "Good morning!" to which he replied without much enthusiasm. She checked the patient and the OR to make sure everything was prepared to her satisfaction. Finally she nodded and said "All right then, let's get started." She took him back to the OR. The monitor showed that Mr. Berg was a little nervous, despite his lorazepam: blood pressure 185/65 mmHg and heart rate 85 beats/min. "Have you already decided on a nice dream, Mr. Berg?"

The induction was performed with propofol, fentanyl, and atracurium. After placement of the central line, the arterial line, a nasogastric tube, and lastly a urine catheter, Mr. Berg was ready for prepping 25 min later. At the first skin incision, his blood pressure was about 120/50 mmHg and heart rate 55 beats/min. Anesthesia depth was maintained with a mixture of desflurane, oxygen, and air, as well as fentanyl and atracurium. Dr. Elen checked everything again. The warming mats were hooked up, a temperature probe had been placed, and the urine collection bag was within view. She was satisfied. Anesthesia tech Heather brought an arterial blood gas (ABG) before saying that she was going to take a breakfast break. The ABG showed:

- *Hemoglobin: 11.8 g/dl (reference 12–14 g/dl)*
- *Hematocrit: 37 % (reference 37–47 %)*
- *Na^+: 131 mEq/l (reference 135–150 mEq/l)*
- *K^+: 3.5 mEq/l (reference 3.5–5.0 mEq/l)*
- *Glucose: 82 mg/dl (reference 70–120 mg/dl)*
- *Lactate: 1.0 mmol/l (reference 0.5–2.2 mmol/l)*
- *pH: 7.35 (reference 7.35–7.45)*
- *P_aO_2: 290 mmHg (reference 70–100 mmHg)*
- *P_aCO_2: 45 mmHg (reference 36–44 mmHg)*
- *HCO_3^-: 23.7 mEq/l (reference 22–26 mEq/l)*
- *BE: –0.8 mEq/l (reference ±2 mEq/l)*
- *S_aO_2: 98 % (reference 95–98 %)*

There was no reason to be worried. The arterial oxygen partial pressure was probably still elevated from the anesthesia induction and would normalize soon. Dr. Elen glanced with interest at the surgical area, in between filling out the anesthetic record. The surgeons had quickly opened the abdomen and were exploring the cavity when the blood pressure suddenly dropped to 60/35 mmHg and heart rate 130 beats/min. Dr. Elen got nervous and thought, "I hope the aneurysm didn't just burst!"

26.1.4 Are Dr. Elen's Thoughts Justified? What Are Your Differential Diagnoses?

Dr. Elen's fears are not totally unjustified (see Sect. 26.1.1). Especially with an undiagnosed perforated aortic aneurysm, reduction of intra-abdominal pressure, via muscle relaxants or laparotomy, can increase the chance of rupture. In this case, there is another more likely diagnosis: mesenteric traction syndrome. During the surgical exploration of the abdomen, mesenteric traction syndrome often appears. The cause is histamine release from the mesenteric cast cells and prostacyclin (PGI_2) release [2].

The symptoms are:

- Sudden tachycardia
- Hypotension
- Flush caused by peripheral vasodilation
- Less commonly, tachyarrhythmia which are caused by increased plasma histamine levels

Untreated, the hypotension persists for about 30 min, gradually improving; only few patients show a pronounced, prolonged hemodynamic impairment [14]. Prophylactic administration of prostaglandin synthesis inhibitors like ibuprofen, indomethacin, or diclofenac can attenuate the response. One study showed that the preoperative administration of H_1 and H_2 blockers cannot prevent the occurrence of flush but helps to maintain a stable blood pressure with reduced reliance on vasoconstrictor therapy [2]. A study comparing both therapies has not yet been carried out.

Routine prophylaxis with prostaglandin inhibitors does not seem justified since mesenteric traction syndrome does not occur in every intra-abdominal procedure and occurs in very different intensities. When the syndrome becomes

evident intraoperatively, treatment with vasopressors and IV fluids to replace the relative volume deficiency should be started. If the symptoms persist, administration of prostaglandin synthesis inhibitors may be considered. But one needs to consider that administration of ibuprofen or indomethacin for this purpose is off-label use.

>> Dr. Elen noticed that Mr. Berg's head had become bright red and then promptly returned to normal. She had seen enough mesenteric traction syndromes already. She gave Mr. Berg 1,000 ml of a crystalloid infusion and started an infusion of phenylephrine. After 10 min, the vital signs were back to normal.

Shortly before 9.00 AM, at the request of the surgeon, Dr. Tristan, she administered 4,000 U heparin IV. Two minutes later he announced that he was going to cross-clamp the aorta. She checked the blood pressure and noted 280 ml of urine on the anesthesia record. As the aorta was clamped, the blood pressure increased to 160/90 mmHg and heart rate was 50 beats/min. Dr. Elen gave 200 µg fentanyl IV, which unfortunately had no effect. The surgeon scolded, "Hey, anesthesia! It's bleeding all over the place in here. I can't see a thing – much less sew it shut! What are you doing over there? What's the systolic?" In a state of panic, Dr. Elen didn't know what she should do next. The suction container was practically overflowing with blood, and the surgeon cursed under his breath. Attending anesthesiologist Dr. Eldridge was busy in another room, anesthesia tech Heather was still away eating her breakfast, and Dr. Elen had no one in the OR to help her.

26.1.5 Was the Problem Mr. Berg or Dr. Elen? What Must Dr. Elen Do Now?

An important phase in aortic surgery is the clamping of the aorta. The closure of the aorta terminates the blood flow distal to the occlusion and increases the arterial blood pressure proximally. The resulting increase of systemic resistance leads to an increase in afterload of the left ventricle. This is usually well tolerated by a healthy heart. In the presence of heart failure, the increase in afterload can lead to a decrease in stroke volume, with a consecutive decrease in cardiac output. The increased afterload increases the wall pressure and the left ventricular load and can lead to left-sided heart failure. In addition, the myocardial oxygen requirement increases due to the increased wall stress. There is danger of myocardial ischemia and arrhythmias.

The extent of the hemodynamic changes depends on the site of cross-clamping, the left ventricular contractility, and the myocardial perfusion. In patients with a healthy heart, the left ventricular filling pressure after infrarenal clamping is usually unchanged or only slightly reduced. If insufficient coronary perfusion is present, signs of acute left heart failure may occur. With suprarenal aortic clamping, the symptoms occur much more often. The cause is the interruption of renal perfusion, which accounts for about 25 % of the cardiac output. A greater increase in left ventricular end-systolic and end-diastolic volume occurs, together with a reduced ejection fraction. Patients with severe aortoiliac occlusive disease have a less pronounced hemodynamic reaction.

In order to prevent or reduce the symptoms of aortic clamping, preventative measures must be taken. These measures include increasing the depth of anesthesia before the clamping (e.g., by increasing the volatile anesthetic) and administration of vasodilators. A suitable vasodilator is short-acting nitroglycerin given as an infusion (Case 3, Sects. 3.1.5 and 3.1.6). Other options include clevidipine, rapidly acting, short-duration calcium channel blockers [1], or, for more severe uncontrolled hypertension, sodium nitroprusside. Clevidipine is more effective than nitroglycerin in lowering blood pressure [1]. The administration should be done in advance of the clamping, to avoid blood pressure peaks. Many authors recommend nitroglycerin immediately before clamping to ease placement of the clamp on the relaxed vessel, therefore reducing slips [16]. At the very least, nitroglycerin is the first choice to reduce the afterload if signs of increased left ventricular load or myocardial ischemia appear.

The goal of the fluid therapy is a physiological filling pressure and a urine excretion of 1 ml/kg/h. To prevent clot formation in the non-perfused area, IV heparin was given shortly before aortic cross-clamping.

>> Dr. Elen thought about it briefly and then gave Mr. Berg 2 buccal puffs of nitroglycerin. The blood pressure promptly decreased. Then she remembered to stop the phenylephrine infusion and to increase the volatile anesthetic MAC. Dr. Tristan appeared to be happy with the result. Dr. Elen thought of him as a good and fast surgeon; therefore, she was especially annoyed at her own negligence.

Forty-five minutes after the clamp was set, the Y prosthesis was sewn in place. Dr. Tristan looked at the surgical area and then turned to Dr. Elen and said, "I'll open the aorta now. I hope you're ready." Dr. Elen nodded. Dr. Tristan opened the clamp, and the monitor immediately sounded: pressure 80/45 mmHg. Dr. Tristan sent her an accusing glance and mumbled under his breath. Dr. Elen reached for another 1,000 ml crystalloid bag, reduced the volatile anesthetic concentration, and quickly restarted the phenylephrine infusion. After 3 min, Mr. Berg had a normal blood pressure again.

26.1.6 Of Course You Know What the Problem Was, Don't You?

The second important phase of aortic surgery is release of the clamp. Declamping is associated with severe hypotension as the systemic resistance decreases, and relative hypovolemia occurs. The cause is the ischemic anaerobic metabolism of the lower extremities, especially the striated muscle. Accumulation of acidic metabolites leads to temporary vasomotor paralysis and vasodilation. Reactive hyperemia causes shifting of blood volumes. These conditions reinforce the reduction of systemic resistance after the aortic clamp is opened. Due to the reduction of the venous return, caused by the shifts of blood volume, a relative volume deficiency occurs, which is termed central hypovolemia syndrome.

The patients exhibit a pronounced reduction in preload, the cardiac output decreases, and the consequence is a decrease in renal, hepatic, mesenteric, and coronary perfusion. Due to the decreased perfusion of the myocardium, myocardial ischemia and related complications can result. "Declamping shock" is used to describe the more serious cases.

In order to reduce hemodynamic problems after declamping, preparation and vigilance are necessary. This includes adequate volume therapy and monitoring of cardiac output, central venous pressure, and the left ventricular filling pressure before declamping. The goal is optimal ventricular filling for the maximum cardiac output, keeping in mind that hypovolemia accentuates the decrease in cardiac output. A wedge pressure of 4–6 mmHg above the baseline value is recommended [16].

26.1.6.1 Volume Therapy for Declamping

With declamping, healthy hearts have a good correlation between changes in central venous pressure and changes in the pulmonary capillary wedge pressure. Therefore, this parameter can be used for estimating volume substitution. Target values recommended:

- CVP 7–11 mmHg
- Wedge pressure 10–15 (20) mmHg

Additional requirements for stable hemodynamics:

- Timely discontinuation of vasodilators
- Reduction of the volatile anesthetic concentration
- Slow release of the clamp by the surgeon

If a significant reduction in systemic resistance and blood pressure occurs, vasoconstrictors should be used [16].

>> Shortly thereafter, anesthesia tech Heather came into the OR, realized that the surgery had progressed nearly to completion without her, and immediately began to make excuses for her prolonged absence. "In the next OR, there was a complicated induction. I had to help while the other anesthesia tech was helping with line placements in another room…" Dr. Elen didn't say anything and just asked her to draw an ABG. Here are the results:

- *Hb*: *9.6 g/dl* (*reference 12–14 g/dl*)
- *HCT*: *30 %* (*reference 37–47 %*)
- *Na^+*: *133 mEq/l* (*reference 135–150 mEq/l*)
- *K^+*: *4.2 mEq/l* (*reference 3.5–5.0 mEq/l*)
- *Glucose*: *88 mg/dl* (*reference 70–120 mg/dl*)
- *Lactate*: *1.2 mmol/l* (*reference 0.5–2.2 mmol/l*)
- *pH*: *7.26* (*reference 7.35–7.45*)
- *P_aO_2*: *125 mmHg* (*reference 70–100 mmHg*)
- *P_aCO_2*: *63 mmHg* (*reference 36–44 mmHg*)
- *HCO_3^-*: *19.8 mEq/l* (*reference 22–26 mEq/l*)
- *BE*: *–5.6 mEq/l* (*reference ±2 mEq/l*)
- *S_aO_2*: *97 %* (*reference 95–98 %*)

26.1.7 How Would You Interpret This ABG? What Is Your Explanation?

The ABG shows a mixed metabolic and respiratory acidosis and mild anemia. This can be easily explained by the ischemia distal to the aortic clamp. Correction of the acidosis with sodium bicarbonate is seldom needed. In most cases, ventilation adjustments to accommodate the increased CO_2 after reperfusion is all that is needed to reach a normal pH value. ABGs are an essential part of the monitoring.

>> Dr. Elen finally had the feeling that she had the anesthesia back under control. Despite not having had assistance, she had overcome the difficulties of this surgery. She requested anesthesia tech Heather to stay in the OR until the surgery ended. Dr. Tristan concentrated on his work, complaining to himself "that's gonna bleed" once in a while. Mr. Berg's blood pressure could be kept stable with a little phenylephrine. Dr. Elen infused another 1,000 ml of crystalloid solution for hemodynamic stability. At about 12.30, she obtained a new ABG:

- *Hb*: *8.0 g/dl* (*reference 12–14 g/dl*)
- *HCT*: *25 %* (*reference 37–47 %*)
- *Na^+*: *131 mEq/l* (*reference 135–150 mEq/l*)
- *K^+*: *4.5 mEq/l* (*reference 3.5–5.0 mEq/l*)
- *Glucose*: *95 mg/dl* (*reference 70–120 mg/dl*)
- *Lactate*: *1.3 mmol/l* (*reference 0.5–2.2 mmol/l*)
- *pH*: *7.27* (*reference 7.35–7.45*)
- *P_aO_2*: *165 mmHg* (*reference 70–100 mmHg*)
- *P_aCO_2*: *61 mmHg* (*reference 36–44 mmHg*)
- *HCO_3^-*: *20.3 mEq/l* (*reference 22–26 mEq/l*)
- *BE*: *–5.5 mEq/l* (*reference ±2 mEq/l*)
- *S_aO_2*: *97 %* (*reference 95–98 %*)

Dr. Elen wondered why nothing much had changed since the last measurement. She increased the respiratory minute volume 20 % in order to normalize the P_aCO_2 and compensate for the metabolic acidosis. "I should have done that sooner!" she thought and was annoyed with her performance. There was 800 ml of blood in the suction collection tank, and the surgical area looked rather dry. She couldn't really determine where the blood loss in her ABG came from.

A few minutes later, her attending anesthesiologist Dr. Eldridge came in to check up on her. "So, doing well?" he asked. She nodded. Dr. Eldridge noticed how well the surgery was progressing and glanced at the anesthesia record and the monitor. He became obviously upset about something and raised his eyebrows. "Since when have you had this much phenylephrine running in?" Dr. Elen squirmed internally. Yep, she had been increasing the pressure infusion pump at regular intervals. Although she had properly documented the increases, she had somehow not realized that the dose was now unusually high. Mr. Berg was receiving phenylephrine 180 µg/min to maintain a satisfactory blood pressure and had a heart rate of 90 beats/min. "It's about time you gave 2 units of RBCs!" ordered Dr. Eldridge and disappeared again from the OR.

Dr. Elen gave anesthesia tech Heather the job of preparing everything for the blood transfusion. Heather came back a few minutes later and said that there was no blood crossmatched. Angrily, Dr. Elen drew blood and ordered 4 emergency units. After half an hour, she received a green light from the blood bank to begin the transfusion.

At about 1:20, the surgery was completed. Dr. Elen was not especially proud of herself. She brought Mr. Berg to the ICU intubated, ventilated, and hemodynamically stable. Finally, she recorded a total blood loss of 1,000 ml, urinary output of 320 ml, and phenylephrine infusion requirement of 20 µg/min. The receiving colleague on the ICU inquired about preexisting conditions, after she had reported the details of

the surgery. "*Yeah*, *the usual*," *answered Dr. Elen*, "*hypertension*, *peripheral vascular disease with various previous surgeries*, *non-insulin-dependent diabetes with peripheral polyneuropathy*, *and hyperlipidemia*."

26.1.8 Didn't Your Colleague Forget Something in Her Patient Report?

Mr. Berg's urine output was only 40 ml within the past 2 h. Perioperative renal failure is an important morbidity in aortic surgery; the ICU must be made aware of the impending renal failure.

>> *On the following days*, *Dr. Elen went to the ICU every day to see Mr. Berg. He was extubated relatively quickly*, *but urinary output never picked up*, *and he developed acute renal failure with oliguria and azotemia. She asked the ICU attending*, *Dr. Ernest*, *if that happens often*. "*Well*," *he began* "*not too often. We see more acute renal failure in suprarenal aneurysms. We've got a nephrology consultation today. He will probably need dialysis. We're keeping the pressure up and keep giving more volume. The renal ultrasound was unremarkable*; *we've been wondering if maybe there was a surgical cause. The surgeon will come by later today*."

As the surgeon Dr. Tristan came to the ICU, *he couldn't see any reason for the big fuss*. "*It is completely normal for the kidney to have quit. I had to clamp it. In a few days it will spring into action again. Everything is in the angiography report*." *And with that*, *Dr. Tristan was gone. Dr. Ernest immediately located the finding*, *and sure enough*, *he read the following*: "*Neither renal artery nor kidney present at normal anatomical position. Dystopic kidney*, *inferior portion overlying right common iliac artery. Visualization of 3 renal arteries. The first from the infrarenal aorta*, *the second from the bifurcation of the aorta*, *the third from the left common iliac artery*." *This explained the postoperative acute renal failure* (Fig. 26.1).

As the surgeon had said, *after 3 days the kidney began to function again. On the 8th day post-op*, *Mr. Berg was transferred to the ward with normal renal function*.

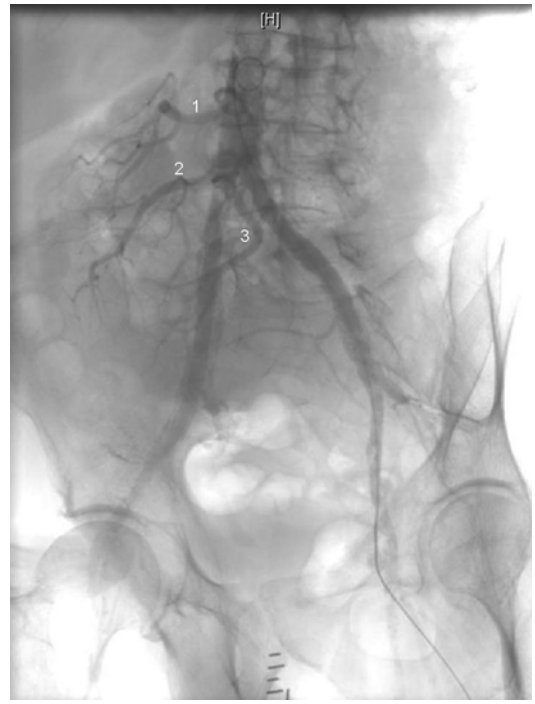

Fig. 26.1 Preoperative angiography of Mr. Berg (*1* renal artery branching from the infrarenal aorta, *2* renal artery at the level of the aortic bifurcation, *3* renal artery branching from the left common iliac). Note that the radiology definition and nomenclature "infrarenal" apply to the standard anatomical kidney position

26.1.9 Name Possible Causes of Postoperative Renal Failure and List Strategies for Prevention!

Until now, only very few studies have analyzed the usefulness of the RIFLE criteria for defining renal failure (RIFLE = risk, injury, failure, loss, and end-stage kidney disease), which makes it more difficult to compare results between studies. After abdominal aortic surgery, about 15 % of patients experience an acute renal dysfunction, defined as an increase in the serum creatinine value of >0.5 mg/dl (= 44 μmol/l) [3]; however, due to differing definitions, literature often cites the rate as between 6 and 22 %.

There are multifactorial causes for renal complications in aortic surgery. Many pathophysiological processes are involved, such as surgical manipulation, alteration of renal perfusion, nephrotoxic and inflammatory substances,

intrarenal vascular reflexes, direct mechanical trauma, embolization, atheromatous material, and the body's neuroendocrine stress response to the surgery. The infrarenal clamping results in a significant increase of the renal–vascular resistance and a decrease in renal blood flow and glomerular filtration rate, leading to decreased urinary output. The kidney's renin secretion is stimulated. Renin and angiotensin plasma levels are intra- and postoperatively increased. Apart from the listed changes, the suprarenal clamping also causes renal ischemia, which ceases the urine production for that time period. Infrarenal aortic clamping leads to transitory oliguria and impairment of renal function; very few patients, however, develop an acute renal failure.

Volume replacement therapy has the largest renal protective effect. The goal is a diuresis of 1 ml/kg body weight/h before clamping. After declamping, sufficient left ventricular filling and sufficient perfusion pressure must be maintained, and mechanical impediments must be ruled out. Usually, within 2 h, sufficient urine production returns. If the urine output remains abnormally low, diuresis can be stimulated with loop diuretics. Hypervolemia must be avoided in patients with cardiac, pulmonary, and gastrointestinal dysfunction. On the other hand, hypovolemic states in such patients significantly increase morbidity and mortality [11].

For optimal volume balance in the postoperative phase, enhanced and perhaps more invasive monitoring (PAC, PiCCO, TTE, TEE) may be indicated. If the cause of the acute renal failure is cardiac, inotropic therapy is indicated [15]. Administration of mannitol to improve kidney function is controversial. A recently published article showed that the incidence of acute renal failure is not decreased with mannitol therapy [7].

If oliguria persists despite the therapy listed above, surgical complications must be ruled out. In this case, there is an abnormal position and form of the renal arteries so that extended ischemia occurred in the kidney during the clamping, as is the case with suprarenal aneurysms. Therefore, the prolonged renal failure is the result of the intraoperative clamping of an anatomical anomaly.

26.2 Case Analysis/Debriefing

26.2.1 Which Medical Mistakes Do You See in the Presented Case?

26.2.1.1 Standby of Blood from the Blood Bank

Serious blood loss must be anticipated in every aortic surgery. The intraoperative risk of bleeding is additionally increased in this case, due to the patient's clopidogrel therapy. The anesthesia team is responsible for making sure that blood bags are actually prepared and available before the surgery begins. Ideally, they should be placed in a portable blood refrigerator in the OR shortly after incision.

26.2.1.2 Intraoperative Monitoring

Since Mr. Berg had no cardiac problems, extended hemodynamic monitoring such as a pulmonary artery catheter, PiCCO, or TEE was not necessary. However, no attention was paid to the fact that Mr. Berg had all cardiovascular risk factors. As is described above, patients are in critical danger of developing myocardial ischemia during aortic aneurysm surgery. Therefore, the continual ST segment analysis, at least leads II and V_5, should have been used, for early detection of perfusion deficits in the anterior and lateral wall of the left ventricle.

26.2.1.3 Preparations for Each Phase of the Procedure

Part of the preparation for this surgery includes setting up the infusion pumps and preparing the medications which may be needed for each phase of the procedure (e.g., vasopressors and vasodilators). The anesthesiologist must be informed about the upcoming surgical phase and, ideally, should implement measures to prevent the anticipated hemodynamic changes before they occur.

26.2.1.4 Measures to Reduce the Need for a Blood Transfusion

Due to the technicalities of the surgery and the preoperative therapy with clopidogrel, one must prepare for extensive blood loss. Therefore, using a cell saver to collect and re-transfuse the blood from the surgical area is beneficial. This technique

minimizes the patient's requirement for allogenic blood, which reduces transfusion complications and also decreases cost.

Techniques to reduce reliance on allogenic blood and blood components are briefly listed below. Some of them have only minimal effects and are therefore only seldom performed these days:

- Preoperative homologous blood and plasma donation
- Normovolemic hemodilution
- Surgical techniques which are particularly gentle on surrounding tissues
- Permissive hypotension (hypotensive resuscitation)
- Permissive perioperative anemia
- Timely discontinuation of antiplatelet therapy
- Possible administration of desmopressin

26.2.1.5 Ventilation Adjustment

The last arterial blood gas showed acidosis, which was not completely metabolic and was not treated. After declamping, acidic metabolites and waste products come into circulation from the ischemic area. The reactive hyperemia after the ischemia caused an increase in metabolism. In Mr. Berg's case, the increased arterial PCO_2 was treated by increasing ventilation. Often, this action is all that is needed to normalize the pH, thereby the vascular tone. Catecholamines have reduced efficacy in an acidic pH.

26.2.2 Which Systems Failures Can You Find in the Presented Case?

26.2.2.1 Supervision by the Attending Anesthesiologist and Presence of an Assistant

In such a high-risk surgery, a competent second person should be present during the critical phases of clamping and declamping. Only then can a timely response be guaranteed when the hemodynamic changes arise. The attending anesthesiologist Dr. Eldridge should be present during these critical phases of the procedure. The attending anesthesiologist's duty is to be aware of the knowledge and experience of the resident and to be present for critical portions of the procedure. If Dr. Eldridge was too busy, then another attending should be called to assist Dr. Elen. Also, arterial blood gas analyses should be carried out at regular intervals to guarantee detection of plasma electrolyte abnormalities. The anesthesia tech should have also stayed to help out during the critical phases. Stopping to help out in the neighboring OR was inappropriate.

Algorithms to prevent such behavior were missing at this hospital.

26.2.2.2 Information About the Exact Localization of the Aneurysm?

Preoperatively, Dr. Elen neglected to find out exactly where the aneurysm was located (see Sect. 26.2.1). She neglected to study the angiography findings or the X-ray.

26.2.3 How Could the Communication Problems Have Been Prevented?

26.2.3.1 Conducting a Careful Preoperative Evaluation

In this case, only the surgeon, not the anesthesiologist, had read the angiography report. The inexperienced anesthesiologist had realized that the patient had only one kidney but was not interested in its location and its blood supply. Such a finding has immense importance for the anesthesiologist due to the varying severity of hemodynamic changes depending on supra- or infra-aortic clamping. Furthermore, she didn't report the important information about the missing kidney to her attending Dr. Eldridge or on handoff to the ICU team.

It is important to prevent the loss of relevant information by mistakes of judgment or by inadequate knowledge to recognize the importance of this information. Even though such facts may seem irrelevant, the information should be relayed to the team for further consideration when forming a complete picture of the patient. It is easier to relay all important information and observations to your colleagues concisely during

a patient transfer and during situations in which a decision is being made.

Protecting your image of competence can create dangerous barriers to communication and may ultimately compromise your patient's safety. Avoid thoughts like "Better to not say anything at all rather than say something wrong!" or "Nothing's gonna happen!"

26.2.3.2 Communication Between the Specialties/Technical Terminology

An infrarenal aortic aneurysm is, according to the strict anatomic definition, distal to the kidney. In the presented case, the aneurysm was described as if normal anatomy was present, when in fact the single dystophic kidney had highly atypical vascular supply.

As far as renal perfusion for Mr. Berg is concerned, the aneurysm was suprarenal.

The parties involved were not aware of the differing definition of "infrarenal." Dr. Elen thought the term "infrarenal aneurysm" referred to the vascular supply. The surgical and radiology colleagues were defining the term according to the location of the orthotropic kidney. The various specialties involved in this case lacked shared terminology.

Apart from the differing definitions, the complete lack of communication between the two departments deserves severe criticism. At the beginning of the surgery in the preoperative checklist, the surgeon should have, at the very least, informed the anesthesiologist about the suprarenal clamping. That seemingly small bit of information has extensive and serious consequences. He also should have mentioned it in the checklist dealing with postoperative concerns.

The communication deficit could have been overcome if a multidisciplinary person had been assigned responsibility for all information regarding the case as well as disseminating it to all team members. This would include all relevant information about problems, tasks, and primary and secondary goals before, during, and after the surgery. Use of a formalized checklist for handoffs that include all of this information would also have helped.

26.2.3.3 Diffusion of Responsibility

Especially in critical situations, the problem of diffusion of responsibility becomes evident, as it leads to many small areas of responsibility of each specialty *without* an overview of the entire situation. If a well-qualified leader takes responsibility early enough, with the agreement of the team, the patient's care becomes complete again.

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Case 27: Dyspnea

27

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27.1 Case Introduction

>> Ms. Snyder was a feisty old lady. She lived in an idyllic house where she was born in 1915, and that's where she wanted to stay during her last days. "I will not go to a retirement home!" she always said to her daughters. Every Wednesday, Ms. Snyder met at Café Paradise with her ever-shrinking group of friends. She was on her way there when she tripped and fell. Her right leg twisted and she cried out in pain. Then everything happened so fast – a few minutes after her fall, she was in an ambulance on the way to the hospital.

In the hospital, a new face appeared every 5 min; she didn't know who was who or what was what. Someone drew her blood, the next person took her to radiology, and the next two wanted signatures. "Your leg is broken; you need an operation," said the last person to her. "We will do a spinal; it's best at your age." With her hand shaking, she signed the form even though she had not quite understood. The man dressed in green gave her an injection, and then she couldn't hold her eyes open any longer.

She woke up in a hospital room, which she shared with another woman. She had never ever slept in a room with strangers. Her daughter, Ms. Turner, was sitting next to her and held her hand. "I have to get back home!" said Ms. Snyder. "Someone will break in and steal the jewelry!" Her daughter tried to calm her. "The most important thing now is for you to regain your strength and get healthy."

The next day, Ms. Snyder was very confused and didn't want to get out of bed. She didn't

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recognize her daughter and insulted her roommate. Dr. Alexander took the patient's daughter into his office and tried to explain the symptoms to her.

27.1.1 You Recognize Ms. Snyder's Problems, Don't You? Which Complication Is She Most in Danger Of?

Ms. Snyder has the typical symptoms of postoperative delirium. Postoperative cerebral dysfunction has been extensively discussed in Case 18 (see Sect. 18.1.2).

Of all possible complications which could now arise, Ms. Snyder is in most danger of deep vein thrombosis (DVT). DVT leads to partial or complete displacement of the deep and intramuscular veins by a blood clot, which exhibits appositional growth and tends to cause pulmonary embolism. Despite effective anticoagulation, early mobilization, and the use of compression stockings, DVT and coinciding pulmonary embolism is still the most common cause of perioperative mortality [2].

27.1.2 Which Score Would You Use, If You Fear a DVT?

The individual symptoms of DVT are valuable but they are very nonspecific [4]. In asymptomatic patients, especially bedridden patients, it is better to consider the clinical probability. The Wells score is most often used [7] (Table 27.1).

With ≥2 points, the possibility of DVT is high, and further investigations should be done.

Table 27.1 Wells score

Clinical characteristic	Point value
Malignancy	1
Paralysis/immobilization of a lower extremity	1
Bedridden >3 days or extensive surgery <12 weeks earlier	1
Pain/swelling localized along the deep venous system	1
Swelling of entire leg	1
>3 cm swelling at the calf	1
Pitting edema in the symptomatic leg	1
Collateral veins	1
Previous DVT	1
Alternative diagnosis at least as likely	−2
≥2 points = high probability of DVT	

>> Ms. Turner was reassured by her discussion with Dr. Alexander. As he predicted, her mother's confusion improved over the next few days – she was almost back to normal. The wounds were healing, and her transfer to the rehabilitation clinic was arranged. The patient said, "I would like to practice walking more, but I get out of breath so fast when I walk down the hall."

Her daughter was already gone when Ms. Snyder's dyspnea got worse. She rang for the nurse. "I can't catch my breath," sputtered Ms. Snyder.

27.1.3 What Differential Diagnosis Can You Think Of?

Dyspnea is a very unspecific symptom and can have many causes [6]. When considering organic causes, it is recommended to systemically consider pulmonary and extrapulmonary causes. About 2/3 of all diseases which present with dyspnea have cardiopulmonary causes. But neurological and psychological diseases must also be brought into the differential and investigated if probable [3].

27.1.3.1 Pulmonary Causes

Pulmonary causes include ventilation, diffusion, and perfusion disorders. The ventilation disorders can be obstructive (e.g., COPD/asthma) or restrictive (e.g., pleural effusion/pneumothorax). Common examples of acute diffusion disorders are pulmonary edema, pneumonia, acute perfusion disorders, and pulmonary embolism.

27.1.3.2 Extrapulmonary Causes

The most common extrapulmonary causes of difficulty breathing are cardiac in nature. Cardiac causes include acute heart failure with decreased cardiac output and acute coronary syndrome. Post-op patients often are anemic, which worsens oxygen delivery ($\dot{D}O_2$) and therefore increases the risk of myocardial ischemia.

>> The floor nurse called the hospitalist surgeon Dr. Cassidy. "What seems to be the problem, Ms. Snyder?" he asked as he entered her room. "My right arm has been hurting since my fall, but now I can't breathe" was her answer. Dr. Cassidy was alarmed that he hadn't heard a word about this problem. He did a quick physical. "Ouch!" shrieked Ms. Snyder as he pressed on the right side of the thorax. His percussion and auscultation reinforced his suspicion.

27.1.4 You Also Know What's Wrong, Don't You?

The pain with thorax pressure speaks for a rib cage fracture, which Ms. Snyder probably obtained from the fall. Auscultation revealed quiet breath sounds, and percussion of the thorax in that area had a dull thud. In the differential, hemothorax, rib fractures, and a one-sided pleural effusion or pneumonia should be considered.

>> Dr. Cassidy was sure that Ms. Snyder had a broken rib or two and a hemothorax. The floor nurse hooked Ms. Snyder up to a portable monitor. The first measurement showed a pulse of 100/min, blood pressure of 140/80 mmHg, and S_PO_2 of 85 %. Ms. Snyder promptly received 4 l of oxygen via a nasal cannula. "Ms. Snyder," Dr. Cassidy began, turning to her, "We need to do a chest x-ray. It looks like you have broken a rib or two during the fall. The fractures may have been slowly bleeding this whole time. Due to the blood in your chest, your lungs can no longer expand to accommodate enough air. That's the reason you're having difficulty breathing."

"Am I going to die?" was her fearful response. "No, of course not," responded Dr. Cassidy. We just need to make a small cut and let the blood drain out. Don't be afraid, I'll make sure that it doesn't hurt a bit."

Dr. Cassidy left the room and called the on-call anesthesiologist Dr. Miriam, who was newly board certified. "We need to place a chest tube in the OR for an old lady with a hemothorax. She's on her way to radiology now – I'll call you when we're ready to go into the OR."

Dr. Cassidy was right. The chest X-ray showed a unilateral hemothorax and broken ribs, and Ms. Snyder was brought into the preoperative holding area. There Dr. Miriam was waiting along with CRNA Sabine. Dr. Miriam reviewed the previous anesthesia record and explained the planned anesthesia to the patient. Then CRNA Sabina took her to the OR and hooked up the monitors. Ms. Snyder was now suffering from severe shortness of breath. The monitor showed:

- *S_PO_2: 71 %*
- *HR: 120/min*
- *Sinus rhythm*
- *ST segment depression of 0.2 mV in V_2 and V_5*
- *Blood pressure: 160/90 mmHg*

27.1.5 What Do These Vital Signs Indicate?

The low S_PO_2 value is a sign of hypoxemia (see also Case 4, Sect. 4.1.11). There is a compensatory increase in heart rate to increase oxygen delivery. The hypertension is a sign that the patient is in distress. This constellation of findings with significant ST segment changes indicates a critical situation.

>> "We've got to do something fast," said Dr. Miriam to CRNA Sabine as she tightly pressed a face mask on Ms. Snyder's face. Ms. Snyder fought vehemently against the face mask, turning her head back and forth. "I am suffocating!" she cried. Despite the best efforts of the anesthesia team, the saturation only increased to 75 %. The ST segments got more depressed. Heart rate increased to 140 beats/min, and the monitor showed supraventricular extra systoles. Dr. Miriam considered her options briefly and then came to her conclusion. "We're going to induce her now!" she said to CRNA Sabine. "If we don't, she's going to have a myocardial infarction. Give her 200 µg of fentanyl, 1 mg/kg propofol, and 80 mg succinylcholine." CRNA Sabine had already prepared the injections, so the meds were quickly administered. Dr. Miriam was relieved once she got the endotracheal tube in and thought to herself, "Now I can get Ms. Snyder oxygenated again."

But contrary to her expectations, the S_PO_2 decreased to <60%. Dr. Miriam checked the ventilator settings and whispered to herself, "100% oxygen, 14 breaths per minute, tidal volume 450 ml, PEEP 6 mmHg. Everything is okay. Now, what's going on with the hemodynamics?" The first blood pressure measurement after intubation was 60/30 mmHg, heart rate had increased to 160 beats/min, and end-tidal CO_2 was 25 mmHg.

Dr. Miriam administered phenylephrine while CRNA Sabine ran in 1,000 ml of crystalloid solution, with the help of a pressure infuser. The treatments did not bring about the desired effect, and the systolic decreased to below 50 mmHg. The monitor showed several premature beats. Panic overcame Dr. Miriam. "Dammit! She's crashing!" she shouted, and then she ordered CRNA Sabine, "Give an amp of epi – stat!"

CRNA Sabine had just given the epinephrine as the door to the OR opened and Dr. Cassidy entered. He glanced at the monitor, which was sounding off with various alternating alarms. He very nearly exploded with anger. "What are you doing? Why did you intubate her?"

27.1.6 What Did the Surgeon Dr. Cassidy Mean? What Are Your Differential Diagnoses of Circulatory Depression?

Various causes come into the differential diagnosis.

27.1.6.1 Anesthetic Agent Effect

The anesthetic agents which were administered exhibit dose-dependent hemodynamic depression and can cause the symptoms presented in the case.

27.1.6.2 Hypovolemia/Anemia

After anesthesia induction and beginning positive pressure ventilation, a relative or absolute hypovolemia can manifest as decreased ejection fraction, especially in the right ventricle. Anemia with adequate intravascular volume status does not necessarily lead to cardiac depression. In the presented case, however, there were already ECG changes indicative of inadequate coronary perfusion. Such findings increase the chance that the anemia will lead to acute heart failure.

27.1.6.3 Acute Coronary Syndrome

An acute coronary syndrome, especially acute myocardial infarction, leads to acute heart failure. Coronary perfusion is made worse by the low blood pressure and the shortened diastole with tachycardia.

27.1.6.4 Pulmonary Embolism

An acute pulmonary embolism can cause the observed symptoms. The probability, however, in this situation is minimal.

27.1.6.5 Tension Pneumothorax

A pneumothorax may develop into a tension pneumothorax after beginning positive pressure ventilation, as air escapes into the pleural space. The result is acute hindrance of the venous return to the right atrium and subsequent hypotension and tachycardia. It is a life-threatening situation and immediate treatment is necessary [5].

>> *"Didn't you see the chest x-ray?" Dr. Cassidy asked the anesthesiologist. "Ms. Snyder also has a pneumothorax! Give me a large bore cannula!" Dr. Cassidy took the cannula and pointed it towards the right side of the thorax, in the third intercostal space, in the midclavicular line. Immediately, a swishing sound was heard, and Ms. Snyder's heart rate decreased by 30 beats/min. The next measured blood pressure was 120/63 mmHg, and the S_pO_2 began to increase. Dr. Miriam overcame her state of shock and was so very annoyed with herself. "That will never happen to me again!" she swore under her breath.*

Now that Ms. Snyder was in stable condition, Dr. Cassidy prepped the patient and competently inserted a chest tube and attached a drainage canister, which quickly filled up with 1.5 l of blood. All the while, he was hoping Dr. Miriam was watching closely and had now learned her lesson.

Emergence went well. Ms. Snyder was transferred to a monitored bed. "Finally I can breathe again," she said to Dr. Miriam. "Thank you so much for helping me. I thought I was going to die." Dr. Miriam felt very uncomfortable by the patient's gratitude but couldn't muster up anything to say.

After 72 h, the lungs had expanded and the blood was completely drained, and the chest tube

was removed. Ms. Snyder was looking forward to finally making it to rehab.

27.2 Case Analysis/Debriefing

27.2.1 Which Medical Errors Do You See in the Presented Case?

27.2.1.1 The Missed Thoracic Injury

The chest injury should have been diagnosed in the hospital on the day of admission. A complete physical and history was not properly done.

27.2.1.2 Placement of the Chest Drain/ Induction of General Anesthesia

The placement of a chest drain in pneumo- or hemothorax is usually done under local anesthesia, in order to reduce the chance of a tension pneumothorax, even though some authors favor placement in general anesthesia [1]. When general anesthesia is performed – for whatever reason – the surgical team must be present during induction, in order to immediately treat any tension pneumothorax which may arise. Especially with Ms. Snyder's poor condition prior to induction, the surgical team should have been present.

27.2.1.3 Preoperative Evaluation

As part of the preoperative evaluation, Ms. Snyder received a chest X-ray. The anesthesiologists never reviewed the film.

The blood loss in a hemothorax cannot be ignored. Despite the time pressure, at least one Hb should have been done. Furthermore, no one checked with the lab to see if blood was (still) crossmatched.

27.2.2 Which Systems Failures Can You Find in the Presented Case?

27.2.2.1 Placement of a Chest Drain

As a general rule, placement of a chest drain in general anesthesia must be done with the OR team standing by (see Sect. 27.2.1). Appropriate standard operating procedures to obtain this goal should be implemented.

27.2.2.2 Communication Between the Surgeon and Anesthesia Team

Dr. Cassidy gave notice that a patient would be coming in for a chest tube, but his information was minimal and vague. He didn't mention that he only wanted monitored anesthesia care. He didn't relay the important finding of free air in the thorax. The anesthesiologist never asked for more information.

Establishing set algorithms and conducting open discussion between the surgeon and anesthesiologist can avoid the drama of such situations.

27.2.3 Discussion with the Surgeon? Why?

Dr. Miriam saw that the patient was seriously ill and breathing poorly. Her algorithm as an anesthesiologist was:

> Patients who can't breathe must be ventilated.

Emotionally stressed, the anesthesiologist carried out her tasks. Since it was too late for a complete analysis of the situation, risks related to the pneumothorax were not taken into consideration as she decided upon a therapy. As a result, an inadequate treatment plan was carried out. Communication deficiencies between surgery and anesthesia, as well as no attempt by the anesthesiologist to obtain information, led to incorrect understanding of the situation and its treatment.

This case clarifies the meaning of a *shared mental model* for a team. Tasks, goals, and treatment plans are not just assumed to exist for each member, but are specifically defined. If information gets lost within the team, incomplete situational models are constructed, which can lead to inadequate assessment of the situation. Since individuals accept and interpret information differently, one can never assume that another team member possesses a similar understanding of the situation.

How can we prevent that?

The safest way to establish a shared mental model within the team is to communicate it explicitly and to make sure that your own mental model is coordinated with the team.

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Case 28: Strabismus Surgery

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28.1 Case Introduction

>> The young mother was less than enthusiastic about bringing her 5-year-old daughter to the ophthalmology clinic. Carrie's congenital convergent strabismus had to be corrected. The child was not having success in the paint and play therapy in her early childhood special education. Due to upper respiratory tract infections, the date of the surgery had been postponed twice already – but today was the day! Carrie's mother had taken her to the pediatrician this morning and, after a careful physical exam, got the OK for the strabismus surgery. Now she was waiting in the hallway of the ward for her preoperative evaluation by an anesthesiologist.

"Isn't this just great?" thought Dr. Aric, "I've been board certified 2 weeks and now they schedule me with everything under the sun." Another anesthesiologist had called in sick, and the ophthalmology clinic was desperate for help in order to get through their fully booked schedule. On top of everything today was "Strabismus Day," and another two sets of parents were waiting with their toddlers for preoperative evaluations as well.

"Just like it always is; parents come with kids on the day of the surgery for a quick hospital admission and preoperative evaluation," Dr. Aric muttered to himself before starting to work.

After reviewing the first patient's medical record, he began to feel bogged down. Dr. Aric had all the needed documents: yellow checkup report, current weight (12 kg), temperature (36.7 °C), and completed informed consent form and referral

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from pediatrician with diagnoses (*dated the previous day*):

- *Partial trisomy 18*
- *Mental retardation*
- *Muscular hypotonia*
- *Recurrent respiratory tract infections*
- *Atopic dermatitis*
- *Convergent strabismus*

28.1.1 What Implications Does This Diagnosis Have for the Anesthetic?

28.1.1.1 Partial Trisomy 18

If separation of each haploid chromosome of a normal egg cell does not occur (nondisjunction), fertilization by a normal haploid sperm cell causes trisomy. Autosomal and gonosomal trisomy is possible. Most trisomies end as spontaneous abortion before the 12th gestational week.

Trisomy 18, also known as Edward's syndrome, was first described in 1960 by John H. Edwards from Britain. It is clinically important because it is one of four autosomal trisomies that are capable of completing intrauterine development. Trisomy 18 is the second most common trisomy, after trisomy 21. Trisomy 13 (Pätau syndrome) and trisomy 22 follow. Trisomy 18 has 3 copies of chromosome 18 in every cell, instead of 2, resulting in 47 chromosomes, instead of 46.

The incidence of trisomy 18 is 1:3,000–8,000 living births, with a very high rate of prenatal death. The chance of surviving 1 year is <10 %. Almost 100 % have congenital heart malformations, craniofacial dysmorphia, severe central nervous system defects, psychomotor and physical developmental delays, and renal malformations. Clinically similar forms are Nager syndrome and Pierre Robin syndrome. In <5 %, a partial trisomy occurs, as a translocation which can be inherited and where extra chromosomes are only present in a portion of cells. The clinical phenotype is less severe with better chances of survival [4].

28.1.1.2 General Mental Retardation

In 100 % of trisomy 18 patients, there is a general mental retardation of some degree [7]. Additionally, there are arterial and venous malformations and cerebral and cerebellar defects. Reduced cooperation and a reduced psychosocial bonding are to be expected.

28.1.1.3 Muscular Hypotonia

The large number of various myopathy syndromes (about 500 different types) reflect the many different pathological mechanisms which can be the cause of the muscular hypotonia. Clinically, muscle weakness or abnormal muscle tone is present. The pathological muscle tone is expressed as either spastic paralysis, muscular hypotonia, hyper- or hyporeflexia, tonic clonic, or spastic movements. Ophthalmic abnormalities due to strabismus may be present. When pharyngeal muscles are affected, there is dysphagia and an increased risk of aspiration.

There is increased sensitivity for nondepolarizing muscle relaxants, which should therefore be avoided. Not all myopathies are clearly associated with malignant hyperthermia. However, it is generally recommended to avoid trigger substances.

Muscular hypotonia is typical for trisomy 18.

28.1.1.4 Recurrent Bronchitis

The syndrome includes pulmonary artery hypoplasia and atypical lung lobes, as well as esophageal atresia with or without a tracheoesophageal fistula. Due to the muscular hypotension, dysphagia with consecutive microaspiration is often present.

As the complications of upper respiratory infection are easily avoided and managed, only clinically manifested upper airway infections with fever above 38.5 °C, feelings of malaise, or infected sputum or nasal secretions are criteria to postpone elective surgery [8].

28.1.1.5 Atopic Dermatitis

Atopic dermatitis is an oversensitivity type of skin disorder, occurring in localized scaly and itchy patches, and usually appears in early childhood. In 80 % of the cases, immunological changes in the form of polyvalent type 1 sensibility reactions are present. Allergic asthma and allergic rhinitis are also atopic disorders [2].

28.1.1.6 Strabismus Convergens

Strabismus surgery is the most common eye surgery in pediatrics and is usually done during preschool age. A maximum of two muscles per eye are shortened and reinserted. The surgery takes about 45–90 min. There is no definite association with muscular dystrophy; however, children suffering from strabismus are at risk, on a collective basis, for malignant hyperthermia [6]. The incidence is approximately 1:25 when succinylcholine and halothane are used.

>> Dr. Aric was concentrating hard to recall information about partial trisomy 18. "What should I watch out for? Intubation problems? Cardiac malformations? Association with malignant hyperthermia? Contraindication for premedication with midazolam?" He simply couldn't remember.

After an in-depth conversation with Carrie's mother, and after studying the patient records, Dr. Aric could exclude serious structural heart defects and acute bronchopulmonary infection. Assessment of the airway didn't show evidence of a possible difficult airway. As a preop sedative, he ordered 6 mg of midazolam rectally. He was going to plan the remaining anesthetic after taking a peek in his "pocket anesthesia" book.

The second patient of the day was 14 years old, was scheduled for a recurrent chalazion excision, and was already cleared for surgery during her preadmission anesthesia consult. The child had already been called for the OR.

The chalazion surgery was under general anesthesia with a laryngeal mask airway and proceeded without incident. Dr. Aric had shut off the sevoflurane a bit too late, so it took a while before the child's airway reflexes were sufficient enough for extubation.

28.1.2 Which Form of Anesthesia Would You Choose for Carrie?

The surgery will be done under general anesthesia. Endotracheal intubation or laryngeal mask airways are acceptable techniques. The accessibility of the airway is limited after the surgical drapes are placed. A trigger-free anesthesia is recommended.

>> Carrie was scheduled next. The ward had just called reporting her heightened agitation, despite the fact that she had received 6 mg midazolam rectally.

28.1.3 What Reasons Can You Name for an Insufficient Effect of Rectal Sedation?

Generally, good sedation is obtained 15–20 min after rectal administration of 0.5–0.75 mg/kg body weight midazolam. The absorption, however, is difficult to predict and is dependent on:

- Application form (suppository, liquid)
- Presence of feces in rectum
- Induced bowel movement
- Depth of application

With higher placement of the suppository, the corpus cavernosum recti drains via the portal vein, leading to a significant first-pass effect.

A common mistake in rectal suppositories is holding the legs up and elevating the pelvis after administration. The result is that a large part of the suppository is absorbed into the superior rectal veins, which drains into the portal vein, resulting in the mentioned first-pass effect [5, 9].

>> While Dr. Aric was caring for the first patient in the PACU, CRNA Susan was preparing everything for a trigger-free anesthesia for Carrie. The chief ophthalmologist was getting irritated while waiting and was counting the minutes it took to get the next patient into the OR. For him, this was an unnecessary delay in his schedule, and he impatiently called for Carrie without discussing it with the anesthesia team.

Shortly thereafter, Carrie appeared in the OR, fearful and tearful, squirming in the arms of an OR nurse. CRNA Susan had already removed the gas and changed the tubes and asked someone to inform Dr. Aric that Carrie was now in the OR. "Thank goodness I can see her hand veins," thought Dr. Aric, as the ophthalmologist

complained that it was already 9 a.m. and the second surgery hadn't even begun. Dr. Aric silently swore at him and gave Carrie 50 µg of fentanyl as CRNA Susan was busy preparing the laryngeal mask airway. Then induction was completed with 100 mg propofol IV.

Mask ventilation and placement of the size 2 laryngeal mask airway went without incident. "*All the questions that I had during the informed consent visit have been avoidable due to the use of a trigger-free anesthesia, lack of muscle relaxant, and use of the laryngeal airway.*" *Dr. Aric silently congratulated himself. Of course, his pocket anesthesia book didn't have a word about trisomy 18. He planned to maintain the depth of anesthesia with continual propofol infusion and fentanyl bolus administration.*

Dr. Aric monitored all hemodynamic parameters and adjusted the ventilator. He was happy that CRNA Susan had thought of the rectal thermometer. "*I would have totally forgotten that,*" *he thought to himself.*

He gave the ophthalmologist the signal that he could start, and Dr. Aric began documenting his procedures on the anesthesia record. He had hardly begun his documentation when the intervals lengthened dramatically between beeps of the monitor, and bradycardia began to blink on the screen. Carrie's heart rate was only 15 beats/min.

28.1.4 Why Did the Bradycardia Occur?

Intraoperatively, bradycardia often occurs during manipulation of the eye muscles or pressure on the bulbus. It is called the oculocardiac reflex and causes bradycardia and even asystole [3]. Other possible causes of the reflex include intraorbital injection, hematomas, acute glaucoma, or sudden stretch of the eye lid muscles. The lid reflex is referred to as blepharocardial reflex. The oculocardiac reflex is a so-called trigeminovagal reflex, being that the ophthalmic branch of the trigeminal nerve is the afferent tract and the vagus nerve is the efferent tract (Fig. 28.1).

Prevention of the oculocardiac reflex is recommended, by administering IV anticholinergics immediately before surgery. The oculocardiac reflex occurs less frequently when manipulations are done more carefully and gently. The bradycardia usually disappears when manipulations are halted.

>> Dr. Aric jumped up and yanked open the anesthesia cart drawers searching for atropine. CRNA Susan tapped his shoulder, holding up a syringe ready for use. "*We always give glycopyrrolate, but usually before the operation begins,*" *she calmly explained. Dr. Aric gave 0.1 mg IV. The ophthalmologist had already interrupted his manipulation of the eye when he heard the alarm, so after 1 min the heart rate was back to normal.*

28.1.5 With Which Anticholinergics Are You Familiar?

Anticholinergics are medications which competitively block the neurotransmitter acetylcholine on the choline receptors. There are natural (atropine, scopolamine) and synthetic derivatives (glycopyrrolate). Their clinical effect occurs at the muscarinic (M_1–M_5) postganglionic acetylcholine receptors in organs, and at the nicotinic postganglionic acetylcholine receptors at autonomic ganglia and at the motor end plate.

Standard doses hardly have any effect on the nicotinic acetylcholine receptors, so that anticholinergics are normally classified as being selective antimuscarinergic [1].

Table 28.1 compares the effects of various anticholinergic medications.

Due to the minimal central nervous and ophthalmologic effect, glycopyrrolate is used for the prevention of the oculocardiac reflex. Atropine is preferred to treat acute reflexive bradycardia due to its fast onset of action.

Especially in pediatric patients, the usual anticholinergics, even when given in the normal doses, often cause "atropine fever" due to the inhibition of perspiration.

>> Dr. Aric thought to himself: "*How embarrassing! All my concern about the trisomy while I totally forgot to review the characteristics of this*

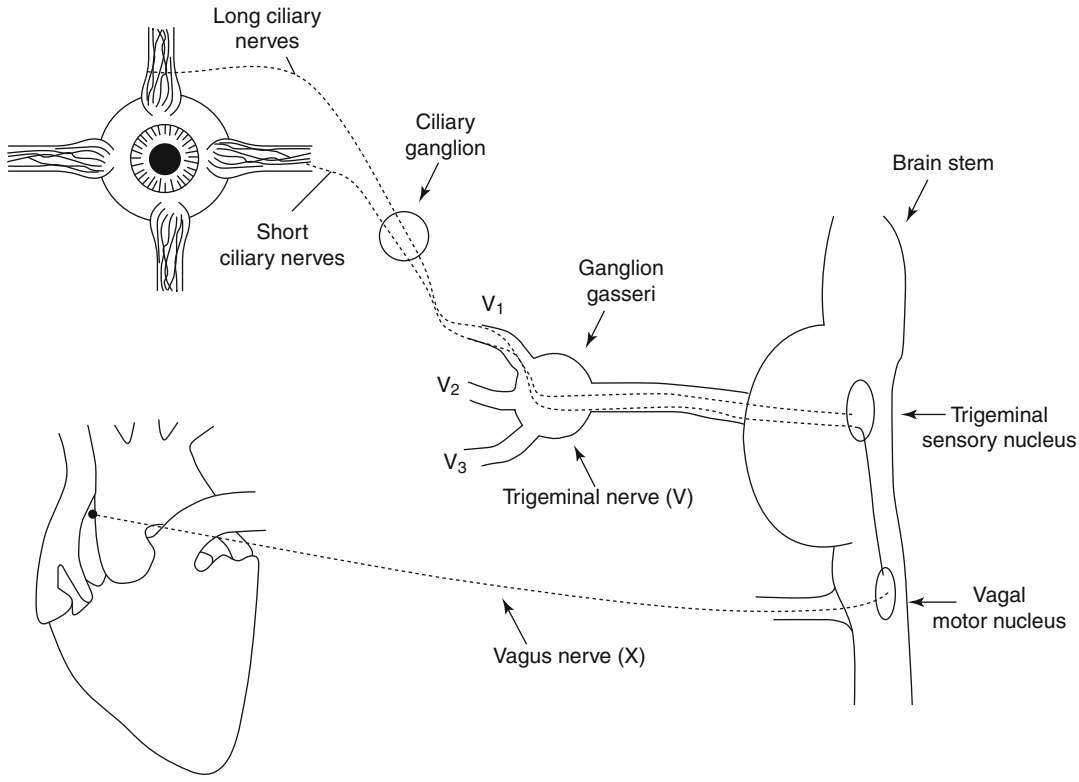

Fig. 28.1 Schematic presentation of the oculocardiac reflex. The afferents (*dotted line*) travel via the ophthalmic branch of the trigeminal nerve. After synapsing at the brain stem, the efferent impulse travels via the vagus nerve (*dashed line*)

Table 28.1 Comparison of effects of anticholinergic medications

	Atropine	Scopolamine	Glycopyrrolate
Structure	Tertiary amine	Tertiary amine	Quaternary ammonium
Solubility in lipids	+++	+++	+
Cross blood–brain barrier	+	+	0
Sedation	+	+++	0
Mydriasis/cycloplegia	+	+++	0
Increase in heart rate	+++	+	+++
Relaxation of smooth muscle cells	++	+	++
Antisialagogue effect	+	+++	++

Effect: 0 none, + mild, ++ moderate, +++ severe

surgery… and my board certification exam was only 2 weeks ago!" *He stood up at the computer screen again and completed his record.*

"*I don't think you've ever worked with us before, have you*?" *questioned the ophthalmologist. "Next time, I'll warn you in advance. The rest of my tugs on the eye shouldn't change the heart rate again*." *With that, he sunk deeply into his work. Twenty minutes later the monitor sounded an alarm again. Dr. Aric looked up to see the following on the screen*:

- *Sinus rhythm*
- *Heart rate: 110 beats/min*
- *NIBP: 187/110 mmHg*

28.1.6 What Are You Thinking About?

An increase in heart rate and blood pressure during anesthesia is often an indication of insufficient depth of anesthesia. Apart from the increasing hemodynamic parameters, signs such as sweating, tears, movements, and initiating breathing/dyssynchrony are important clues. The dose of analgesics and hypnotics given repetitively or continually are determined by pharmacodynamic and pharmacokinetic parameters, the pain caused by the procedure, as well as many individual factors. For this reason, there is not yet any fully reliable technique available to monitor anesthetic depth.

If an isolated parameter unexpectedly changes, a technical cause must always be ruled out first. Here, for example, causes could include compression of the blood pressure cuff by the surgical team, double counts of the R and T waves after a change in position of the ECG electrodes, or artifacts caused by diathermy.

>> *"The induction was already more than 30 min ago – Oh man, she's waking up! I hope the laryngeal mask airway doesn't slide up!" Dr. Aric gave 20 μg fentanyl and 60 mg propofol IV and increased the continuous propofol infusion from 180 to 250 μg/kg/min. In response, the heart rate increased to 120 beats/min, and the blood pressure didn't show any improvement.*

28.1.7 What Do You Suspect Now? What Additional Parameters Would You Check Now?

Several causes must now be checked/ruled out:

- Rule out hypoxia or an atypical form of malignant hyperthermia:
  - Temperature measurement
  - Ventilation parameters checked with endtidal CO_2
  - Respiratory minute volume
  - Oxygen saturation
  - F_iO_2
- Check the medication delivery:
  - Integrity of the intravenous access
  - Draw up new syringes of the anesthetics and rule out an accidental medication switch or incorrect medication dilution
- Rule out technical mistakes in the blood pressure measurement:
  - Check the position of the blood pressure cuff
  - Consider a contralateral blood pressure check
- Ask about medications administered intraoperatively by the surgeon

>> *The alarms and resulting bustle and burrowing under the drapes disturbed the surgeon who now commented, "—, Whatever it is this time, it's not my fault. What sort of a problem do you have?" Dr. Aric felt embarrassed; he shut off the alarms and began to carefully, but unsuccessfully, count off the possible causes of the abnormal vital signs. The rectal temperature had increased slowly since the induction, from 36.6 °C to 37.2 °C. The respiratory parameters remained stable, the IV access was intravascular, and the experienced CRNA Susan expertly demonstrated the proper dilution with each empty amp, which she always reserved until the surgery was completed. The hypertension and the tachycardia remained. CRNA Susan suggested requesting advice from a more senior colleague. "As soon as the procedure ends, this little episode will fade away," replied Dr. Aric and declined her offer to call for help.*

After an additional 10 min with a blood pressure of >180/110 mmHg and a pulse of 110/min, CRNA Susan suddenly entered the OR with flushed cheeks somewhat concealed by her face mask. She said to Dr. Aric, "I think with all the rush earlier that I forgot to change the CO_2 filter." Dr. Aric suddenly changed into a totally different mode of action. "Call the anesthesiologist-in-charge immediately. He should come ASAP – and bring dantrolene!" At the same time, Dr. Aric thought of what the anesthesiologist-in-charge had said to him, after congratulating him on his board certification: "From now on, you'll defend yourself alone in court."

A short time later, anesthesiologist-in-charge Dr. Eldridge entered the OR as the procedure was

coming to an end. Dr. Aric, as well as the ophthalmologist, was relieved to see a familiar trusted face. The hemodynamic parameters had normalized within the past few minutes. The surgeon requested eye gel and then covered the eyes with full dressings, instead of the usual clear eye patch. He explained, "*Due to the fundoscopy, both eyes will be sensitive to light for a few hours*." *Anesthesiologist-in-charge Dr. Eldridge listened intently to this comment and then asked Dr. Aric for a recap of the events. At the conclusion, Dr. Eldridge stated with conviction,* "*This is most certainly not malignant hyperthermia*!"

28.1.8 Which Other Diagnosis Does Anesthesiologist-in-Charge Dr. Eldridge Have in Mind?

In optometry, there are many topical mydriatics and cycloplegics used for fundoscopy and refraction measurements (e.g., atropine, scopolamine, cyclopentolate, tropicamide, phenylephrine). Local application of eye drops can be absorbed via the mucus membranes of the nose and conjunctiva. Systemic effects are often seen. It is recommended to compress the nasolacrimal ducts for 1 min after application, in order to inhibit the drain off and to minimize the absorption.

>> *After requesting to see the drops that were used, anesthesiologist-in-charge Dr. Eldridge was handed a bottle of phenylephrine tropicamide 10 %.*

28.1.9 What Is the Mechanism of Action of Phenylephrine Tropicamide 10 %?

Topical phenylephrine leads to mydriasis within 15 min; the duration of action is about 4 h. Side effects of the α-agonist include significant peripheral vasoconstriction and arrhythmia (possibly in combination with halothane). Solutions between 5 and 10 % can be found on the market. Only a 2.5 % solution is approved for pediatric use. Care should be used if repetitive doses are necessary. Bilateral application is forbidden.

Topical tropicamide causes mydriasis within 5–8 min; the duration of action is 4–6 h. Side effects of this anticholinergic is contact dermatitis, acute glaucoma, tachycardia, worsening of myasthenia gravis, urinary retention, and possible bradycardia in preterm neonates. A 0.5 % solution is available.

If phenylephrine must be used at all, pediatric patients should receive a combination of 2.5 % phenylephrine with 0.5–0.8 % tropicamide as the safest and most effective combination. Up to 5 % phenylephrine with tropicamide 0.5–0.8 % may be used in older children.

>> *The OR nurse instantly realized her mistake with the mix up of the adult and the pediatric eye drops. A grave sense of shock and dismay overcame the room. The problems which the anesthesia team was experiencing had not been communicated to the surgical team. In order to rule out a hypertensive retinal bleed, the dressings were removed and the fundus was inspected again. Findings were unremarkable, and the blood pressure and heart rate remained elevated but normal, so that no pharmacological intervention was needed. The emergence and the PACU time were without incident, except for two episodes of vomiting. Carrie's neurological status was unchanged postoperatively.*

28.2 Reflections on the Case

28.2.1 What Medical Errors Do You See in the Presented Case?

28.2.1.1 Preoperative Evaluation

This anesthesiologist was not familiar with the syndrome of trisomy 18 or the aspects relevant to anesthesia. Researching the syndrome was not done due to time constraints, and the potential risks were underrated.

28.2.1.2 Malignant Hyperthermia Preparation

There are clearly defined steps to reduce the risk of malignant hyperthermia. Neglecting to abide by such standards puts the life of your patient at risk. The CO_2 filter (soda lime) must be changed.

28.2.1.3 Prophylaxis of Oculocardiac Reflex

Prophylaxis of the oculocardiac reflex in strabismus surgery is currently used in most cases and helps to avoid protracted and possibly dangerous bradycardia.

28.2.1.4 Prophylaxis of PONV in Strabismus Surgery

In addition to quantification of pain, PONV is considered to be a major problem in pediatric anesthesia. Prophylaxis of the PONV in strabismus surgery is recommended (Case 4, Sect. 4.1.5).

28.2.2 What Systems Failures Can You Find in the Presented Case?

28.2.2.1 Know Your Infrastructure

Dr. Aric, the relatively inexperienced anesthesiologist, was placed in a position of responsibility because he had passed his board exams. He did not appear to have received an introduction to the specifics of pediatric anesthesia or ophthalmology in this hospital. The head of department doing the OR assignments did not know or ask Dr. Aric if he felt experienced and well trained enough to work in pediatric ophthalmology with a difficult surgeon. This could be seen as an organizational deficiency.

28.2.2.2 OR Scheduling

Despite the risk of malignant hyperthermia, this child was not given the first slot of the day. The ophthalmologist did not know that the patient might be afflicted by the very relevant syndrome of trisomy 18, and he scheduled the surgeries according to outpatient/inpatient status as usual.

28.2.2.3 Calling for Patients to the OR

Without consulting with the responsible team members, the patient was called to the OR. Carrie was brought into the OR before the anesthesiology team had finished their preparations. Even more aggravating, the anesthesiology team was not even asked if the uncooperative and undersedated child could be brought into the OR.

An optimally prepared working space is a requirement for safe pediatric anesthesia in order to avoid mistakes. Clear agreements and discussions must be carried out, even when the schedule is tight.

28.2.2.4 Algorithms and Binding Agreements

Time pressures, as in this case, are part of our daily life but can lead to avoidable mistakes (e.g., forgetting to change the soda lime). Algorithms and standard routines help to avoid mistakes during hectic activity.

28.2.3 Has It Ever Happened That You Missed Clues or Failed to Accept Help or Advice That Were Offered to You?

If you were to be honest, the answer is probably "yes." There are many motives for such behavior. In the presented case, Dr. Aric's main motive was to protect his sense of competence:

Dr. Aric was sure he could handle the situation, and he expected a prompt resolution of his problems. He concentrated on the therapy of sympathetic stimulation and did what he knew how to do which was increase the depth of anesthesia. He felt safe and secure doing so as this was his area of competence. By suppressing facts that caused him to feel insecure, i.e., signs of malignant hyperthermia, he mistakenly thought his competence was preserved.

Protecting one's sense of competence can occur consciously, for example, to justify one's position. It can occur subconsciously when one's feeling of competence is threatened. In such a case, it is very difficult for the person to consider opening the protective wall. In this case, Dr. Aric was secure in his expert competence, as long as malignant hyperthermia was ruled out. Believing that malignant hyperthermia was not even possible, he refused to accept the offer of help.

How do we know when we are unjustifiably protecting our sense of competence?

This case is a good example. Imagine the worst case scenario and then gauge your competence

to deal with it. Dr. Aric realized that he needed help after he learned that the soda lime had not been changed. At that point he could no longer deny the possibility of malignant hyperthermia.

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Case 29: Thoracic CT Scan

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Contents

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29.1 Case Introduction

>> Dr. Canute had so much to do. He was a second-year anesthesiology resident and had been working on the intensive care unit for the past few weeks. He enjoyed the work; he could deal with the challenges better than he had anticipated.

It was vacation time now, and it seemed like half the hospital staff was gone. The ICU fellow was away at a conference, and the resident who was assigned to be working with him had called in sick. So Dr. Canute was the only resident with 11 critical care patients. The attending physician came in for the morning rounds. In addition to routine work, Dr. Canute had to handle three patient transfers, two scheduled admissions from surgery, and a trip to the CT scanner. Actually, accomplishing all of this wasn't really possible. He just hoped that no emergencies would be added to the mix. He thought of

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his "crash team beeper" which he wore as the on duty ICU doc. At least his attending had promised to help out in case of catastrophes.

Just as Dr. Canute began his morning rounds with patients and considered further therapy plans, he was interrupted by a call from the OR. One of the new admissions was being transferred. As he turned back to his work, ICU nurse Sherry came from the next room and announced that the patient was ready to be taken to the CT scanner. "Radiology called and we can come now," she reported. Dr. Canute reluctantly interrupted his thoughts and followed the nurse.

The patient, Walter Peterson, was 76 years old. He had contracted pneumonia and was in acute septic shock. In addition, he had chronic COPD, hypertension, generalized peripheral vascular disease (status post several lower extremity vascular surgeries), and chronic kidney disease, compensated. Despite antibiotics and protective ventilation, neither the blood gases nor the clinical picture was improving. A thoracic CT was now planned in hopes of gaining insight into the cause of the problem.

29.1.1 How Does COPD Change Lung Morphology?

Chronic inflammatory processes in the lungs thicken the smaller airways [6]. Not only are the basal membranes affected but also the bronchial musculature of the smaller airways. Simultaneously, increased collagen expression stiffens the extracellular matrix. The loss of elasticity leads to collapse of the alveoli and causes air trapping – even with intrinsic positive end-expiratory pressure (iPEEP). The last stage includes central lobular destruction of the alveoli, which leads to the formation of bullae. These morphological changes result in increased dead space ventilation and decreased surface area for gas exchange, leading to decreased oxygenation.

29.1.2 How Should the Ventilation Be Adjusted?

A main goal of ventilation in COPD patients is to inhibit dynamic hyperinflation [10]. The necessary extended expiration time can be achieved by adjusting the inspiration to expiration ratio (I:E). The use of an extrinsic PEEP can inhibit the collapse of the small airways. If the airway resistance is high, a helium oxygen mixture (Heliox) can be used [3] – a technique not available in all facilities.

>> Dr. Canute glanced at the patient, at the transport monitor that was fixed to the head of the bed, and at the tower of syringe infusion pumps. The blood pressure was 110/80 mmHg, pulse 93/min, and S_PO_2 of 91%. Mr. Peterson was ventilated with an inspiratory oxygen concentration (FiO_2) of 60%, a PEEP of 9 cmH_2O, a peak pressure of 25 mmHg, a tidal volume of 500 ml, and a rate of 18/min. Dr. Canute turned to nurse Sherry: "For the transport, let's take the infusion pumps with midazolam, sufentanil, and norepinephrine." The latter was running at 0.5 µg/kg/min.

Nurse Sherry was edgy because she had so much to do before her shift ended. She shouldn't have been stuck with this CT transport, anyway. She waited for Dr. Canute to hook up the transport ventilator. He was studying it. "Good thing these are usually self-explanatory," he thought to himself, because he had never had an official medical device briefing. He just set it the best he could, following the ventilator settings from the ICU machine.

The pressure module on the E-cylinder oxygen tank read 1,000 psi. Dr. Canute thought of the words of his attending: "Make sure that the pressure is over 1,000 psi, before you leave with a patient." As Dr. Canute increased the FiO_2 to 100% just to be safe, ICU tech Thomas walked in and showed him an arterial blood gas of another patient who was in the weaning process. For a second, Dr. Canute wished he were in the OR again. In the OR you could concentrate on your patients without any interruptions. The arterial blood gas was satisfactory, no therapeutic intervention was needed.

29.1.3 How Long Will the Oxygen Last with These Ventilation Settings?

Boyle–Mariotte's law says that the pressure of an ideal gas at a constant temperature and constant

pressure is reversely proportional to the volume. This relationship can be used to estimate the volume of gas in a pressurized tank.

The following applies [1]:

$$t = \text{remaining time} = \frac{P_r \times V}{P_i \times Q} \qquad (29.1)$$

The formula uses pounds per square inch for pressure, volume in l, and flow rate in l/min. V_r is the volume of the E-cylinder (660 l) and P_i is the pressure when full (1,900 psi). The approximate formula is based on rounding down V_r to 600 l and using a value of 2,000 psi for P_i. This yields [1]:

$$t(\text{hours}) = \frac{P_r \times 600}{2{,}000 \times Q \times 60} = P_r / (200 \times Q) \qquad (29.2)$$

Applied to our case with 1,000 psi of pressure, this means that the tank used for transport has about 300 l of oxygen. Mr. Peterson has a respiratory minute volume of 9 l, so that the oxygen tank will theoretically last for another 33 min at these settings.

>> *The transport ventilator appeared to be working, so Dr. Canute signaled to nurse Sherry that it was time to go. She removed the syringe infusion pumps from the tower and placed them on the patient's blanket. They had gotten Mr. Peterson as far as the exit of the ICU when the alarm sounded. Nervously, Dr. Canute looked at the alarm: the blood pressure was only 60/30 mmHg.*

29.1.4 What Could Be the Cause of the Decrease?

The most likely cause of the decrease in blood pressure is the cessation of the catecholamine infusion. In order for catecholamines to be administered without being affected by the rate of other infusions, they should be run through a separate lumen of a central venous catheter.

Regardless, raising a pressure infusion syringe over the bed of a lying patient leads to administration of a bolus. The hydrostatic pressure in the infusion system and the change in height influence the released volume. Conversely, lowering a syringe to a level significantly lower than the patient's bed leads to a short but serious decrease in the infusion rate [4]. The hydrostatic pressure decreases, and the pump needs a bit of time to reestablish the internal force to increase the pressure. Figure 29.1 shows an example of such a pressure pump decrease when a pressure infusion pump is lowered.

Pressure infusion syringe pumps, which are used to administer cardiovascular medications, must always be secured at the level of the patient. This ensures that the rate of application remains the same, despite movements and repositioning. Fortunately, newer pumps now used in most institutions have safeguards to prevent positional changes in infusion rates.

>> "*Why does this always have to happen*," *commented nurse Sherry*. "*We get to the door to exit and the pressure hits rock bottom. I'll give Mr. Peterson a bolus. Usually 0.2 ml is enough*."

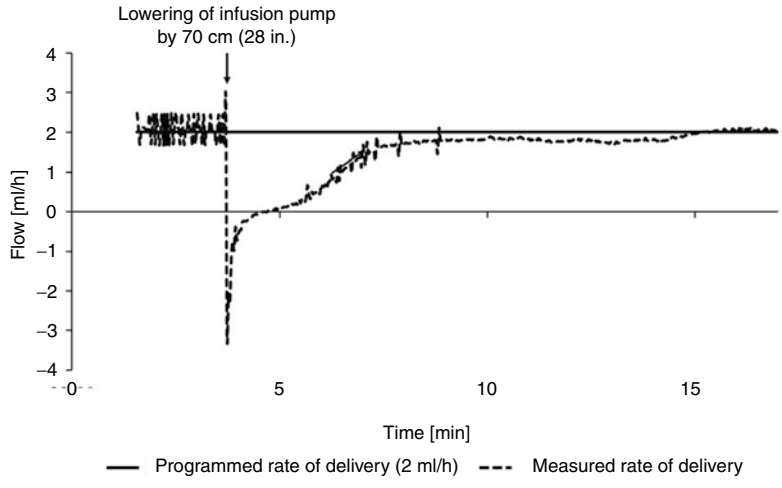

Fig. 29.1 Influence of a height decrease of 70 cm (28 in.) on the delivery rate of a pressure pump. The change in the level of the pump relative to the patient leads to a temporary (4 min) insufficient rate of administration. In a set rate of administration of 2 ml/h, this equates to a volume of about 0.2 ml

Dr. Canute was relieved that he had such a knowledgeable and helpful nurse, and Mr. Peterson's blood pressure increased promptly. As they were pushing the gurney down the long hallway, Dr. Canute realized that he forgot his emergency bag. He asked nurse Sherry to run get it. She rolled her eyes, "What for? What do you think is going to happen?" The CT scanner was only one floor below, and the obvious inexperience of Dr. Canute was beginning to get on her nerves. But she got the emergency bag for him.

They had to wait before they were allowed into the CT room. They knocked on the door a second time and the radiologist said it was going be about 10 more minutes. "Typical radiologist," thought Dr. Canute, "no clue about the clinical condition of a patient. Why did I even bother to call them to ask when we could come down?"

"OK then, I'll run back to the ward," began nurse Sherry. "I have so much to do. I'll be back here before Mr. Canute is called, I promise!" Dr. Canute hesitated, but Sherry was already on her way, so he called after her to bring a full oxygen tank with her on the way back. Due to the extended waiting period for the CT, there was a possibility that the patient's oxygen might run out.

Nurse Sherry was just out of ear shot when the monitor sounded another alarm. The systolic was only 90 mmHg. Dr. Canute examined the blood pressure curve on the monitor (Fig. 29.2) *and he knew what he had to do.*

29.1.5 What Would Your Therapy Be for the Decrease in Blood Pressure?

The blood pressure is 90/70 mmHg, which means the mean is about 75 mmHg. When examining the wave form, one sees that the amplitude is low and the incisures are missing. The most likely cause is partial obstruction of the system (see Sect. 29.1.6), for example, by a bend in the tube proximal to the pressure transducer or via obstruction of the tip of the catheter by a small blood clot. The decrease in blood pressure does not need medical therapy, only a catheter flush.

Adjustments and flushing are needed relatively often during a transport because the usual continual flushing of the system had been paused.

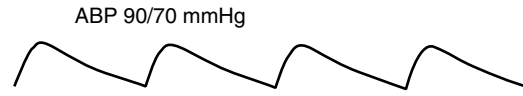

Fig. 29.2 Mr. Peterson's invasive blood pressure curve

29.1.6 What Do You Know About the Invasive Blood Pressure Damping, and How Can You Test if There Is Adequate Damping?

Every pressure system – consisting of a tube, connectors, fluids, and a pressure transducer – has its own frequency, which is termed resonance frequency. If the heart rate is close to this frequency, amplification of the measured signal can occur. As a result, the systolic value would be falsely high, and the diastolic falsely low. In order to prevent this signal amplification, pressure measurement systems have a resonance frequency which is at least 8 times larger than the frequency of the measured signal.

Here is a calculation example:

Heart rate 180 beats/min is equal to a signal frequency of 3 Hz. Eight times this value – also called the eighth overtone – is 24 Hz. The resonance frequency of the pressure system in this scenario must be >24 Hz.

Commercially available pressure measurement systems with standard short access lines have a resonance frequency which is significantly higher than the usual critical areas. Therefore, one can be assured even in extreme situations, realistic values are measured.

The clinical user can influence the resonance frequency of the system by placing exceptionally long access lines between the place of measurement and the pressure transducer. Such a change in the ratio of the oscillation of the mass to its resonance frequency can increase the amplitude.

In hospital use, underdamping as described is much less common than overdamping. Apart from the causes listed in Sect. 29.1.6, a common cause of damping is compressible air bubbles in the system. Therefore, the number of 3-way stop cocks must be kept to a minimum, due to the small air bubbles which tend to hide in them. Figure 29.3 shows how a simple test checks if the damping in a pressure system is correct.

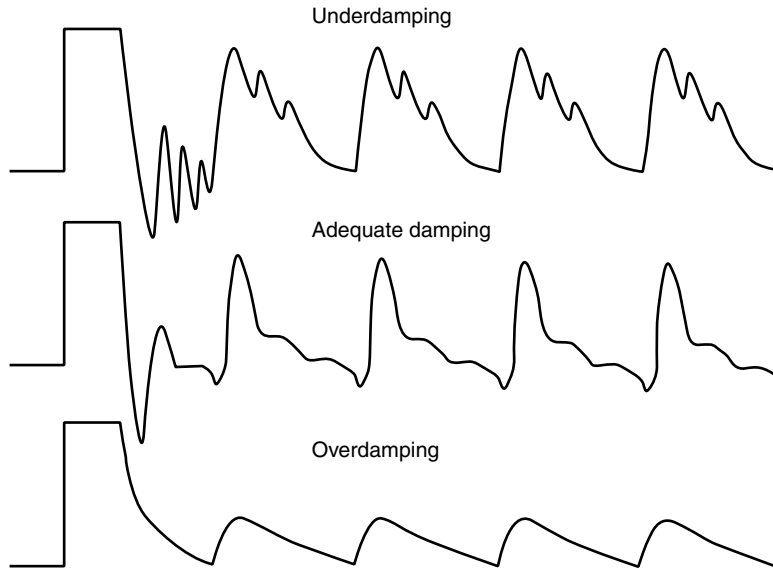

Fig. 29.3 The adequate damping of a pressure system can be checked with a short high pressure flush. With normal damping, the curve swings 1–2×, with inadequate damping it swings much more, and with excessive damping, it does not swing at all. The insufficient and the excessive damping cause incorrect systolic and diastolic readings; the mean value, however, is correct in both scenarios

>> Dr. Canute searched through Mr. Peterson's bed for the arterial transducer pressure bag. After he found it, he briefly flushed the system. Immediate success: the blood pressure curve looked normal again, at 130/55 mmHg.

Dr. Canute waited some more. The parameters on the monitor were stable, and the infusion pumps were still full enough. As he waited, he received two phone calls from the ICU.

The waiting time seemed to be very, very long. Just as Dr. Canute thought to check again with the radiologists, the monitor's alarm sounded again. The systolic blood pressure had dropped below the alarm level again. The curve form was normal this time. Dr. Canute cursed himself for not stopping nurse Sherry from leaving. The door to the radiologists was locked, and there was no one in the hallway except a patient in a wheelchair with head tilted back, closed eyes, and an open mouth emitting snoring noises. "I'd better do what the ICU nurse did, and give a bolus," thought Dr. Canute.

He searched around Mr. Peterson's feet for the norepinephrine pressure pump – which was hidden under the blanket. As he reached it, he saw that the battery had run out and it was off. "Dadgummit!" mumbled Dr. Canute who was really frustrated now. "How could that happen? I must not have heard the alarm when I was on the phone! Why was it under the blanket, anyway?"

… and the blood pressure dropped further to 50/30 mmHg.

29.1.7 What Options Does Dr. Canute Have Now?

The half-life of norepinephrine is very short, so that continual administration must be ensured immediately. In this case, the simplest solution would be to use one of the other infusion pumps. Midazolam and sufentanil can be given as a bolus.

>> Dr. Canute grabbed the pump, yanked the syringe out, and gave a bolus. Then he tried to start the infusion pump again. At the same time, he called the ICU and asked for nurse Sherry to return immediately. The pressure pump was not showing any signs of life. Dr. Canute thought hard. Sufentanil and midazolam were still running. One of those pumps he could use for the norepi, and the sedatives he could give as individual boluses. He fastened the norepi syringe into the sufentanil pump and started the infusion.

In the meantime, the blood pressure had increased to 90/65 mmHg and was still steadily increasing, actually very quickly increasing. Dr. Canute seemed to have been a little too generous with the bolus. Briefly, the pressure was 240/130 mmHg, but then slowly decreased to 115/85 mmHg. Nurse Sherry arrived out of breath as she had run up the stairs to save time. As soon as she was close enough, she checked the monitor. Everything looked normal except Dr. Canute. He looked really angry. The new oxygen

tank was still full but still upstairs on the ward as she had forgotten to bring it with her.

Dr. Canute was losing his patience and knocked on the door of the CT radiology office yet again. A very unfriendly technician appeared and said abruptly, "Another 5 min – at least!" Dr. Canute thought, "I hope this doesn't turn out to be a bad day."

Suddenly Mr. Peterson began to fight the ventilator. The pressure alarm sounded. Dr. Canute decided to change the setting to BIPAP to allow Mr. Peterson to breathe spontaneously. The new setting was well accepted.

Ten minutes later, they were allowed to enter the CT room. Transfer to the CT table went well. Dr. Canute put the ventilator beside the CT, turned the monitor toward the window, and left the room together with nurse Sherry. Following right after them was the radiology tech, who had quickly hooked up the contrast infusion pump to the CVC on her way out.

A little bit later, just as the CT scan was in full swing, everything happened at once: blood pressure and oxygen saturation hit rock bottom, and the ventilator sounded an alarm or two as well. Dr. Canute ripped open the door to the CT scanner and headed straight for his patient. Cursing could be clearly heard from the control room. The CT stopped and the telephone in Dr. Canute's pocket rang. He answered and heard a very upset nurse say that an ICU patient had removed his own trach and his breathing wasn't sounding right, and please come quick. Before Dr. Canute could respond, the nurse hung up.

Dr. Canute cursed the day. He couldn't simultaneously tackle two emergencies in different locations. He was in way over his head and all he wanted to do was say "I quit!" and leave.

29.1.8 What Would You Do if You Were Dr. Canute?

Dr. Canute is now between a rock and a hard place. The emergencies he now has to handle demand more than he is capable of. It is important to now identify individual tasks and delegate them to be done.

29.1.8.1 Falling Blood Pressure

Finding the causes and solving the problem with the CT patient's BP can be delegated to the ICU nurse Sherry. The urgency is high.

29.1.8.2 Decrease in the Peripheral Oxygen Saturation

Finding and treating the cause of the CT patient's decrease in peripheral oxygen saturation also has a very high urgency. The ICU nurse may be capable of checking the ventilator settings.

29.1.8.3 Emergency on the ICU

Here, too, the urgency is very high, but Dr. Canute can't be at two places at the same time.

>> Dr. Canute forced himself to think hard. He couldn't help the ICU patient. Dr. Canute remembered what his attending had said to him this morning, namely, that he would help in case of emergency. He yelled to the radiologist tech and told her to call his attending and say there was an emergency – he told her the phone number. "Sherry," he said, turning to the ICU nurse, "find the norepinephrine pump."

In the meantime, Mr. Peterson's saturation was 79%. Dr. Canute had found the problem – the oxygen tank was empty. "I must have misread the measurement earlier," he thought to himself. He quickly opened his emergency backpack, took the Ambu bag out, and began to bag. Mr. Peterson put up quite a fight, and not much air went in. In the meantime, nurse Sherry had found the cause for the drop in blood pressure: the contrast was hooked up to the same CVC line as the norepi, and the 3-way stop cock was closed to the norepi. With an expert bolus of norepi, nurse Sherry successfully treated the hypotension. Then Dr. Canute gave a sedative. Shortly afterward, Mr. Peterson could be ventilated again, and the S_PO_2 began to slowly climb. As Dr. Canute was bagging, nurse Sherry hooked up the transport ventilator to the central oxygen outlet on the wall. "Whew, made it out alive!" Dr. Canute exhaled as he went back into the control room. "Again, everything turned out OK."

But there was no time for a second wind. "Who was it that you wanted me to call?" asked the

radiology tech. "*I didn't catch all of that before, and I had a lot of other things to do…*"

29.2 Case Analysis/Debriefing

29.2.1 Why Didn't the Oxygen Last 33 min?

Transport ventilators have an increased gas consumption rate in BIPAP and CPAP mode, as opposed to controlled ventilation mode. The remaining tank time can no longer be calculated.

29.2.2 Why Didn't the Pressure Infuser Sound an Alarm After the Radiology Tech Closed the Outlet?

All infusion pumps have an alarm for increased pressure, such as when the tubing is occluded. In older models, the pressure settings are determined by the manufacturer and cannot be adjusted. Pressure limits of 1 bar are common. With an infusion rate of 10 ml/h, the pressure limit is reached after about 10–15 min, and at an infusion rate of 2 ml/h, it can take up to an hour [2]. In modern pumps, the pressure limits allow for variable adjustments. It is important to set the alarm individually for each medication for each patient. For instance, a low rate of application/administration of a medication with a short half-life requires a very low alarm limit.

In the presented case, there was obviously an improperly set alarm in an old style pump.

29.2.3 How Common Do You Think Such Mishaps Are During the Transport of Critically Ill Patients? What Are the Most Common Causes?

Mishaps during intrahospital transports of critically ill patients occur in about 2/3 of cases [5], a mishap being defined as the necessity to take an action to treat a problem. This emphasizes the importance of the qualifications of the person doing the transport. In highly unstable patients, more senior physicians should accompany the patient, sometimes with an ICU nurse. A respiratory therapist should accompany a ventilated patient, leaving the MD and RN free to monitor the patient and administer medications. In almost half of the cases, mechanical failures were to blame – both mechanical failures and improper use – and in one-fourth of the cases, there was a problem with an invasive line [8].

29.2.4 Which Medical Errors Do You See in the Presented Case?

29.2.4.1 Sedation/Ventilation

As Mr. Peterson's sedation was no longer sufficient to allow controlled ventilation, it should have been increased. Mr. Peterson was suffering from sepsis due to pneumonia, on top of a long history of COPD: he was receiving protective ventilation in the ICU. Even though it may be beneficial to allow spontaneous breathing in patients with severe lung disease, this is only allowed under monitoring in the ICU, not during transport. The problem of the increased gas use was discussed in Sect. 29.2.1.

29.2.4.2 Norepinephrine as a Bolus

Due to the blood pressure decrease when Dr. Canute was standing alone in the hallway with the patient, Dr. Canute gave a manual norepinephrine bolus. Exact dosing was not possible. Accordingly, he administered too much, and there was an uncontrolled increase in blood pressure. A bolus of norepinephrine should only be given in extreme cases and then only programmed as a set bolus through the infusion pump.

29.2.4.3 Hooking Up the Contrast

The radiology technician singlehandedly hooked up her contrast pump to the CVC, without informing the anesthesiologist or the nurse. She closed the 3-way valve for the norepinephrine, which lead to a critical decrease in blood pressure.

29.2.4.4 Recognition of Hypotension

Dr. Canute was first made aware of the blood pressure decrease as the transport monitor alarm went off. During a critical transport, the monitor must be positioned so that the responsible person can see the blood pressure and respond to changes before the alarm sounds.

29.2.4.5 Contrast and Renal Failure

Mr. Peterson suffered from chronic kidney disease, currently in a stage of compensated retention. Due to the administration of contrast, he was at an increased risk of developing acute renal failure. Even though studies are not completely clear [9], sufficient hydration and possibly administration of *N*-acetylcysteine is recommended [7].

29.2.4.6 Medical Device Briefing

Legally, health-care providers cannot use a device on a patient until they have been trained in the use of that device. The organization should see to it that all providers have been trained in all equipment which they need to use. In the presented case, Dr. Canute wasn't briefed on the ventilator which he was about to use, and he used it anyway. Instead, he should have called respiratory therapy to help in the transport. His action could have legal consequences.

29.2.5 Which Systems Failures Can You Find in the Presented Case?

29.2.5.1 Standards for Transport

Standard monitors for transportation include ECG monitoring, automatic blood pressure, and S_PO_2. Depending on the patient's comorbidities, other invasive measurements of blood or intracranial pressure may be needed. Capnography for ventilated patients and controlled ventilation settings are required.

For continual administration of the pharmacological therapy, battery-powered infusion devices and sufficient supplies of medications are needed. Ventilators must be suitable for the patients, and the alarms must be properly set (e.g., airway pressure, deficiencies of respiratory gases, respiratory volume, disconnection alarms). One must take care to ensure a sufficient oxygen supply. Additionally, the endpoint of the transport should have wall outlets for stationary use of the ventilator. In case of emergency, an emergency backpack should be available, with medications, venous puncture supplies, manual resuscitator, laryngoscope, various sizes of endotracheal tubes, and a suction pump. For some critically ill patients, a portable defibrillator is also indicated.

In this case, the emergency backpack was only taken after the resident specifically requested it. Obviously, there were no standards for when the bag should be taken, or the standards were ignored or forgotten. The completeness of the contents of the backpack was not fully checked by Dr. Canute. For instance, the infusion pump battery was not checked. During transport, it is important to maintain the monitor and infusion pumps in view.

29.2.5.2 Staffing of the Intensive Care Unit

During the transport to the CT, there was no physician in the ICU. The resident neglected to inform his attending of this issue. However, the attending implicitly understood the problem, because the planned CT was discussed in the morning meeting. Better options such as rescheduling the CT to a better time when more help was available, passing off the code beeper to the ICU attending, or using an anesthesia team from the OR to provide monitored anesthesia care for the CT scan were not considered. It appears that the ICU is missing algorithms about how to proceed in emergencies when physicians are not present/and who can be called for extra help.

There was an extremely large amount of work to be done in the ICU. The still relatively inexperienced resident in his second year of training was responsible for 11 critically ill patients, the code beeper, and a patient transport. Being so stressed and overworked leads straight to mistakes.

29.2.5.3 Algorithm for Procedures with Critically Ill Patients Outside of the ICU

Despite arranging the CT in advance, Dr. Canute had to wait with Mr. Peterson a long time. The

resulting problems were listed in the case. The radiology technician seemed to be unaware of the difficulties. A complete and detailed arrangement between the two departments was missing.

29.2.5.4 Hooking Up the Contrast Infusion

Non-ICU personnel may not be familiar with proper handling of infusion and infusion lines and stopcocks. For this reason, connections of additional infusions – such as the contrast – should only be done by trained ICU personnel. Furthermore, lines with cardiac medications should be well marked, to minimize the danger of accidental interruption.

29.2.5.5 Pressure Alarm on the Infusion Pump

This aspect was already discussed in Sect. 29.2.2. Ideally, use of modern infusion pumps which alarm at very low pressures or use of older infusion pumps with the adjustable pressure alarms should be used. Setting the pressure alarm must be done according to the manufacturer's instructions.

29.2.6 Which Psychological Techniques Could Help Dr. Canute to Regain Control of the Out-of-Control Situation?

Due to a series of unrelated mishaps, Dr. Canute was stressed to the limits of his knowledge and abilities. As he was standing in the middle of the radiology department, with various highly acute treatment plans which needed to be coordinated simultaneously, Dr. Canute didn't possess any strategies for delivering care in a timely manner. Luckily, he could push aside the dangerous thought process of "I can't do any more!" and regain mental control over the entire situation. The technique which he used was *critical self-reflection*.

In overtaxed, overstressed moments, critical self-reflection of one's own thoughts and feelings helps to escape from dangerous thought processes. External signals and key stimuli have a specific purpose: to support or to trigger self-reflection. Examples of signals and key stimuli include helpful hints, questioning out loud, or mental recollection out loud. The signals can be given by the person needing the self-reflection, by other team members, or by operating standards set by the organization.

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Case 30: Cerebrovascular Accident

30

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30.1 Case Introduction

>> It was a quarter to 7 in the evening, and anesthesiologist-in-charge Dr. Eldridge yawned with relief. Finally his day was beginning to relax a little. Most surgeries of the day were finished, so the late shift was looking at getting out of the hospital right on time at 8 p.m. Then the telephone rang. A neuroradiologist colleague, Dr. Dado, needed help with a patient who was already on his exam table in radiology. "The patient presented with a hemiparesis. Our suspected diagnosis is severe stenosis of the carotid artery." He went on: "We want to dilate the stenosis and insert a stent. The patient was neurologically normal, but then she suddenly stopped responding to verbal commands, and now she is becoming more and more agitated. We already tried to sedate her, which only caused a decrease in her oxygen saturation. Now we could really use your help!"

"Not a problem," replied the anesthesiologist-in-charge Dr. Eldridge, "I'll send someone over immediately."

Dr. Eldridge called an experienced colleague, a specialist in anesthesiology, who was still on duty. "Are you finished caring for your trauma patient?" "Yes, just now finished," said Dr. Berenice with a sigh of relief. "I transferred the patient to the ICU and now I'm headed to the PACU." Dr. Berenice's work day began at 8 this morning, and she was hoping to have a moment for a cup of tea now and to finish reading chapter 29 in her book "Mishaps in Anesthesiology." She

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was just at the part where Dr. Knut's ICU nurse left him alone, standing in the hallway outside of the CT room with his patient… She couldn't stand the suspense…

"*I'm sorry – but you've got to go immediately to neuroradiology*," *said Dr. Eldridge*. "*I'll send a CRNA right after you. There is a stroke patient with carotid stenosis, who needs to be dilated and stented. She'll need anesthesia for the procedure*."

Dr. Berenice sped up her stride, heading towards neuroradiology. As she arrived, she saw that CRNA Judy was already there. To her dismay, the neuroradiologist on duty was Dr. Dado, whom she had not spoken to since a very merry and boozy Christmas party during which they got just a little too intimate with one another. She decided to concentrate totally on the patient. The medical record was very thin, but the patient had only been in the hospital for 40 min.

- *Ms. Ross, 59 years old, 159 cm, about 60 kg*
- *History unobtainable*
- *Known hypertension, therapy not known*
- *Regular medication: duloxetine 60 mg 1x daily for a dissociative identity disorder*
- *No known allergies*
- *Blood pressure, 115/70 mmHg; heart rate, 95 beats/min; S_PO_2 with 4 l O_2 via nasal cannula, 90 %*
- *1 pink venous access left (18 G)*

Dr. Berenice told Ms. Ross about the planned anesthesia, but she wasn't sure if Ms. Ross understood a thing. She stared straight ahead and showed no reaction. Then Dr. Berenice turned to CRNA Judy. "*Ms. Ross has good veins – please put in another large bore IV, then we'll begin. We'll place the arterial line after induction, and skip the CVC. You know: Time is brain*!" *Turning to the radiology technician she said*, "*You can begin as soon as I've got the endotracheal tube in. I can do the arterial line from the feet*."

30.1.1 Duloxetine, What Is It?

Duloxetine is the most potent serotonin–norepinephrine (noradrenaline) reuptake inhibitor on the market. It works on the serotonin as well as the noradrenaline uptake in the central nervous system. Its main indication is for treatment of depression, panic disorders, and pain syndromes such as with diabetic neuropathy. The analgesic effect in most pain syndromes with coexisting depression has been shown to be minimal in a recent meta-analysis [6]. However, duloxetine was approved by the FDA in 2008 for specific treatment of fibromyalgia.

As with almost all centrally active substances, potentiation of the effect is to be expected when used in conjunction with common anesthetic hypnotics, sedatives, and opioids. From an anesthetic point of view, serotonin syndrome is possible, although very rare. The syndrome is characterized by hyperthermia, vegetative instability, and disorders of consciousness, perhaps even coma [5]. Therefore, substances which inhibit the reuptake of serotonin or mimic the action of serotonin should not be administered simultaneously, such as meperidine hydrochloride (Pethidine), pentazocine, and tramadol.

30.1.2 And What Is Dissociative Identity Disorder?

Dissociative identity disorder is commonly referred to as multiple personality disorder [4]. It is a psychiatric disorder in which various personalities of a person live separated in one body, each possessing its own identity. Triggers for the personalities may be abusive or traumatic events. To live with or to experience another person has a protective function for the true "me."

Affected patients react with total dissociation to gestures, facial expressions, or voices which they find threatening, referred to as dissociative stupor. When in such a stupor, they are unable to move or react to verbal stimuli but can hear everything.

30.1.3 Which Anesthetic Considerations Are Different for This Patient?

30.1.3.1 Dissociative Identity Disorder

As a basic rule in all patients with dissociative identity disorders, every physical contact – even

when it is only a reassuring touch – should be announced first. When a dissociative stupor occurs, all attempts to overcome it with medications or force are useless. The anesthesia must be initiated in a quiet area; the extubation must be done in significantly deep-enough anesthesia with spontaneous breathing. The use of ketamine is contraindicated, as are other so-called club drugs, which can trigger a dissociative episode [2].

Ms. Ross' acute cerebrovascular accident complicates her dissociative identity disorder. She will probably remain intubated and be transferred to the intensive care unit after the surgical procedure. It is important to inform the patient of the ICU plan before beginning general anesthesia, even if there is no response.

30.1.3.2 Difficult Access to the Patient due to Positioning During the Procedure

The difficult access during the intervention requires planning, so that access to the patient is assured, even after the sterile drapes and the radiology machines have been set up.

The positioning of the patient on the hard X-ray table (sometimes the edges are not padded) requires special attention. Also, excessive warming or cooling must be avoided.

30.1.3.3 Avoidance of Hypotension to Maintain Cerebral Perfusion/ Avoidance of Hypertension due to the Danger of Bleeding

Due to the severe carotid artery stenosis (of the left internal segment), perfusion of the brain occurs primarily over the vertebral artery and the right internal carotid. Autoregulation of the cerebral blood flow in the infarcted area may no longer properly function. Perfusion in this area is directly dependent on the systemic blood pressure; drops in the pressure must be avoided. Increased blood pressure is often observed directly after a stroke. Controversy exists about whether to lower the blood pressure and, if so, how and how much [3]. A target of 180 mmHg systolic and 100–105 mmHg diastolic is recommended for patients with known hypertension [1]. In the presented case, vascular intervention is planned. During the endoluminal manipulation, there in an increased danger of rupture, so the neuroradiologist will usually want the systolic kept below 160 mmHg.

30.1.3.4 Questionable Fasting State

It is assumed that Ms. Ross does not have an empty stomach. Rapid sequence induction is indicated (see Sect. 1.1.3). The acute hemiparesis is not a contraindication for succinylcholine which is the first-line medication for RSI.

30.1.3.5 Unclear Respiratory Insufficiency

A S_PO_2 value of 90 % with oxygen is not normal. There are many possible causes, but there is no time for clarification now. The lungs should be auscultated, at the least.

>> After induction, with 200 µg fentanyl, 120 mg propofol, and 80 mg succinylcholine, Dr. Berenice intubated the trachea without incident. Upon auscultation, there was significant bilateral wheezing. The peak airway pressure (Paw) was 35 cm H_2O with a tidal volume (V_T) of 430 ml. After intubation, the S_PO_2 increased to 96 %, blood pressure was 95/60 mmHg, and the heart rate decreased to 45 beats/min.

30.1.4 Which Action Would You Take Now?

Ms. Ross has two problems that often occur after induction of anesthesia.

30.1.4.1 Bronchospasm

Even though bronchospasm can have many different causes, the two most common causes are insufficient depth of anesthesia and medication-induced histamine release. To deepen the anesthesia with an inhaled anesthetic is often helpful.

30.1.4.2 Hemodynamic Changes

The hemodynamic changes are to be expected. As explained in Sect. 30.1.3, hypotension must be treated. First-line treatments include fluids and vasopressors [3].

>> Dr. Berenice opened the sevoflurane vaporizer, so that the end-expiratory concentration was 1.5 vol%, and gave Ms. Ross another 300 µg fentanyl. "Judy," she said, turning to the CRNA, "hang a bag of crystalloid. And give her a bolus of atropine and phenylephrine. When that's in, hook up a phenylephrine infusion while I do the arterial line."

The phenylephrine didn't have an effect, but the atropine caused the heart rate to increase to 120 beats/min. As the neuroradiologist began preparations, Dr. Berenice was able to cannulate the left radial artery. She felt proud of herself. "Why did I have such a hard time with that in the past?" she thought.

"The phenylephrine infusion is ready" said CRNA Judy. Dr. Berenice started it at 50 µg/min and increased it after 5 min to 100 µg/min. The blood pressure would not increase above 130/70 mmHg. S_PO_2 was now 100%, so she decreased the FiO_2 to 60%. Ms. Ross received pressure-controlled ventilation with a tidal volume of 410 ml, a rate of 10/min, an I:E ratio of 1:2, and a PEEP of 5 cm H_2O. The peak pressure was 35 cm H_2O, despite the additional fentanyl. The end-tidal CO_2 was 35 mmHg. "Alright, now I can start filling out my record." Dr. Berenice sat down in the control room behind the lead window, where she had all machines, screens, and the patient in view.

30.1.5 What Did Dr. Berenice Forget?

The high airway pressures with the limited tidal volume are not normal. The next step would be to review the effectiveness of all actions carried out, and if the bronchospasm continues, appropriate therapy should be given. The end-tidal CO_2 was normal, but because bronchospasm can lead to air trapping and an increase in dead space, an arterial blood gas should be done. This is the only way to identify hypercapnia, which would have serious consequences for Ms. Ross' cerebral blood flow.

Due to the danger of bleeding during neuroradiologic interventions, the best visual image possible is required, which requires suppressing the patients' spontaneous movements. For this reason, continued muscle relaxation, preferably with appropriate monitoring, is recommended.

>> Dr. Berenice was not finished with her documentation when CRNA Judy interrupted: "There's even less air going in now, and the pressure is dropping again." Sure enough, the tidal volume was only 350 ml, and the blood pressure was 105/60 mmHg. Only the atropine-induced tachycardia remained unchanged. "Strange," thought Dr. Berenice and asked CRNA Judy to increase the respiratory rate and the phenylephrine infusion pump and to administer a unit of albumin. The tidal volume decreased further, to 300 ml. Ms. Ross received 200 µg fentanyl in order to increase the depth of anesthesia. The desired effect did not appear. The phenylephrine dose was now 200 µg/min and seemed to have no effect whatsoever on the blood pressure.

Dr. Berenice didn't know what to do now and called the anesthesiologist-in-charge Dr. Eldridge. She was relieved as he walked through the door. "Hello! What nice surprises have you set up for me here?" Dr. Berenice reported about Ms. Ross and outlined the current problems. "I don't exactly know, but I am sure I have overlooked something important here," she said. Dr. Eldridge thought for a moment. "Let's all go through this together. If I understand you correctly, the two primary concerns of Ms. Ross are the high ventilation pressure and the low blood pressure. Are your measurements correct?" Dr. Berenice was sure that the measured values were correct.

30.1.6 What Would You Do Now?

First of all, recheck all the actions which were taken and search for a cause of the abnormalities.

30.1.6.1 Problem: Elevated Peak Airway Pressure

An elevated peak airway pressure could have a technical cause, such as endobronchial intubation or a kinked tube, or a patient cause, such as insufficient anesthetic depth, bronchospasm, pulmonary edema, pneumothorax, secretions, aspiration, etc.

The most important actions to take are:

- Check the technical components
- Auscultate the lungs
- Rule out obstruction of the endotracheal tube by endoluminal placement of a suction catheter

30.1.6.2 Problem: Low Blood Pressure

Hypotension/inadequate response to phenylephrine can have many causes. It is important to make sure that the set phenylephrine dose is actually being delivered. Afterwards, cardiac and noncardiac causes should be considered.

>> *Dr. Eldridge briefly went into the radiology treatment room and came out confirming Dr. Berenice's report. Ms. Ross had pronounced bronchospasm. The ventilation was symmetrical, the tube was patent, and the ventilator was functioning properly. The peripheral IV line was correctly positioned and the phenylephrine was running. However, as he suctioned through the tube, he encountered thick, sticky, cloudy secretions. "Have you told Dr. Dado about the blood pressure?" he asked his colleague. "And, let me see the arterial blood gas. I think Ms. Ross has pneumonia." Dr. Berenice said she had not informed Dr. Dado, but she didn't mention the reason she was avoiding the neuroradiologist.*

Dr. Dado picked up on their activity. "What's the problem?" he asked. He had to smile when they reported on the hypotension. "Let me show you something" (Fig. 30.1).

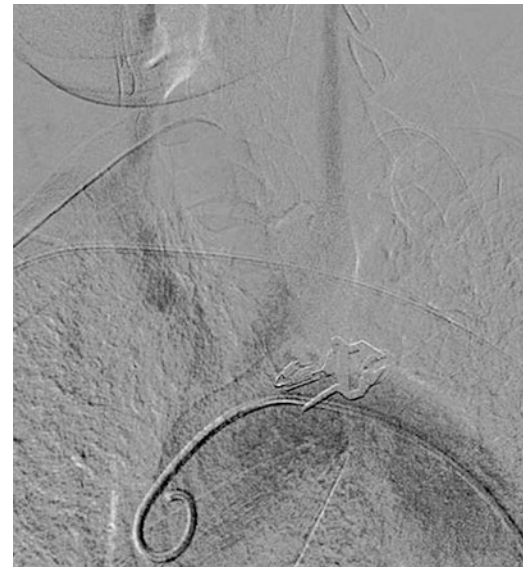

Fig. 30.1 Digital subtraction angiography (DSA)

30.1.7 What Do You See on the DSA Image?

The digital subtraction angiography shows the aortic arch with a catheter clearly visible. Further on, the branches of the left common carotid artery, the brachiocephalic trunk, the right common carotid artery, and the right subclavian artery are visible. There is no contrast in the left subclavian artery. Figure 30.2 outlines the vessels for better understanding.

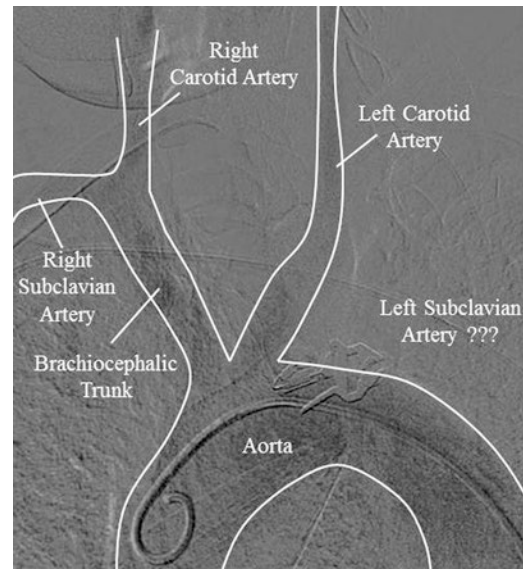

Fig. 30.2 The same image as in Fig. 30.1, with the vessels outlined

30.1.8 What Is Ms. Ross's Diagnosis?

Ms. Ross has a proximal occlusion of the left subclavian artery with a corresponding subclavian steal syndrome. Blood supply to the left arm occurs via the left and right vertebral arteries, which is blood considered to be "stolen" from the brain. The subclavian steal syndrome is often asymptomatic. When neurological symptoms occur, these appear as cerebellar symptoms [7]. The result of the occlusion is a much lower measured blood pressure on the affected arm.

>> *Dr. Berenice punctured the right radial artery. The measured blood pressure was 210/120 mmHg, and the phenylephrine was promptly reduced.*

CRNA Judy obtained an arterial blood gas in the meantime; *the stat lab showed*:

- *pH*: *7.15* (*reference 7.35–7.45*)
- *P_aO_2*: *75 mmHg* (*reference 70–100 mmHg*)
- *P_aCO_2*: *72 mmHg* (*reference 36–44 mmHg*)
- *HCO_3^-*: *17.8 mEq/l* (*reference 22–26 mEq/l*)
- *BE*: *–8 mEq/l* (*reference ±2 mEq/l*)
- *S_aO_2*: *87.5 %* (*reference 95–98 %*)
- *Lactate*: *0.6 moll/l* (*reference 0.5–2.2 moll/l*)

30.1.9 How Would You Interpret the ABG? How Large Would You Estimate the Dead Space Ventilation to Be, if the End-Tidal CO_2 Is 35 mmHg?

Ms. Ross is hypoxic, with a mixed metabolic respiratory acidosis. The relative dead space ventilation ($\dot{V}_D / \dot{V}_T$) can be calculated according to the following formula:

$$\dot{V}_D / \dot{V}_T = \left(\frac{\text{arterial}\,CO_2 - \text{end} - \text{tidal}\,CO_2}{\text{arterial}\,CO_2} \right) \quad (30.1)$$

The dead space ventilation is therefore about 49 % (reference value <35 %).

>> *Together*, *the anesthesiologists again discussed a treatment plan. Then they doubled the respiratory minute volume*, *increased the inspiratory oxygen concentration*, *and administered a bronchodilator. Afterwards*, *Ms. Ross was much easier to ventilate. In the meantime*, *Dr. Dado had successfully placed a stent*, *and preparations were made to transfer Ms. Ross to the intensive care unit*.

30.2 Case Analysis/Debriefing

30.2.1 Which Medical Mistakes Do You See in the Presented Case?

30.2.1.1 Preoperative Evaluation

Dr. Berenice walked into an emergency – she had to quickly induce general anesthesia. Despite the time pressure, a minimal preop evaluation is standard. Pulmonary and cardiac auscultations are mandatory. Apart from the pulmonary findings, she may have heard a cardiac murmur, which may have led her sooner to the diagnosis of a subclavian steal syndrome.

30.2.1.2 Monitoring

The invasive blood pressure was placed under difficult circumstances, which could have been avoided. The anesthesiologist-in-charge who was helping out ordered the neglected arterial blood gas analysis. Placement of a central venous catheter is not indicated, especially since injury of the carotid artery during puncture would be disastrous.

30.2.1.3 Preparation for Anesthesia

As mentioned in Sect. 30.1.3, close monitoring of blood pressure in stroke patients is obligatory. The phenylephrine infusion pump should have been set up before induction of anesthesia.

30.2.1.4 Checking the Measures Taken

Dr. Berenice neglected to carry out a full physical exam of her patient. Yes, the anesthesiologist she called for help later rechecked positioning and proper function of all invasive techniques: one never knows what may be found.

30.2.1.5 Anesthesia

As mentioned in Sect. 30.1.5, there was an indication for the administration of muscle relaxants.

30.2.2 Which Systems Failures Can Be Found in the Presented Case?

30.2.2.1 Discussion Between the Physicians

With all patients, especially with patients who were not evaluated in advance, a discussion of the case and findings should be done between the services before anesthesia is begun. During the sharing time, the goals and possible problems, such as the pulmonary infection and the subclavian steal syndrome, can be discussed and made known to all team members. A pre-procedure checklist should be utilized, even in nonsurgical procedure areas such as interventional radiology.

30.2.3 Do You Remember What We Discussed in the Analysis of the Last Case?

"Time is brain," Dr. Berenice had said to the CRNA. In this case, the statement was meant for two recipients, the CRNA and Dr. Berenice herself. By stating her (correct) goal, to recanalize the carotid artery as soon as possible – she took away the time she needed to rethink. At the same time, it was clear to her that she had missed something (with the increase in phenylephrine and the respiratory rate), but she couldn't mentally identify what it was. If you have been paying close attention to the end of each chapter, you will remember that a reevaluation should be done in such a case, and the best time is at the time of transition of care. By verbalizing critical points, the anesthesiologist and the radiologist can begin to think as a team.

Setting goals together, communicating with one another, taking the lead role, checking and rechecking procedures and results, and stepping back are important tools for preventing complications and mishaps in anesthesiology and in other areas as well. Try it!

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Case 31: Prostatectomy

31

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31.1 Case Introduction

>> CRNA Marid didn't feel well this morning. His head was pounding, his joints were aching, and he had a fever. He diagnosed the cause as the flu his little daughter had brought home from kindergarten. Ever since she had been going to school, his immune system had to deal with bugs that it had long since forgotten. "That's just how it goes when you get older. Or maybe I have a type of B cell Alzheimer's." He had to grin at the thought. At least the flu hadn't taken away his sense of humor. So maybe he wasn't that sick after all.

In CRNA Marid's department, several coworkers were out sick. If anyone else called in sick, surgery would have to be cancelled. CRNA Marid's helper T cells spoke to him: "You can't stay home

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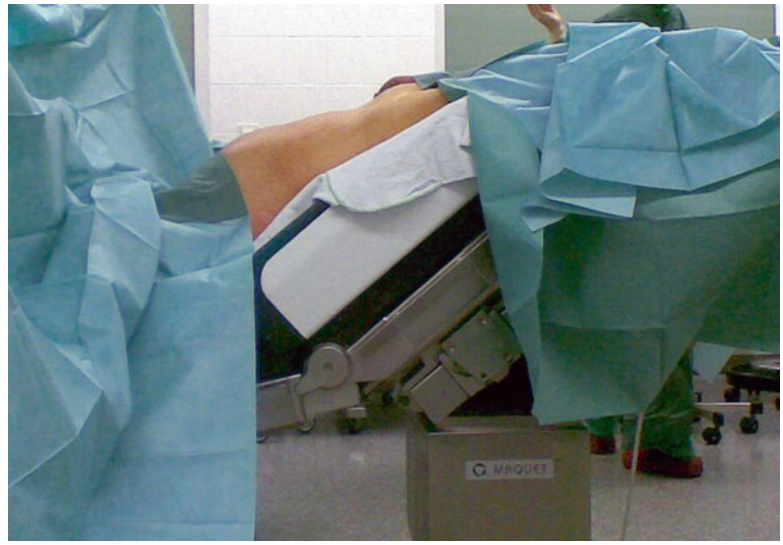

Fig. 31.1 Trendelenburg positioning for a da Vinci prostatectomy. The angle of the joint of the OR table emphasizes the extreme head-down-feet-up position. The knees are bent; legs are raised, spread, and fastened. Many clinics use shoulder pads, to prevent the patients from sliding cranially. However, the shoulder pads can cause pressure, which may cause the especially dreaded brachial plexus palsies

today! *We'll hold down the fort in here – you go do your job*!" *they whispered*! *His breakfast was a cup of black coffee*, *seasoned with 600 mg ibuprofen and 20 mg pantoprazole. Freshly showered and cruising along on his bike*, *he realized that the drugs had had the desired effect*.

Marid was an experienced CRNA. He was assigned to urology at the moment. Coming into the OR, *he wiped a little sweat from his upper lip*, *greeted student nurse anesthetist Al*, *and turned to the patient*, *Mr. Scott. Mr. Scott was planned for a robot-assisted laparoscopic prostatectomy. In hospital jargon*, *this was the* "*da Vinci prostatectomy*," *named for the robotic surgery technique*.

31.1.1 What Do You Know About da Vinci Prostatectomy?

The da Vinci prostatectomy was one of the first medical robotic techniques. Originally, this procedure was developed by the US Military, to operate on wounded soldiers or astronauts without the necessity of the surgeon's physical presence. Technological difficulties prevented its breakthrough. With the availability of improved computer techniques, further development of the field robots has continued.

The newest development is its use in laparoscopic surgery. A surgeon using a computerized telemanipulator controls a robot. The computer transfers the exact hand movements of the operator, who moves within a 3-dimensional optic to the robotic arms. Due to the assistance in orientation and maneuverability, the robotic arms can carry out minimally invasive complex operations.

31.1.2 What Must the Anesthesiologist Watch Out For?

Basically, as far as the anesthesia is concerned, there is not much difference between the da Vinci prostatectomy and a normal laparoscopic prostatectomy. Endotracheal intubation is necessary due to the pneumoperitoneum. The pneumoperitoneum is discussed in detail in Sect. 16.1.3. For surgical access it is especially important that the patient is tilted in steep Trendelenburg position (see Sect. 16.1.4) (Fig. 31.1). Due to the extreme tilt, extra care must be given to avoid injuries due to positioning.

The duration of a da Vinci prostatectomy varies according to the patient's anatomy, the type of procedure planned (e.g., a nerve sparing surgery), and the surgeon's experience. Operating room times of 4–6 h are not unusual. The physiological effects of the positioning and the capnoperitoneum can be a challenge for the anesthesiologist (Fig. 31.2). The following problems should be anticipated:

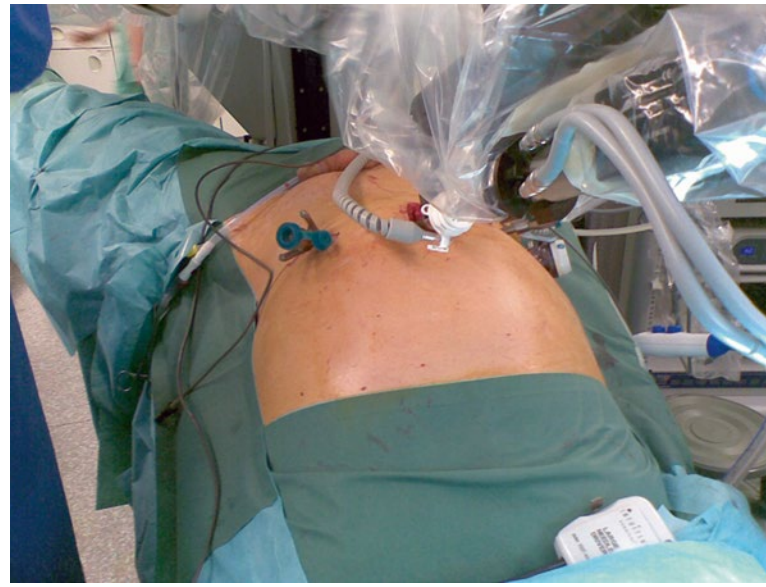

Fig. 31.2 Anesthesiologist's view of patient with a capnoperitoneum and placement of the first robotic arm. The position was not changed for the photo and highlights the steep Trendelenburg position and the massive pressure on the thorax

- Drastic reduction in functional residual capacity and total lung capacity.
- Difficulty in CO_2 elimination.
- Changes in the acid–base balance as a result of insufficient CO_2 elimination and reduced renal perfusion.
- Increase of the cerebral blood flow. The general anesthesia will be done as a balanced anesthesia; however, the choice of the anesthetics is not of primary concern. Muscle relaxation is very important, in order to minimize the effect of the extended pressure and to offer the surgeon optimal operative conditions.

>> *CRNA Marid studied Mr. Scott's medical record. The following information was noted*:

- *65-year-old patient*, *168 cm*, *85 kg*
- *Hypertension*, *therapy with metoprolol and a combination preparation with candesartan and hydrochlorothiazide. Cardiovascularly stable*
- *Indigestion after eating certain foods* (*therapy with pantoprazole*)
- *Renal insufficiency*, *in the compensated retention phase*, *with a creatinine of 1.43 mg/dl* (*reference 0.70–1.20 mg/dl*), *all other lab values unremarkable*
- *A recent admission to a neurologist due to sudden headache*

CRNA Marid stopped at that last detail and had to think about it for a minute.

31.1.3 Do You Have to Stop and Think, Too? Can You Think of Other Discoveries That May Cause You to Stop and Think?

You should take a minute to think about this, because a sudden headache can be a sign of a subarachnoid hemorrhage (see Sect. 2.1.1). As presented in Sect. 31.1.2, da Vinci prostatectomies increase the cerebral perfusion and intracranial pressure. Even when there was no previous case study reported in the literature, a cerebral artery aneurysm is a contraindication to the operation. The danger of aneurysm rupture cannot be equated with the benefits of the operative technique, especially since other techniques are available. For all other comorbidities, a risk–use analysis should be considered. Risks include:

- Glaucoma (danger of an acute glaucoma episode) [4]
- Serious pulmonary diseases, with emphysema-type changes, especially changes in the pulmonary parenchyma or formation of bullae
- Heart failure

>> After further questioning, Mr. Scott shared with Marid the tip he had received from neurologist: If the headache arises again, try to breathe fresh oxygen. "I have a little oxygen tank at home now; it stands beside my bed, but I have only had to use it once so far.

"Alright, then it's not an aneurysm – must be a cluster headache," thought CRNA Marid to himself. He had had patients with headaches that went away with oxygen, but they were all much younger than Mr. Scott.

As a preoperative sedative, Mr. Scott had received 7.5 mg PO midazolam. His usual medications had not been held. "Oh dear," thought Marid, but now he wanted to get started with the induction.

31.1.4 Would You Have Held the Usual Medications?

The discussion of preoperative ACE inhibitors and angiotensin receptor antagonists has been going around for many years. The basis of the debate is the observation that patients who take their usual dose before surgery often develop severe hypotension during surgery. Intraoperative hypotension is especially likely to occur when the patients have additionally taken a diuretic [6]. A pragmatic plan of action for this problem is to hold ACE inhibitors and angiotensin receptor antagonists when:

- Large volume redistribution may be expected.
- A volume restrictive anesthetic is planned (Case 21).
- The patient takes an additional diuretic.
- An additional indication worth considering is planned intraoperative thoracic epidural anesthesia, which almost always leads to relative hypovolemia.

An exception to this recommendation should be made when the patient's hypertension is difficult to manage and when initiating treatment again postoperatively may be difficult. Furthermore, the European Society of Cardiology recommends continuing the therapy with ACE inhibitors in patients with left ventricular systolic dysfunction [14].

>> In CRNA Marid's department, it was usual to do da Vinci prostatectomies with total intravenous anesthesia (TIVA). Student nurse anesthetist Al had already prepared the TIVA "tower" with propofol and remifentanil and started the pressure infusions. After an initial bolus of 1 mg/kg body weight propofol and 0.5 µg/kg body weight remifentanil, Mr. Scott slowly fell asleep and CRNA Marid began with the mask ventilation, which went well. Student nurse anesthetist Al turned on the twitch monitor, finally administering rocuronium 0.5 mg/kg. As they each waited for the muscle relaxant to take effect, the monitor's alarm sounded. The heart rate was only 30 beats/min.

31.1.5 You Know the Cause, Don't You?

In Mr. Scott's case, three factors have affected the heart rate:

- Bradycardia is a typical side effect of µ-agonists. The bradycardia is probably mediated by central vagus stimulation [8]. It is often seen after administration of a bolus of remifentanil.
- Propofol, depending on the dose, causes cardiovascular depression, which is usually not accompanied by reflex tachycardia.
- Mr. Scott had taken his β-blocker before surgery.

>> "Please give him 0.5 mg atropine," CRNA Marid said to student nurse anesthetist Al. "I don't want the laryngoscopy to cause asystole." The vagolytic effect appeared quickly, and intubation went without complications. "Now we've just got to complete the monitoring," thought CRNA Marid, and began preparations.

31.1.6 Which Monitoring Would You Choose Now?

The choice of perioperative monitoring should be guided by two factors:

31.1.6.1 Patient Factors

The patient's individual comorbidities must be considered independently of the surgery scheduled (see Case 9, Discussion in Sect. 9.2.1). Mr. Scott's main comorbidities include systemic hypertension, renal failure in the stage of compensated retention, and obesity.

31.1.6.2 Procedural Factors

The reverse is also true; the factors of the procedure must be independently considered, independent of the patient-specific factors. For a da Vinci prostatectomy, these factors include blood loss and ventilation problems. In addition, just as in abdominal surgery, the anesthesia is done increasingly with restrictive volume therapy, in order to reduce perioperative blood loss. Carrying out restrictive volume therapy was already discussed in Sect. 21.1.3; however, urine production and central venous pressure are not measurable during a da Vinci prostatectomy. Therefore, the decision to place a central venous line is based on patient, not procedural, characteristics. Quantitative assessment of neuromuscular blockade was already set up for Mr. Scott.

>> *CRNA Marid cannulated the left radial artery, as nurse anesthetist student Al placed 2 more large bore venous lines. CRNA Mark wiped the sweat from his face and thought, "I'm not quite healthy today; when we're finished with these preparations, I'll take a little break." Turning to the student he said, "Al, let's hook up the BIS. Come on, let's make the surgeons happy and let them begin prepping the patient*."

31.1.7 What Do You Know About BIS Monitoring?

Anesthetics have a dose-dependent effect on the synchronized electrical activity of cortical neurons. Electroencephalography (EEG) can be used for the measurement of the level of consciousness [5]. BIS monitoring (BIS = bispectral index) is a commercial neurological monitoring procedure which processes the EEG signals and transforms it into an easy-to-understand index value. The BIS value ranges from 0 (no electrical activity) to 100 (awake). During anesthesia, the desired value is between 40 and 60. The BIS monitoring can be used for individual titration of anesthetics or for automated computer-controlled anesthesia [7].

Neurological monitoring techniques for the measurement of anesthesia depth have two goals:

- Reduction of side effects by unnecessarily high doses of anesthetics
- Prevention of awareness episodes as a result of insufficient anesthetic depth.

The original hopes of neuromonitoring have only partly been realized. There have been reports that titrating anesthetics according to clinical criteria tends to lead to excessive administration, so that the neuromonitoring helps. On the other hand, some users tend to employ the BIS to maintain the anesthesia depth as light as possible, whereby the danger of an awareness episode is increased. Therefore, an awareness episode can occur during BIS monitoring [2, 3, 10]. In addition, BIS-guided anesthesia (titrated to maintain BIS levels of 40–60) is equally efficacious in reducing awareness as maintaining end-tidal gas concentrations at 0.7–1.3 age-adjusted MAC [2, 3].

A disadvantage of the technique is that there is a latency period in the index value before a change is registered. If the value increases over the threshold, this means that the patient had an insufficient depth of anesthesia for about 30 s. In order to maintain a reliable EEG signal, artifacts must be excluded. Electromyographic activity may also interfere with the EEG signal in some models.

BIS-guided anesthesia reduced the incidence of awareness compared to routine care when the anesthetic is titrated to BIS levels of 40–60 in patients at high risk for awareness [10]. In addition, a recent comparative effectiveness study demonstrated a decrease in awareness with BIS-guided anesthesia compared to routine care, especially for TIVA, as end-tidal anesthetic gas concentrations are not available [9]. Despite the weaknesses, neuromonitoring techniques are increasing year to year, and it is probably only a question of time until the costs, technical developments, and time make this technique a standard part of patient monitoring.

31.1.8 Is BIS Monitoring Indicated in This Case?

Mr. Scott is receiving general anesthesia without volatile anesthetics with simultaneous continuous muscle relaxation – therefore, he is at increased risk for awareness (see Sects. 8.1.8 and 8.1.19). The ASA practice advisory on intraoperative awareness [1] recommends use of brain functioning monitoring on a case-to-case basis, depending upon the risk of awareness. Given the absence of end-tidal gas monitoring during TIVA, use of the BIS monitor is indicated, despite its imperfections.

>> It took 30 min until CRNA Marid found a moment to responsibly excuse himself for a break. "Thank goodness I've got Al by my side today," he thought. "He's so experienced and clever, I can trust him to watch over my patient." In the OR, the two of them had together hooked up the monitoring and ventilation and carefully checked the patient positioning to make sure there would be no problems later. The urologist created a capnoperitoneum and inserted the trocars. As expected, the peak airway pressure (Paw) increased to 25 cmH_2O, even though the nerve stimulator showed a TOF value of 0. CRNA Mark had set a PEEP of 6 cmH_2O and the respiratory rate was 18/min with a tidal volume of 450 ml.

"Do you think you'll be all right for a minute without me?" he asked student nurse anesthetist Al. "I gotta get a cup of Joe to get myself movin'. I'll be in the break room when or if you need me." Student Al nodded in agreement. He valued Marid for his cautious and calm demeanor, and he had already noticed that Marid wasn't performing at his peak this morning.

On the way to the break room, Marid grabbed an ibuprofen "to help with the sweating." In the break room, he encountered his attending, Dr. Eldridge, who was immersed in a medical journal. "I only want to drink a quick cup of coffee; the patient is stable and Al is with him." CRNA Marid knew his boss didn't like patients left alone with uncertified students. "And who is watching over Al?" was his sarcastic response as he looked up. "Hey, are you OK? You look a little pale around your nose," the boss said, softening a bit. "I'm fine," said CRNA Marid, "but my little daughter has the flu. Perhaps a little of her pale color rubbed off on me."

As usual, there was no coffee made. Marid set a new pot on and, as the coffee filtered, conversed with Dr. Eldridge about his career perspectives in – and around – the hospital. This was a theme which was occupying his mind at the moment. After a little more than half an hour, he found himself in his OR again, "Anything special happen?" he asked his tech. Al replied, "No, not really. I gave more muscle relaxant, and set the remifentanil infusion higher. The urologist seems satisfied. The Paw has increased a bit, because the ventilator was truncating the inspiration. With the maximum pressure cutoff level, I increased the frequency." CRNA Marid glanced at the monitor and saw the following value:

- *Blood pressure: 92/60 mmHg*
- *Heart rate: 90 beats/min*
- *Respiratory rate: 20/min*
- *V_T: 400 ml*
- *Paw: 30 cmH_2O*
- *PEEP: 6 cmH_2O*
- *End-tidal CO_2: 63 mmHg*

31.1.9 Do You See a Problem?

You should see two problems:

The blood pressure is borderline for a patient who has hypertension, and the heart rate is relatively high, especially for a patient who took his β-blocker before the surgery. Many factors could cause this including hypervolemia, hypovolemia, anesthetic overdose, acute heart failure, etc.

The second problem is the ventilation: Mr. Scott is receiving a minute volume of 8 l. Simultaneously, the CO_2 is increased. A 63 mmHg CO_2 equates to a relative partial pressure of 8 % in room air. This means that Mr. Scott's oxygen consumption is about 8 % of 8 l, or 640 ml/min. An oxygen consumption of 7.5 ml/kg min – Mr. Scott weighs 85 kg – is unusual for a patient under general anesthesia – especially with the additional muscle relaxant. Usually an oxygen consumption of 4 ml/kg min is assumed, a value which is seldom exceeded.

Higher values are seen with states of high metabolism. Examples include sepsis, postoperative shivering, and the commonly feared, but seldom seen, malignant hyperthermia.

In the presented case, the cause is probably not increased endogenous CO_2 production, but exogenous CO_2 influx via the capnoperitoneum. The CO_2 absorption is so high that the set respiratory minute volume is not sufficient to breathe it off. The result is relative hypoventilation with an increased expiratory CO_2.

>> CRNA Marid studied the BIS monitor. "40," he thought, "Mr. Scott isn't awake. I'll give a vasopressor and optimize the ventilation." Shortly after beginning a phenylephrine infusion, the systolic increased to 110 mmHg. The blood loss was minimal so CRNA Mark saw no reason to increase the fluid administration. He set the ventilator to:

- *Respiratory rate: 26/min*
- *V_T: 450 ml*
- *PEEP: 10 cmH_2O*

He increased the inspiratory pressure limit to 35 cmH_2O. Confident that his measures would bring success shortly, he sat down on his stool and watched the surgeon perform the steps of the surgery on the screen. The mobilization of the prostate took a little extra time, being that a new surgeon was being trained in this technique, and he was at the very bottom of his learning curve.

CRNA Marid was changing a syringe in an infusion pump as the monitor sounded an alarm again: heart rate 180 beats/min. "Something's not right," he thought, as he shut off the alarm. "The pressure curve looks totally normal and regular." A rate of 90 beats/min was shown there.

31.1.10 Do You Have Any Idea What the Cause Could Be? Haven't We Had a Similar Incident Already in This Book?

Yes, of course we have had something similar in this book already, or we wouldn't even ask! Do you remember the case with the low pressure pulmonary edema (Sect. 24.1.10)? No? Here's a hint: succinylcholine. Does that lift the fog a little?

When the arterial pressure curve (or the pulse oximetry curve) shows exactly half of the actual value, a rhythm disorder could be present, which leads to a relevant and measureable output with only every second synchronized electrical myocardial excitation. Another possibility is display of double the actual value, in which case the automatic frequency recognition of the monitoring software counts each QRS complex double. A possible cause is changes in the ECG curve form – usually T wave increases.

31.1.11 What Would You Do Now?

Before you jump into action (see Sect. 24.2.3), you should go through all the possible causes. An elevated T wave can occur due to hyperkalemia and must be treated. An electrolyte check is called for.

>> As CRNA Marid looked closer at the ECG curve, he immediately suspected hyperkalemia as the cause. He quickly took an arterial blood gas and handed it to Al to take to the lab. A few minutes later, he returned with the following values:

- *pH: 7.10 (reference 7.35–7.45)*
- *P_aO_2: 120 mmHg (reference 70–100 mmHg)*
- *P_aCO_2: 115 mmHg (reference 36–44 mmHg)*
- *HCO_3: 21.9 mEq/l (reference 22–26 mEq/l)*
- *BE: −3.1 mEq/l (reference ±2 mEq/l)*
- *S_aO_2: 99.5 % (reference 95–98 %)*
- *Lactate: 1.5 mmol/l (reference 0.5–2.2 mmol/l)*
- *Na^+: 135 mEq/l (reference 135–150 mEq/l)*
- *K^+: 7.2 mEq/l (reference 3.5–5.0 mEq/l)*
- *Glucose: 85 mg/dl (reference 70–99 mg/dl)*

"Yikes!" thought CRNA Marid. "Now I've got to do something quick!"

31.1.12 Why Is Hyperkalemia Dangerous? How Can You Explain the ECG Changes?

Mr. Scott's arterial blood gas confirms the diagnosis of hyperkalemia. A quick increase of the extracellular potassium, especially in patients

who do not usually have increased potassium, is potentially life threatening.

The intra- and extracellular sodium and potassium level determine the electrophysiological activity of the cardiac myocytes. The resting membrane potential is usually −90 mV and is primarily determined by potassium gradients. If the extracellular potassium concentration increases, the gradient decreases, and the resting membrane potential increases (becomes less negative).

The value of the resting membrane potential has a deciding effect on the first phase (phase 0) of the cardiac action potential. In phase 0, the sodium channels open, and the extracellular sodium flows into the cell. The number of activated sodium channels is dependent on the resting membrane potential: the higher the resting membrane potential (less negative), the fewer channels are activated, and the slower the sodium influx occurs. The result is a slowed impulse conduction and a slowed impulse duration, visible as a QRS widening and an extended PQ interval.

Simultaneously, the resting membrane potential approaches the depolarization threshold, so that the danger of spontaneous action potentials increases and rhythm disorders can result. If the extracellular potassium continues to increase, the resting membrane potential will also increase, and an increase in the depolarization threshold will occur, so that excitability of the cardiac myocytes decreases and a cardioplegia results.

Due to unknown causes, extracellular hyperkalemia also affects the potassium channels, which are responsible for the plateau phase (phase 2) and the repolarization (phase 3). The following rule applies: the higher the extracellular potassium concentration, the higher the K^+ efflux from the cell. This phenomenon causes an increase in the curve for phases 2 and 3, and a shortened repolarization time, and is therefore responsible for the typical ECG changes such as ST depressions, elevated T waves, and shortening of the QT interval.

31.1.13 What Would You Do Now?

Rapidly occurring hyperkalemia with ECG changes is especially dangerous and requires immediate treatment. The goal is to decrease the extracellular potassium concentration to avoid arrhythmias. Figure 31.3 shows an algorithm to follow in acute hyperkalemia.

Acute hyperkalemia therapy is accomplished in three steps:

31.1.13.1 Stabilization of the Myocardial Membrane

Ca^{2+} works fast and is also effective when the calcium level is normal. The duration of action is about 1 h. If the ECG changes persist, the dose can be repeated.

31.1.13.2 Get the K^+ into the Cells

Mr. Scott's arterial blood gas showed a pronounced respiratory acidosis. The intracellular shift of H^+ ions results in hyperkalemia. Therapy of respiratory acidosis can cause a quick reversal of the shift and can reduce the extracellular potassium concentration. Administration of a buffer solution is only helpful when an acidosis is present and respiratory compensation isn't possible.

The inhaled or intravenous (off-label use) form of β_2-mimetics rapidly decreases the potassium level (onset of action in 1–2 min). Relatively high doses must be given, and care must be taken in patients with coronary artery disease or serious rhythm disorders. The β_2-mimetic medication does not work as desired for about 1/3 patients, due to an unknown reason. Another possibility is administration of insulin together with glucose. An effect should be seen 15 min after administration. Obviously, careful blood sugar measurements are required.

31.1.13.3 Eliminate K^+

To explain the various steps in this algorithm would take more time than is allowed in our case. Nevertheless, it is important to be aware of the additional escalating steps in the treatment of hyperkalemia, in order to fall back on them if needed. These include hyperkalemia due to an incompatible blood transfusion with consecutive hemolysis or hyperkalemia due to large soft tissue trauma. In such cases, early CVVH can be lifesaving.

>> CRNA Marid knew exactly what he had to do. Recently he had seen how a renal transplant recipient had gone into asystole due to

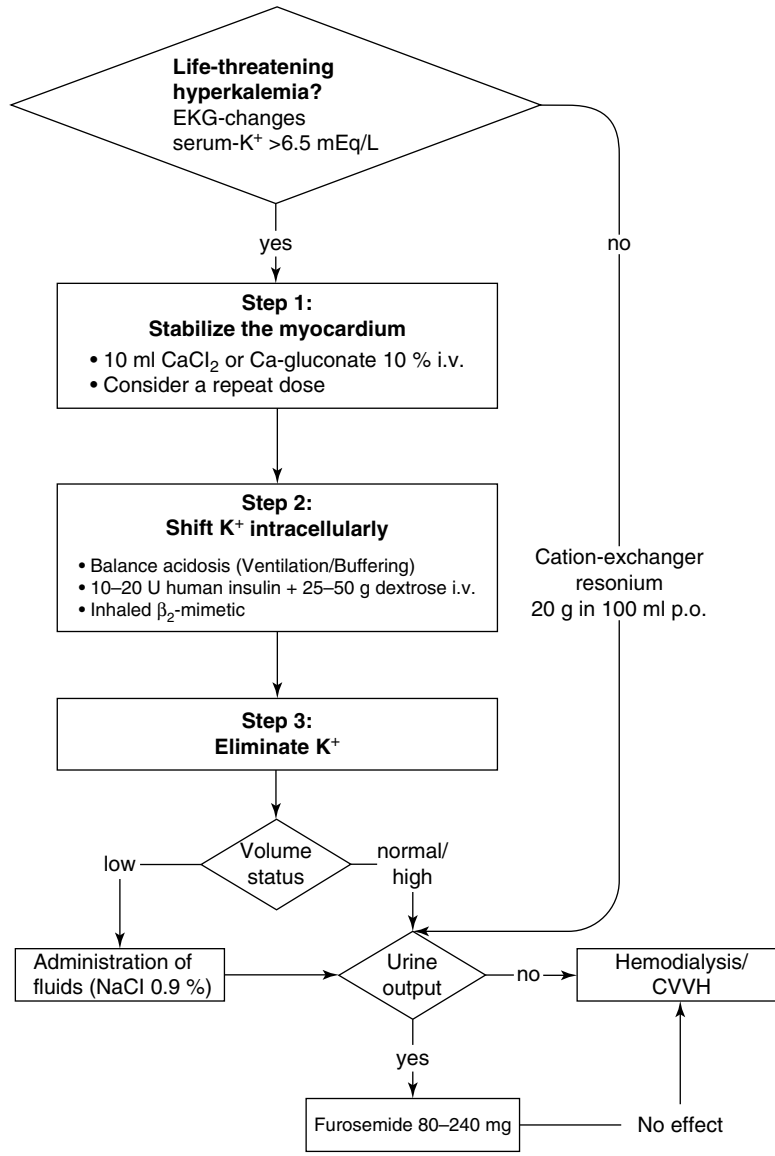

Fig. 31.3 Management of acute hyperkalemia. The modified algorithm of Toronto General Hospital (From Sood et al. [11], with permission) (*CVVH* continuous venovenous hemofiltration)

hyperkalemia right after the venous anastomosis of the new kidney was opened. Only with clear instruction of the attending Dr. Eldridge was the cardiopulmonary resuscitation successful. The steps were now imprinted in Marid's brain.

Turning to the student who was waiting to see what would be done about the blood gases, Marid said, "Al, get me calcium gluconate and terbutaline, and bring me some insulin and dextrose 50%. I think we're gonna need it all." Then CRNA Marid changed the ventilator adjustments as follows:

- *Respiratory rate: 30/min (from 26/min)*
- *V_T: 500 ml (from 450 ml)*

During the few minutes when Al was gathering supplies, CRNA Marid attempted to run through in his head the upcoming steps he needed to take. He noticed that he wasn't able to concentrate very well, although he was trying his best. "Dadgummit! Think! Today of all days – you can't be sick now!" He felt faint. He dashed to the anesthesia medications, grabbed phenylephrine, and dropped a few drops under his tongue. He shivered due to the extremely bitter taste, but he felt stronger immediately.

Shortly thereafter, Al returned with the supplies, and Mr. Scott received:

- *10 ml calcium gluconate 10%*
- *0.5 mg terbutaline subcutaneous* (*SC*)
- *50 mg glucose*
- *10 U regular insulin IV*

The high T waves normalized shortly after the administration of Ca^{2+}. "*Thanks for your help*," *CRNA Marid said, turning to Al.* "*In half an hour, I'll need another blood gas*."

After only 20 min, CRNA Marid went ahead and drew a blood gas. The ECG was still unremarkable, but the heart rate had increased to 110 beats/min. Simultaneously, an increase in the continuous phenylephrine infusion was necessary in order to maintain a sufficient blood pressure, although the blood loss was minimal. All this made CRNA Marid very anxious to see the blood gas values now:

- *pH*: *7.05* (*reference 7.35–7.45*)
- P_aO_2: *108 mmHg* (*reference 70–100 mmHg*)
- P_aCO_2: *135 mmHg* (*reference 36–44 mmHg*)
- HCO_3–: *20.9 mEq/l* (*reference 22–26 mEq/l*)
- *BE*: *–3.3 mEq/l* (*reference ±2 mEq/l*)
- S_aO_2: *99.4%* (*reference 95–98%*)
- *Lactate*: *1.8 mmol/l* (*reference 0.5–2.2 mmol/l*)
- Na^+: *138 mEq/l* (*reference 135–150 mEq/l*)
- K^+: *6.5 mEq/l* (*reference 3.5–5.0 mEq/l*)
- *Glucose*: *135 mg/dl* (*reference 70–99 mg/dl*)

"*Dammit*," *thought CRNA Mark*, "*why can't I get this* CO_2 *out*?!"

31.1.14 What Would You Do Now?

The immediate danger of hyperkalemia seems to have been avoided for now; therefore, the answer to the question is: steps must be taken to normalize the pH. But what?

From an isolated arterial blood gas result, the diagnosis would be respiratory acidosis. However, this is only partly true in our case. The ventilation is insufficient to exhale the CO_2, but a respiratory minute volume of 15/min is already an unusual value for a healthy patient under general anesthesia. The hypercapnea is most certainly due to the exogenous influx of CO_2. The term "resorptive CO_2-acidosis" takes the pathophysiology into account. Essentially, CRNA Marid has two possibilities:

- Optimize the ventilation
- Stop the exogenous CO_2 administration

Note: Malignant hyperthermia is extremely unlikely, because there is no increase in the lactate acid.

31.1.15 How Do You Explain the Cardiovascular Changes?

The cardiovascular changes must be viewed in context with the surgery, the medical procedures, and the acidosis. As already stated, there is no large blood loss. The remaining possible causes include:

31.1.15.1 Acidosis

Acidosis influences the cardiac output, leads to peripheral vasodilation, and decreases the efficacy of endogenous and exogenous catecholamines.

31.1.15.2 β-Stimulation

Terbutaline is a so-called β_2 agonist. Unfortunately, it is not completely specific for β_2, as we may have been taught. Over time, absorption from a subcutaneous injection can increase the heart rate via β_1 stimulation, especially when a large amount is given.

31.1.15.3 Hypovolemia

Despite the extreme Trendelenburg positioning, relative hypovolemia is possible. The main causes are the peripheral vasodilation as a result of acidosis, and influence of the venous return by the capnoperitoneum (analogous to the vena cava compression syndrome in pregnant women).

31.1.15.4 Stress on the Right Side of the Heart

Acidosis causes an increase in resistance in the pulmonary arteries, with the associated increase in stress on the right side of the heart.

31.1.15.5 Hypervolemia?

Above all, in patients with a cardiac predisposition, the extreme Trendelenburg position can also lead to acute stress on the right side of the heart.

Fig. 31.4 Explanation see Sect. 31.1.16

>> CRNA Marid wasn't sure which treatment option he should begin next – he decided to call his attending. Dr. Eldridge came, listened to the details of the case and the actions which had been taken thus far. Then he asked the urologist to interrupt the operation and he systematically checked the ventilator. "Hmmm," mumbled Dr. Eldridge, "a problem seldom comes alone." He asked, "What do you think of this?" and brought the attention of CRNA Marid and Student Al to the CO_2 curve and to Fig. 31.4.

31.1.16 What Do You Think of Fig. 31.4?

The CO_2 absorber of the ventilator is illustrated. The dark color in the bottom part of the CO_2 absorber (actually it is violet in color) shows that it is saturated and used up.

The CO_2 curve is not pictured here, but Marid and Al noticed that the inspiratory curve didn't begin anywhere near 0.

>> "This morning there was only a small violet layer near the bottom," said student Al. "I thought it would be sufficient for this surgery, that's the reason I didn't change it." Feeling guilty, he left the OR in order to obtain fresh CO_2 absorber.

Attending anesthesiologist Dr. Eldridge carried out a recruiting maneuver with a peak pressure of 40 cmH_2O for one minute, finally increasing the PEEP to 20 cmH_2O. All three treatment modalities together (interruption in the CO_2 administration for the surgery, fresh soda lime, recruitment) led to a normalization of the cardiovascular system and a decrease of the CO_2 expiratory to 45 mmHg. Attending anesthesiologist Dr. Eldridge gave the urologist a sign that he could continue his surgery.

The course of the anesthesia was uneventful for the rest of the surgery. One hour after the interruption, surgery was finished. Shortly before the remifentanil pump was stopped, CRNA Mark administered 100 µg fentanyl IV. Then he reversed the muscle relaxant and extubated the patient without difficulty. As Marid arrived in the PACU with his patient, his attending was already there waiting for him. "Now let's take a good look at you. Are you sure you didn't catch your daughter's flu?"

31.2 Case Analysis/Debriefing

31.2.1 What Is the Difference Between $CaCl_2$ and Calcium Gluconate?

In Sect. 13.1.12, Ca^{2+} therapy for acute hyperkalemia was mentioned. Which of the two is better? Are there differences between the two? After all, both are distributed as 10 % solutions.

Yes, of course there are differences – we wouldn't bother to ask if there weren't…

31.2.1.1 Calcium Content

The calcium content in $CaCl_2$ is about three times higher than that in calcium gluconate.

- Calcium gluconate (10 ml): 93 mg (2.3 mmol) elemental Ca
- Calcium chloride (1 0 ml): 272 mg (6.8 mmol) elemental Ca

31.2.1.2 Metabolism

Calcium gluconate must first be metabolized in the liver, so that Ca^{2+} is made available.

In cases of severe cardiovascular depression, or in an extreme situation such as cardiopulmonary

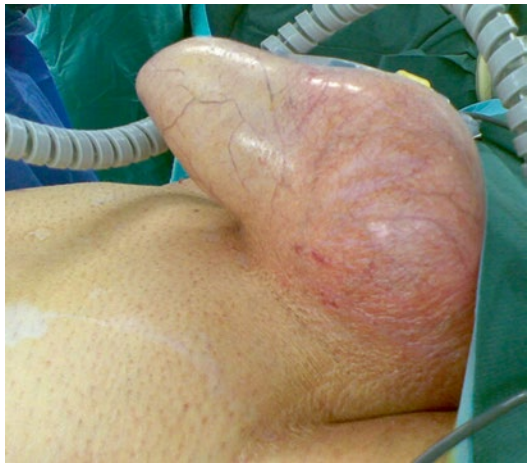

Fig. 31.5 Severe scrotal and penile subcutaneous emphysema. The subcutaneous emphysema expands unhindered through subcutaneous tissue, sometimes reaching the thorax during longer surgeries. Even facial emphysema is observed. Additionally, the CO_2 can reach the mediastinum via physiological or anatomical diaphragmatic apertures

resuscitation due to hyperkalemia, perfusion of the liver may not be sufficient, and the desired effect may not be achieved.

31.2.2 Do You Have an Explanation of Why the CO_2 Absorption Can Be So Pronounced in This Type of Operation?

The da Vinci prostatectomy is an intra- and extraperitoneal procedure. If the connective tissue in the pelvic area is slack, the integrity of the entire pelvis as a sealed entity may fail. As a result, insufflated CO_2 can leave the pelvic cavity relatively unhindered. The formation of subcutaneous emphysema is dependent on this phenomenon and the duration of the surgery (Fig. 31.5).

The collection of CO_2 in the subcutaneous tissue does not only lead to local acidosis, but also significantly enlarges the reabsorption area. When serious emphysema is discovered, it is important to rest assured that the trocars haven't slipped out of the abdominal cavity.

31.2.3 How Does Soda Lime Work?

Soda lime was originally used by the military shortly before or during the First World War, to avoid CO_2 intoxication by submarine soldiers. The most commonly used soda lime is made up of about 80 % calcium hydroxide [$Ca(OH)_2$], 3 % sodium hydroxide (NaOH), and 15 % water. The previously used potassium hydroxide (KOH) is no longer available, due to interactions with volatile anesthetics. The initial chemical reaction is called neutralization and is the creation of carbonic acid:

$$H_2O + CO_2 \cdot H_2CO_3 \qquad (31.1)$$

From carbonic acid and sodium hydroxide, sodium carbonate (Na_2CO_3) is formed.

$$H_2CO_3 + 2\,NaOH \cdot Na_2CO_3 + 2\,H_2O + Heat \qquad (31.2)$$

In the third step, sodium carbonate is changed into insoluble calcium carbonate ($CaCO_3$). Simultaneously, sodium hydroxide is regenerated:

$$Na_2CO_3 + Ca(OH)_2 \cdot CaCO_3 + 2\,NaOH \qquad (31.3)$$

The addition of sodium hydroxide increases the rate of the reaction, because the direct formation of calcium carbonate from calcium hydroxide and carbonic acid is too sluggish to bring about a complete elimination of CO_2 from the ventilator system.

The soda lime consists of a porous granulate (Fig. 31.4), in order to create the largest surface area possible for the chemical reaction. If the soda lime is too moist, drops of moisture will form on the granules, and the available surface area for the reaction decreases. Silicate is added as a stiffening agent, to prevent dust particles from being formed, which might be inhaled by the patients.

The theoretical capacity of soda lime is about 26 l of CO_2 per 100 g of lime, although in practice, the formation of moisture drops inhibits the absorption to about 10–15 l per 100 g. Color coding helps to determine when soda lime is saturated. This is important in that most anesthesia

ventilators (still) are not equipped with an inspiratory CO_2 measurement.

Let's go back to our case: Mr. Scott had a respiratory volume of 15 l and a CO_2 content of about 8 % of the volume of expired air. This means that per hour $60 \cdot 1.2\ l = 72\ l\ CO_2$ must be absorbed. The absorber illustrated in Fig. 31.4 has a capacity of 1.5 l and can absorb at least 150–225 l CO_2. Therefore, a fresh absorber would most certainly have lasted for more than 2–3 h.

31.2.4 Which Medical Errors Do You See in the Presented Case?

31.2.4.1 Ventilation

The ventilation was inadequate. The initially chosen PEEP of 6 cmH_2O was too low to prevent atelectasis formation [13]. Then, the increase of PEEP to 10 cmH_2O was not enough to recruit the lost alveoli. The recruiting maneuver done by the attending may appear aggressive, but was necessary to achieve CO_2 elimination. The increase in PEEP which followed was necessary to prevent/reduce cyclic alveolar collapse.

31.2.4.2 Check of Tube Placement

A large difference between the end-tidal CO_2 and the P_aCO_2 can be caused by a secondary endobronchial intubation (see Sect. 16.1.8). CRNA Marid didn't carry out a proper tube placement check.

31.2.4.3 Soda Lime Which Has Lost Its Absorbing Capacity

As discussed in Sect. 31.2.3, measurement of inspiratory CO_2 concentration is not (yet) standard. Accordingly, there is usually not an alarm function. On the other hand, all ventilators are equipped with a capnographic monitoring, so that a visual check is easily done. The same applies to the indicating dye placed in every soda lime.

31.2.4.4 Therapy of the Acidosis

Attending anesthesiologist Dr. Eldridge requested that the urologist take a break from the surgery, so that he could get his situation under control again. This measure was indicated much earlier.

31.2.5 Which Systems Failures Can You Find in the Presented Case?

31.2.5.1 Exchange of the Used Soda Limes

If a longer laparoscopic OR surgery is planned, fresh soda lime should be placed in the absorber. Obviously, a set standard operating procedure (SOP) had not been put in place.

31.2.5.2 Too Sick to Go to Work?

There are many different reasons why co-workers show up for work sick. The cause can be organizational, such as when a supervisor or the company exerts pressure, whether directly or indirectly.

31.2.5.3 Supervision

Attending anesthesiologist Dr. Eldridge suspected that CRNA Marid was sick. However, he neglected to follow up on his suspicion or to go into the OR to provide supervision or assistance.

31.2.6 A Sick Doctor/Health-Care Provider?

This is a contradiction for most people. We are healthy by nature; we tend to exploit ourselves like no other professional group. When we are occasionally weak or sick, we rely all too often on medications to help us bounce back.

The medical term for CRNA Marid's behavior "to go to work despite sickness" is called **presenteeism**. There are three categories of factors which trigger presenteeism [12]:

- Personal factors (age, sex, relationship status, sense of duty, etc.),
- Job and organizational factors (job requirements, salary, amount of work to do, leadership, corporate culture, etc.),
- Employment factors (job security, etc.).

Why is it important to take a moment to consider such things? Presenteeism has consequences for oneself personally, for the employer, and for the patients.

- Presenteeism increases the risk of suffering from a chronic disease (especially of the cardiovascular system) and increases prolonged absences due to long-term unfitness for work (absenteeism).
- Sickness is one of the main performance-modulating factors. We are just worse, and we make more mistakes, when we work sick.

Factory supervisors know that the costs which arise from sick workers (mistakes in production) are higher than the costs which arise from their absence (absence of production). Why should medicine be different? Attention: we are not just talking about costs here; we are talking about patients who can be the recipients of our mistakes.

31.2.6.1 The following section is for all employees

Allow yourself a break when you are sick! Your patients do not expect you to be in perfect health, but they do expect a doctor who gives them the full attention they deserve.

Have you ever heard of karoshi? It's Japanese for "death by overwork." The cause of death is the stress caused by work, which leads to heart attacks and strokes, and, not surprisingly, is accompanied by depression. Therefore, don't touch medications in order to try to hide your illness.

31.2.6.2 The following section is for all employees in a supervising capacity

Be alert, and send employees home when they are sick! Set a good example and stay home when you are sick! At regular intervals (e.g., once a year), request a "sick report" from your administration department, and don't be happy when you see that it is very low (under 3 %). That could be a clue that your presenteeism in the department may soon cause one or two employees to be out sick for an extended period of time.

Have you ever heard of karoshi? It's Japanese for "death by overwork." The cause of death is the stress caused by work, which leads to heart attacks and strokes, and, not surprisingly, is accompanied by depression. It is often seen in employees in a supervisory position.

31.2.6.3 Afterthought

There is a positive effect (the so-called salutogenic effect) of presenteeism on health. For certain illnesses such as chronic pain, muscular skeletal diseases, or psychiatric diseases, going to work can act as a self-conquest and has a therapeutic value. But that wasn't the case with CRNA Marid.

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Case 32: Angiography

32

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32.1 Case Introduction

>> *"Where am I?" Mrs. Smith wondered. She looked around as best she could, lying on her back. Her husband leaned over her. "What's wrong with you, Elvira?" As Mrs. Smith tried to prop herself up, her left arm gave way. "I'm so weak," she said. "OK, just stay there!" responded her husband. "I'll call 911!" With that, Mr. Smith ran into the house.*

When the paramedics arrived, the symptoms were totally gone. "Do I really have to go to the hospital?" said Mrs. Smith. "I feel as if nothing happened."

32.1.1 Does Mrs. Smith Have to Go to the Hospital? What's Your Differential Diagnosis?

It appears that Mrs. Smith has experienced an episode of syncope with retrograde amnesia. Syncope

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has many differential diagnoses (see Sect. 2.1.2). Due to the presence of transient neurological symptoms, cerebral syncope is the most likely cause. Cerebral syncope episodes can be caused by:

- Ischemic stroke/stroke precursors such as TIA (transient ischemic attack) or PRIND (prolonged reversible ischemic neurologic deficit)
- Cerebrovascular insufficiency
- Vascular "steal syndrome"
- Epilepsy
- Intracranial bleed

The first episode of cerebral syncope in a patient requires evaluation. Being that the risk of a stroke within the next 3 days is extremely high, secondary prophylaxis should be begun without delay. Secondary prophylaxis includes the following:

- Treatment of vascular risk factors
- Assessment and adjustment of coagulation and platelet function
- Revascularization

Before secondary prophylaxis is begun, intracranial bleeding must be ruled out.

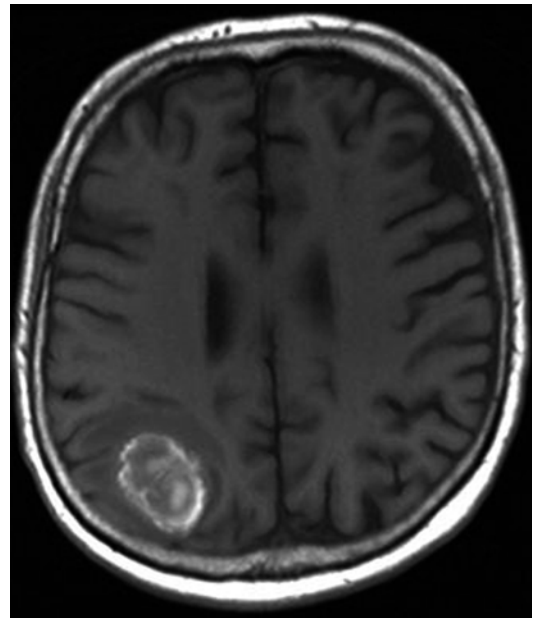

Fig. 32.1 The MRI clearly shows a hyperdensity in the right occipital lobe. An arteriovenous malformation was the cause of the bleeding

>> *Transport to the hospital went without incident. Upon arrival, Mrs. Smith underwent a CT. The scan showed an atypical, right occipital, intracranial hematoma, and Mrs. Smith was informed that she would be transferred to a tertiary care hospital.*

A team was awaiting her arrival at the university neurology clinic. The neuroradiologist had already seen the CT and decided upon the further diagnostic procedures (Fig. 32.1).

After the MRI, the neurosurgeon Dr. Faith came to Mrs. Smith to discuss the findings and further treatment.

"*Mrs. Smith,*" *she said, "we have located the cause of your symptoms. In the back of your head, there is a vascular malformation which began to bleed a little bit. This is both good news and bad news. The good news is that you do not have a tumor; the bad news is that you could bleed again at any time from the abnormal vessels.*" *Dr. Faith paused for a moment to allow the news to sink in.*

With a shivering voice Mrs. Smith responded, "Am I going to die?"

"*Bleeding within the brain is always life-threatening, but the most important thing is that we have found the cause of the bleed and we can now suggest treatment.*"

Dr. Faith had won over Mrs. Smith's trust. She explained the various therapeutic options and concluded with her recommendation of the least invasive method: endovascular embolization. Mrs. Smith agreed to this procedure.

"*We have an excellent specialist here, an experienced neuroradiologist who will perform your procedure,*" *explained Dr. Faith. "However, he is on vacation at the moment and we must be patient for a week. I'll go ahead and call the anesthesiologist for you.*"

Dr. Perk was freshly board certified, and tended to get stuck with all the jobs nobody else wanted to do. Within his department, he was known for his wit and had already been written up once for being a clown in the OR.

When his phone rang, he was talking with a cute female colleague. He answered with, "Department of Anesthesiology, please hold, someone will be with you shortly," then glanced at the display. "Uh-oh!" he thought as he read the screen: Dr. Eldridge. The attending had an unpredictable sense of humor.

"*Perk*," *said the anesthesiologist-in-charge*, "*When are you going to grow up*? *You're in the big boys group now. In neurosurgery there is a patient*, *Mrs. Smith*, *who is waiting for her preoperative consult. I hope that you can rise to the challenge*."

With that, *the conversation was over*. "*Guys*," *said Dr. Perk*, *pulling himself up tall in front of his colleagues*, "*that was the big boss. I have been given a special assignment – and I gotta go double-oh-seven it. Ciao*!"

32.1.2 What Does the Anesthesiologist Need to Watch Out for in This Case?

In case 30 (see Sect. 30.1.3), a neuroradiology case was discussed. The main aspects are:

- Difficult patient access during the intervention
- Careful monitoring of the blood pressure, especially avoiding blood pressure peaks
- Continuous neuromuscular blockade in order to minimize the danger of vessel rupture during the procedure

>> *After viewing the file*, *Dr. Perk had put together the following details*:

- *63 years old*, *165 cm*, *81 kg*.
- *A fall 10 days ago*, *after which there was temporary incomplete paralysis of the left arm*.
- *EEG showed oscillations typical of epilepsy on the right side of the brain* (*epileptiform discharges*), *and treatment was begun with levetiracetam*.
- *Angiography detected a bilateral dural arteriovenous fistula over the occipital arteries*.
- *Other preexisting conditions*:
  - *Systemic hypertension treated with bisoprolol*, *amlodipine*, *enalapril*, *and furosemide*
  - *Asthma treated with salmeterol and budesonide spray*
  - *Type 2 diabetes treated with short- and long-acting insulin*
  - *Renal failure stage II*
  - *Obesity and hyperlipidemia treated with simvastatin*
  - *Aspirin paused*; *pantoprazole to prevent ulcers continued*

Table 32.1 Classification of renal failure

Stage	Glomerular filtration rate [ml/min/1.73 m^2]	Characteristics
1	>89	Proteinuria, normal kidney function
2	60–89	Mild impairment of renal function with or without proteinuria
3	30–59	Moderate impairment of renal function
4	15–19	Severe impairment of renal function
5	<15	Chronic renal failure

32.1.3 What Is Stage II Renal Failure?

The US National Kidney Foundation divides renal insufficiency into five stages (Table 32.1).

Due to the necessary administration of contrast dye, Ms. Smith is at increased risk for post-interventional acute renal failure (see Sect. 29.2.4).

>> "*Totally healthy patient with asthma*, *metabolic syndrome*, *and hypertension*," *thought Dr. Perk*, *as he was making his way to the patient's room. In the lab work-up*, *he noticed an elevated creatinine. But that fit in with renal failure. The ECG was unremarkable*, *apart from minor left ventricular hypertrophy*.

During the conversation, *Mrs. Smith explained that she went regularly to her family physician. Her hypertension was well controlled*, *and her blood sugar fluctuated very little* (*she dosed her insulin very carefully*). *She managed her asthma successfully as well*. "*What do you mean*? *How many flights of stairs can you climb*?" *probed Dr. Perk*. "*Well*, *after 1–2 floors*, *I stop to take a break*," *answered Mrs. Smith*, "*then I inhale my spray and wait a moment until I can breathe OK again*."

32.1.4 What Do You Think of This Physical Fitness Report?

Mrs. Smith complained of dyspnea upon exertion, the cause of which couldn't be determined from the history. It may be asthma, or it may have

a cardiac cause. Useful preoperative tests therefore include:

- Pulmonary function tests including reversibility testing of airflow limitation for possible optimization of the asthma therapy
- Cardiac stress test/stress echo

The indication for these tests must be viewed critically, due to the intracranial bleed and the possible increase in intracranial pressure with exercise, which increases the danger of recurrent hemorrhage. Alternatively, transthoracic echocardiography can safely assess pump function, diastolic relaxation, valve abnormalities, and pulmonary arterial pressure.

Coronary artery disease, however, cannot be ruled out with this technique.

>> Dr. Perk obtained informed consent from Ms. Smith for the anesthesia. "We'll see each other in a week!" he said as he left.

As he was sitting in his office completing the paperwork, he began to worry about whether or not to order additional tests. After short deliberation, he decided to call anesthesiologist-in-charge Dr. Eldridge for advice. After 3 rings, Dr. Eldridge answered "Department of Anesthesiology – please hold – someone will be with you shortly…" Very funny, thought Dr. Perk, and a little resentful. Anyway, he described Mrs. Smith's medical problems. In response to Dr. Eldridge's first question, he reported that Mrs. Smith had denied experiencing a change in her dyspnea within the past few weeks. Together, the anesthesiologists decided against additional diagnostic tests.

"Good morning, Mrs. Smith!" Dr. Perk said to her a week later. He reviewed the patient's medical record once more, and saw to his astonishment, that thoracic CT angiography was done the very same day when he saw her for her preoperative evaluation. The reason for the test was "acutely worsening dyspnea with tachycardia." Pulmonary emboli were ruled out.

In the meantime, CRNA Rose had gotten Mrs. Smith hooked up to the monitor:

- *S_PO_2 92 %*
- *Blood pressure 120/60 mmHg*
- *HR 80 beats/min, sinus rhythm*

Induction of the TIVA with remifentanil, propofol, and atracurium went without incident, as well as endotracheal intubation and placement of an arterial line. To assist in maintaining a stable blood pressure, Mrs. Smith received 1,000 ml of crystalloid and a phenylephrine infusion was started at 40 µg/min. CRNA Rose gave Dr. Perk a copy of the arterial blood gas analysis. "Thanks," he said "but we really don't need this." The abnormal values were:

- *Na^+: 130 mEq/l (reference 135–150 mEq/l)*
- *Hb 9.2 g/dl (reference 11.9–17.2 g/dl)*
- *Glucose 145 mg/dl (reference 70–99 mg/dl)*

Unfortunately, the planned embolization of the vessel abnormality could not be performed due to substantial elongation of the artery. The attempt was aborted after 2 h. "What a shame for Mrs. Smith," Dr. Perk said to CRNA Rose, "Now they will have to do a craniotomy." He stopped the atracurium infusion, and 15 min later the neuroradiologist said goodbye. After another 20 min, the propofol and remifentanil infusions were stopped. Dr. Perk expected a quick awakening. After another 15 min, Mrs. Smith was still not wake-able. She had tachypnea and was breathing with a very low tidal volume.

32.1.5 What Do You Think of When You See the Presented Signs?

The answer shouldn't be too difficult for you:

The symptoms are typical for postoperative residual neuromuscular blockade. The diagnosis is confirmed via nerve stimulation (see Sects. 12.1.4 and 16.1.9). The therapy involves either reversing the neuromuscular blockade or waiting (see Sect. 12.1.4).

>> Dr. Perk was shocked to see that the TOF showed complete muscular recovery. "Well, OK," he thought, "I'll give Mrs. Smith a little more time." Twenty-five minutes later, her spontaneous breathing was sufficient enough to extubate, and the increase in heart rate and blood pressure hinted that Mrs. Smith was going to wake up soon.

"*Let's extubate now*" *he said to his nurse*. "*Stress isn't good for Mrs. Smith's intracranial bleed*." *After extubation*, *Mrs. Smith was hooked up to the transport monitor. Just before they started rolling*, *CRNA Rose glanced at the screen*. "*Dr. Perk – did you see the pulse and pressure*?" *The blood pressure was 185/75 mmHg*, *and the heart rate was 120 beats/min. Dr. Perk replied*, "*Let's get her to the PACU – the guys there will take care of it*."

The handover to the PACU physician was short. Dr. Doxa was busy with many other patients, *and she only had a moment of time. Apart from that*, *she wasn't too sympathetic towards Dr. Perk*, *ever since she found out that he made jokes about her to fellow physicians. Dr. Perk felt that he wasn't welcome*, *and hurried off*.

PACU nurse Agnes received detailed info about Mrs. Smith from her colleague CRNA Rose. Nurse Agnes didn't think the patient looked well at all and immediately brought this to the attention of Dr. Doxa: "*The patient is shivering*, *has a blood pressure of 190/75 mmHg*, *and a heart rate of 120 beats/min. She is completely out of it and constantly yanks her oxygen mask off her face*."

32.1.6 Repeat Question: What Are Your Differential Diagnoses?

Did you forget? Then read again Sects. 18.1.2, 18.1.7, 19.1.9, 19.1.11, and 22.1.3.

32.1.7 What Is Shivering, and How Common Is It? Which Age Group Has the Highest Incidence?

Shivering is involuntary muscle activity for the metabolic production of heat, a process that can be increased up to 600 fold, simply through muscle work [2].

Postoperative shivering is common and is seen in up to 2/3 of all anesthetics [1, 5].

Young adults are most commonly affected, with a slight predominance in males [1]. Actually, newborns, small children, and older people tend to lose more heat intraoperatively. Up until the 4th (to 6th) year of life, heat is formed without shivering through the metabolism of brown fat, and shivering is seldom seen. Additionally, in this patient group, the temperature regulation center is not yet fully developed. In older patients, thermoregulation is weakened; therefore, the incidence decreases with age.

>> *Dr. Doxa was a little stressed. She had been trying for 15 min to reach the orthopedic surgeon about a recurrent hemorrhage. As she was looking for his number and simultaneously telephoning the blood bank to order crossmatching*, *she yelled at PACU nurse Agnes*, "*Give her 12.5 mg of meperidine*!"

Three minutes later, *nurse Agnes was again standing in front of her*. "*The drug had no effect. Mrs. Smith is still shivering. I hooked up the warming blankets already. Should I give her clonidine*?" *Dr. Doxa nodded in agreement*, *the phone stuck between her ear and her shoulder*.

The PACU was more hectic than ever. An 18-month-old boy was crying and could not be consoled. The patient with the postoperative recurrent hemorrhage was receiving blood transfusions, *which were flowing directly out the drainages from the wounds. Three new patients were on their way*, *but there was only one empty space*. "*Who can I send to the floor*?" *thought Dr. Doxa. Mrs. Smith entered her mind first as she was searching for patients to transfer to the ward*.

Dr. Doxa came to Mrs. Smith's bedside. The monitor was blinking an alarm: *hypotension*; *but the volume had been shut off. The blood pressure was 100/55 mmHg*, *and the heart rate 110 beats/min. Mrs. Smith was still shivering and was in no way responding adequately*, *but only moaned. Nurse Agnes had done an arterial blood gas*, *and gave it to Dr. Doxa. The only abnormal values were*:

- *Na^+: 131 mEq/l* (*reference 135–150 mEq/l*)
- *Hb 9.7 g/dl* (*reference 11.9–17.2 g/dl*)
- *Glucose 153 mg/dl* (*reference 70–99 mg/dl*)

"*That doesn't look so bad*," *thought Dr. Doxa to herself*, "*but I still can't transfer her*." *Out loud she said to the nurse* "*Unchanged. Give her 250 ml of 5% albumin*." *Then she went to accept the next two patients*.

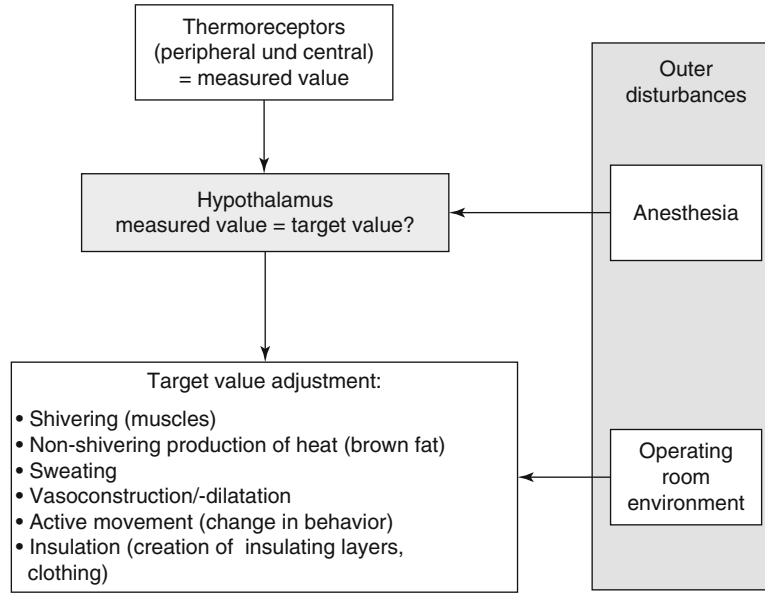

Fig. 32.2 Schematic diagram of temperature regulation. Anesthetics alter the body's usual temperature setting and simultaneously alter the function of the settings

ICU nurse Agnes hung the albumin, and then checked on Mrs. Smith again 25 min later. Meanwhile, the new arrivals had taken her attention. As a result of her therapy, her blood pressure was 190/65 and the heart rate was 130 beats/min. However, her mental status had not improved and Mrs. Smith was still shivering uncontrollably. Nurse Agnes went to Dr. Doxa. "*Mrs. Smith is still shivering; could it be something else*?" *Dr. Doxa shook her head*, "*When you hear hoof beats, don't think of zebras*."

32.1.8 What Do You Know About Temperature Regulation and the Etiology of Postoperative Shivering?

Postoperative shivering is divided into thermoregulatory and non-thermoregulatory shivering. Important aspects of the cause of thermoregulatory shivering include perioperative heat loss, maintenance of the body's set temperature, interruption of the afferent nerves, and adaptation to set values. Temperature regulation works as a control loop, with the goal of maintaining constant body temperature. The set value is determined by the hypothalamus, which receives information from cold and warm receptors located internally and on the skin surface. Calculations derived from the current and the desired body temperature allow for various adaptations (Fig. 32.2).

32.1.8.1 Perioperative Heat Loss

Perioperative heat loss occurs regardless of the type of procedure done; however, heat loss is extensive in open abdominal procedures, due to the large surgical area. Due to the unfavorable ratio of body weight to body surface area, pediatric and cachectic patients are at especially high risk. Physically, the loss of heat occurs via:

- Thermal conduction = heat transfer, such as the transfer of heat energy due to a concentration gradient of bodies in contact
- Convection = heat conduction, such as via air flow or flow of warm central blood into the body's colder peripheries
- Evaporation of fluids and via radiation

32.1.8.2 Adjusting the Set Temperature

Anesthetics cause the hypothalamus to reduce the target body temperature. Counterregulatory peripheral vasoconstriction occurs later [7]. An uncovered healthy subject under general anesthe-

sia first experiences peripheral vasoconstriction after the body temperature decreases by 1.3 °C [6]. In actively cooled neurosurgical patients, the vasoconstriction starts at a body temperature of 34.5 °C (94.1 °F) [6]. Simultaneously, general anesthesia reduces the shivering threshold; shivering reemerges only after the plasma concentration of anesthetics has decreased [3].

The incidence of shivering depends on the anesthetic medications and is highest with inhaled anesthetics, especially if used in combination with small amounts of opioids. Propofol, on the other hand, seems to cause very little shivering [1].

32.1.8.3 Disruption of Afferent Nerve Fibers

Apart from general anesthesia, regional anesthesia techniques also influence temperature regulation. Peripheral nerve blocks have less of an effect than neuraxial techniques, which block more hot and cold afferent nerves. The decrease in the temperature threshold for shivering is directly proportional to the spread of the neuraxial blockade. The simultaneous sympathicolysis causes peripheral vasodilation, which can bring about pronounced heat loss. Subjectively, a sense of warmth is felt, even though heat is being lost. Counterregulation can't occur due to the block of the afferent nerves. The combination of general and regional anesthesia has additive effects on the loss of heat [7].

32.1.8.4 Influencing the Body's Settings

Anesthetics effect thermoregulation and impair various mechanisms for formation and retention of heat. They cause peripheral vasodilation, and the temperature difference between the body's central and peripheral temperatures decreases. As a result, the body's core temperature decreases, and the peripheral temperature increases. The patient's skin often feels warm, which may cause the patient's temperature to be overestimated, if no core measurement is done. Of course, active movements to produce heat are blocked during anesthesia, or at least should be.

An important risk factor is the duration of surgery: the longer surgery lasts, the more often shivering occurs. Most of the heat loss can be minimized by proper precautions.

It is important to prevent heat loss early on, because passively warming patients can be challenging. Possible measures to prevent cooling include:

- Warm blankets in the induction room
- Administration of pre-warmed infusions
- Intraoperative passive patient warming
- Air humidification

32.1.8.5 Non-thermoregulated Shivering

Even patients with a normal body temperature may shiver. The long list of causes of nonthermal regulated shivering – also termed postoperative shivering/tremor – differ in etiology and are not fully understood. Causes include:

- Intraoperative suppression of the sympathetic nervous system activity with postoperative central nervous system overactivity (analogous to norepinephrine storm during awakening)
- Suppression of the adrenal glands
- Non-inhibition of spinal reflexes and early recovery of those reflexes
- Pain
- Release of pyrogenics and cytokines due to the surgical procedures

>> Dr. Doxa didn't have time for, nor was she in the mood for, a long explanation. "Agnes, anesthesia causes temperature regulation to go haywire. We need to continue this therapy a little longer. Give Mrs. Smith more meperidine!"

PACU nurse Agnes administered, as ordered, another 12.5 mg meperidine, and another 5% albumin to stabilize the blood pressure. But 15 min later, nothing much had changed. By this time, Mrs. Smith was more or less unconscious, having been in the PACU for an hour and a half now. Nurse Agnes did an arterial blood gas analysis again and showed Dr. Doxa the following abnormal values:

- *Na^+: 131 mEq/l (reference 135–150 mEq/l)*
- *Hb 10.2 g/dl (reference 11.9–17.2 g/dl)*
- *Glucose 173 mg/dl (reference 70–99 mg/dl)*

"We can't leave her like this," Agnes insisted. "Please come with me to examine her. We must find a proper treatment for her problem!

Something is not right!" *Together they went to Mrs. Smith's bedside*; *she was still shivering*.

32.1.9 Name Some Additional Causes of Shivering and Decreased Level of Consciousness

You might now think that we just asked that question (see Sect. 32.1.6)! Do you think we have run out of new questions to ask? Well, if you read the question carefully, you might notice the "and" between shivering and decreased level of consciousness. Got it now?

The most common cause of shivering is hypothermia. Apart from that, an increase of the target temperature or an allergic reaction can cause shivering. Both can occur with an altered level of consciousness.

>> "*Maybe I should measure the temperature. It's been so chaotic in here today that I didn't yet get around to it*," *remarked Nurse Agnes*. "*I thought you had done that long ago*!" *retaliated Dr. Doxa. The ear thermometer measured 39.2 °C. The patient's warming blankets were removed*.

Dr. Doxa was annoyed that she hadn't cared for Mrs. Smith earlier. "*All right*," *she said to nurse Agnes*, "*we're gonna rack our brains to go through all possible causes of Mrs. Smith's decreased LOC. Give her 1 g of acetaminophen to reduce her fever and then 8 U of regular insulin to decrease her blood sugar. Then give her 100 mg ranitidine and Benadryl 50 mg to treat a possible anaphylactic shock. I'll order a CT to rule out a stroke or a recurrent hemorrhage*.

The therapies didn't improve her condition much. The shivering had stopped, *after the acetaminophen took effect. The body temperature was still over 39 °C. She still had tachycardia* (*130 beats/min*) *and was still hypotensive* (*100/50 mmHg*) *despite administration of 1,000 ml of electrolyte solution. The patient didn't regain consciousness. The CT didn't provide any new information. Another arterial gas showed the following abnormal values*:

- *Na^+: 132 mEq/l* (*reference 135–150 mEq/l*)
- *Hb 10.6 g/dl* (*reference 11.9–17.2 g/dl*)
- *Glucose 125 mg/dl* (*reference 70–99 mg/dl*)

Dr. Doxa was just thinking, "*I am at the end of my medical knowledge*," *when anesthesiologist-in-charge Dr. Eldridge walked in and wrinkled his forehead. He listened as his colleague explained the events up until this point. Then he reviewed the patient's medical record. He had suspected exactly that and called for an ICU bed*.

32.1.10 We're Thinking that You Also Suspected Exactly That, Didn't You?

Let's list the facts all together again:

- Decreased level of consciousness
- Fever with shivering
- Tachycardia
- Hypotension (notably difficult to evaluate due to the various interventions carried out thus far)
- Hyperglycemia
- Mild hyponatremia
- Increasing Hb value, despite repeated volume administration

Mrs. Smith fulfilled the criteria for severe sepsis (see Sect. 25.2.1). Acute encephalopathy with a reduced level of consciousness is often not considered when determining the presence of organ dysfunction. The increasing Hb despite volume administration can be a sign for the increased fluid loss due to the fever or a sign that capillary leakage has begun.

>> *Mrs. Smith was moved to the ICU. There*, *she developed a severe septic shock with multiorgan system failure. She suffered respiratory failure*, *had dialysis*, *and developed a severe leukopenia and thrombocytopenia. The pathogen causing the sepsis was diagnosed in the blood culture*: *Escherichia coli. The search for the focus remained unsuccessful. Mrs. Smith recovered from her serious condition and was transferred to the ward after 20 days in the ICU. Four days*

later, the surgical repair of the dural AV fistula was planned.

We might want to mention that Dr. Doxa had not paid attention to what Dr. Eldridge saw in the medical record – 2 days before the angiography, Mrs. Smith's blood work showed leukocytes at 16,000 cells/mcl (norm: 3,600–9,800 cells/mcl).

32.2 Case Analysis/Debriefing

32.2.1 What Negative Effects Can Mild Hypothermia and Shivering Have on Patients?

Shivering is a common, undesired postoperative complication. Subjectively, patients have very different feelings about shivering, often very unpleasant.

Shivering in order to produce heat increases metabolism and oxygen requirement to about 600 %. The respiratory minute volume increases, in order to blow off the excessive CO_2. In particular, patients with pulmonary conditions may not be able to compensate.

After regional as well as general anesthesia, patients who are not warmed have an increased plasma epinephrine level. Shivering aggravates the stress reaction. The result is an epinephrine storm, with hypertension, tachycardia, and increased lactate. The patients who are especially at risk are those who may not be able to maintain sufficient dissolved oxygen levels (see Sect. 4.1.11), namely, patients with cardiac conditions, anemia, or ventilation–perfusion disorders. Patients with heart conditions are at risk of acute coronary syndrome and acute heart failure.

Shivering per se may increase postoperative pain due to the movement around the wound. In other words, pain may trigger shivering on the one hand, and on the other hand, the uncontrolled muscle activity can potentiate the pain.

Furthermore, shivering has been reported to cause increases in intraocular and intracranial pressures. What negative effect this may cause is unknown, and untested. Last but not least, shivering interferes with ECG and pulse oximetry reading, leading to inadequate monitoring [4].

Additional consequences of hypothermia include:

- Increased rate of wound infections
- Increased postoperative bleeding
- Reduced metabolism of medications

32.2.2 Name Possible Therapies of Postoperative Shivering

Patients should always be passively warmed when postoperative shivering occurs; after all, they feel cold. There are many pharmacological options available; a detailed discussion of which exceeds the space in this chapter [7]. Here are some possibilities:

32.2.2.1 Opioids

Various opioids can be used as a shivering treatment. Meperidine is usually given, with a dose of 12.5–25 mg (or 0.3–0.5 mg/kg). Tramadol may also be used, dosed at 0.5–3 mg/kg body weight. Both take effect quickly. They decrease the shivering threshold, probably via binding with μ- and κ-receptors and via the inhibition of serotonin and norepinephrine reuptake. In addition, central α_2-receptors are also involved. Typical side effects include nausea and vomiting.

32.2.2.2 Clonidine

Clonidine is a centrally active α_2-agonist. Clonidine decreases the thermoregulatory threshold temperature (the set target temperature) and has a positive influence on the epinephrine storm. There is a direct dose to effect ratio: 1.5–3 μg/kg body weight is recommended. Higher doses often bring side effects such as bradycardia and hypotension.

32.2.2.3 Other Substances

Less often used medications include anticholinergics (such as physostigmine), ketamine, granisetron, and magnesium. Exotic therapies include intrathecal injection of meperidine and epidural administration of ketamine. There is no clear evidence of improved effectiveness of any medication [1, 4, 7].

A pragmatic approach is to ask and check for additional signs and symptoms. Meperidine is

the drug treatment of first choice for shivering in the USA. Clonidine may be considered for refractory shivering, but it can cause reflex tachycardia and decreased blood pressure, especially if the patients are volume depleted; cardiac risk factors and the volume status must always be taken into account when deciding upon a therapy.

32.2.3 Which Medical Errors Do You See in the Presented Case?

32.2.3.1 Laboratory Values

Before the planned radiological intervention, a complete blood count was done. The abnormal values therein were not noted by any of the physicians, until physical impairment from tachycardia and dyspnea arose. The symptoms caused the patient to undergo a thorax angio-CT, which was normal. Thereafter, seemingly satisfied with the CT result, no further investigations were done. Dr. Perk should have viewed patient's file a bit more carefully before inducing anesthesia.

32.2.3.2 Extubation

Dr. Perk removed the endotracheal tube. Mrs. Smith was not adequately responding, which means that presence of airway reflexes cannot be assumed. In addition, the extubation was done immediately before transferring the patient into the PACU. Every transport endangers the safety of the patient. What prevented the transferring Mrs. Smith to the PACU intubated?

32.2.3.3 Delayed and Insufficient Therapy of Shivering and the Search for the Causes Thereof

In the PACU, Mrs. Smith shivered for at least 90 min with impaired mental status. A quick and thorough search for the cause was not done; routine therapy for shivering was given instead of evaluating the patient.

32.2.3.4 Monitoring

Monitoring of body temperature is required in every anesthetic, in order to detect and prevent the loss of heat. Despite the long duration of the intervention, over 2 h, Mrs. Smith's temperature was not even measured in the PACU. Active warming was done without monitoring the outcome of the therapy.

32.2.3.5 Hyperglycemia

The topic of this case is not perioperative adjustment of insulin in diabetic patients. However, as you know from your daily work, desires (orders from the anesthesiologists) often clash with reality (orders not carried out) on the ward. If the patients are in our custody, we cannot ignore hyperglycemia, especially when a decreased level of consciousness is present, as in this case.

32.2.4 Which Systems Failures Can You Find in the Presented Case?

32.2.4.1 Communication Between the Two Services

Only by reviewing the medical record did Dr. Perk discover that an angio-CT was done. There was no transfer of information whatsoever from the neurosurgery service. Maybe the leukocytosis wasn't even noticed by the service at all.

The CT ordered by Dr. Doxa was indicated and the right thing to do. There was a possibility of recurrent intracerebral hemorrhage. Dr. Doxa neglected to ask the patient's neurosurgeon or neuroradiologist for their opinion. The same goes for Dr. Perk, because Mrs. Smith already displayed abnormal reactions in the neuroradiology suite.

32.2.4.2 Help in PACU

There was a whole lot to do in the PACU. There were surgical complications and lots of patients. Mrs. Smith was the victim of this work overload, because Dr. Doxa's resources were exhausted. Despite this, not one call for support from additional personnel was made.

The same goes for calling for help from other anesthesiologists. At the very least, assistance should have been requested when Dr. Doxa began to concern herself intently with Mrs. Smith. The anesthesiologist-in-charge, Dr. Eldridge, stopped in the PACU just by chance.

32.2.4.3 Monitoring

Temperature monitoring must be part of the standard. It is hardly invasive and is easily done.

32.2.5 "Anesthesiology Department, Please Hold, Someone Will Be with You Shortly!"

The standard phrase from a hotline – who hasn't been aggravated by having to wait? Standards are sensible and helpful. Unfortunately, they cannot take all circumstances into account. It may be necessary to deviate from a standard, because even the best standard is only appropriate in 90 % of the cases.

Standards lead to laziness – especially mental laziness. We tend to save our resources, save room in our brain for other (more important?) things. Administration of meperidine was perhaps not an official standard, but Dr. Doxa raised it to the level of an internal standard. Inflexibly holding onto a standard might be worse than tunnel vision (see Sect. 15.2.4) because it's not just holding one direction of thought, but giving no thought at all to the current situation.

What can you do to prevent this and not be caught as a victim of mental laziness? You could become a virtual employee for Toyota and ask "Why?" Toyota has developed a technique called the "5 times why" process. In it, one asks and answers "why" five times, in order to identify the true cause of the problem hidden behind the symptoms, which can only be discovered by intense analysis.

What does all this have to do with standards? Ask five times "Why are there standards?" and you will find few reasons to deviate from standards in most cases.

Always think about it: Someone developed the standards, but they can be changed. Standards are created from knowledge and experience and they are not an unabandonable dogma. Therefore, your doubts about alternatives can be nutrients for revision of standards. Remain critical towards *your own* standards. Ten percent of patients will thank you!

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Case 33: Glioblastoma

33

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Contents

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33.1 Case Introduction

>> Dr. Spiegel made it to work just in time. Her husband was on a business trip, so she had to get the kids ready all by herself this morning. Neither of them was too excited about day care today, but finally she got them there.

Dr. Spiegel was assigned to neurosurgery at the moment, and her feelings towards it resembled that of her kids' feelings towards day care. The reason was the view, or lack thereof, just sitting in a dark room all day watching neurosurgeons do their difficult work. She was an experienced anesthesiologist, and anesthesia was no longer a challenge for her.

Mr. Sanford was on the schedule for today, with an intracerebral mass suspected to be a glioblastoma. A colleague had performed the preoperative evaluation yesterday and reported:

- *66-year-old patient*
- *174 cm, 65 kg*
- *Diabetes, treated with insulin and metformin. Metformin held 2 days ago*
- *Chronic renal failure, compensated*
- *Systemic hypertension, treated with valsartan and hydrochlorothiazide*

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- *Asthma, treated with an inhaled β2 sympathomimetic*
- *Hypothyroidism, treated with L-thyroxin*
- *Lab values normal except for increased creatinine*

33.1.1 What Are the Important Anesthetic Considerations for Patients with Brain Tumors?

33.1.1.1 Airway

In all surgeries, care must be given to the head and neck area; in cranial procedures, access to this area is especially restricted (see Sect. 15.1.2). Use of a non-kinkable or reinforced endotracheal tube is recommended. In intracranial procedures, the head is usually held tightly by pins to immobilize the surgical area. Quick removal of the fixation device during the operation is not very possible – dislocation of the tube would be catastrophic. Placing the pins in the skull is very painful, and depth of the anesthesia must be carefully monitored.

33.1.1.2 Positioning

In the anesthesia planning, one must take note of which position in which the surgery will occur. Due to the usually long surgical duration, great care must be given to the positioning. This applies not only to the torso and limbs but also to the eyes [5].

33.1.1.3 Monitoring

More extensive circulatory monitoring is always indicated and includes invasive arterial blood pressure and a urine catheter. In long craniotomies or those with extensive volume shifts, a central venous catheter is also indicated. Surgeries performed in a sitting position should include a right atrial air aspiration catheter and transthoracic echocardiography or Doppler sonography to detect air embolism.

33.1.1.4 Choice of Anesthesia

Even when current research is not completely definitive, with space occupying mass many anesthesiologists (and anesthesiology books) recommend total intravenous anesthesia (TIVA) with propofol and remifentanil [3]. All volatile anesthetics and nitrous oxide have the potential to increase intracranial pressure in a dose-dependent manner. The underlying mechanism is cerebral vasodilation and a subsequent increase in cerebral blood flow.

Complementing the TIVA with muscle relaxation is often sensible to prevent spontaneous movement. However, muscle relaxation should not be used if motor mapping or motor evoked potential monitoring is planned.

33.1.1.5 Atropine Administration

Atropine offsets the remifentanil-induced bradycardia (see Sect. 31.1.5). It is a parasympatholytic and competes on the muscarinic receptors of the parasympathetic nervous system with the neurotransmitter acetylcholine (see Sect. 28.1.5). High cerebral acetylcholine levels induce seizures. Atropine crosses the blood–brain barrier and increases the seizure threshold [4], so that fewer seizures are observed postoperatively. However, despite these potential advantages, atropine is generally not administered prophylactically and instead is only used in the treatment of profound excessive bradycardia.

For the same reason, atropine administration during electroconvulsive therapy should be avoided, since an increased seizure threshold is not desired.

33.1.1.6 Dexamethasone Administration

High-dosed perioperative dexamethasone reduces perifocal cerebral edema. Administration must be done with discussion with the surgeon. Serum glucose levels often increase after dexamethasone administration, and glucose levels should be checked frequently intraoperatively.

33.1.1.7 Emergence and Postoperative Monitoring

Many patients have reduced consciousness after intracranial procedures. Despite this, or perhaps because of this, it is important to evaluate the patients (be able to evaluate) very soon after surgery. During emergence, the common technique of allowing CO_2 to accumulate as a respiratory stim-

ulus cannot be employed unless there is a large amount of room in the head after the resection, since hypercapnia increases intracranial pressure.

Postoperatively, the patients must be observed closely. The monitoring can be done on an intermediate care or intensive care ward.

>> Dr. Spiegel was greeted by an anesthesiology technician named Roger. "*Morning*! *Um, due to another emergency – I was unable to complete preparations here. Please give me a few more minutes*." "*Not a problem*," *said Dr. Spiegel*, "*I'll just go say hello to Mr. Sanford*." *All the nurses loved Dr. Spiegel, especially her gentle and empathetic working style.*

"*Good morning Mr. Sanford*" *she said to her patient.* "*My name is Dr. Spiegel and I will be taking care of you today. Have you decided upon a nice dream to have while you are asleep*?" *Mr. Sanford smiled back*, "*I would like to take my grandkids to the zoo, take them for a ride on a big steam engine*."

"*Oh, really*?" *responded Dr. Spiegel.* "*How old are the grandkids*?"

"*3 and 4*," *he replied.*

"*Wow – what an incredible age*! *My kids are 3 and 5, and they discover something new every day. They love the days with their grandfather more than anything*!" *Dr. Spiegel was beaming. Then she began to hook Mr. Sanford up to the monitor.*

"*So*," *she finally said to her patient*, "*we've got the blood pressure and ECG. Now for the little pricks*." *Dr. Spiegel pulled the tourniquet tight and searched for a vein on the back of his hand. They were all very thin.* "*You look a little pale, are you nervous*?" *she asked.* "*No, but I feel a little strange*," *replied Mr. Sanford.*

Dr. Spiegel squeezed his hand, "*Then just think about your grandkids. Soon you will be sound asleep, dreaming nice peaceful dreams*."

Dr. Spiegel noticed that Mr. Sanford was cool and clammy and had very labored breathing. "*Mr. Sanford, what are you feeling right now*?" *He replied with effort*, "*Not doing too well – I can't breathe*!"

Dr. Spiegel quickly said to the tech, "*Roger – give me the albuterol*!"

33.1.2 How Do You Define Bronchospasm?

Bronchospasm is reversible contraction of the airways, caused by bronchial smooth muscle contraction. The result is an increase in airway resistance, with the characteristic prolonged expiration. Mucus membrane secretions may further worsen the symptoms. Crackles are typically heard. More severe bronchospasm can result in hypoxia and hypercapnia. Breath sounds may barely be present, hence the name "silent lung."

33.1.3 What Is Time Constant τ (tau) and What Does It Have to Do with Asthma?

The time constant τ is calculated from the products of compliance (C) and resistance (R):

$$\tau = C \bullet R \tag{33.1}$$

It is a measurement for the time needed to exhale air. The unit is seconds. The normal value in healthy patients is 0.2 s, in ventilated patients 0.3 s. After one time constant 63 % of air is exhaled, after three time constants 95 %, and after five time constants more than 99 % of inhaled air is exhaled.

Patients with chronic obstructive lung disease (COPD) have an extended expiration time with a higher time constant. The value is usually about 0.9 s.

Ideally, 5 time constants should be available to exhale (4.5 s for an asthma patient). If the expiration time is less than 3 time constants, air remains in the lungs. This air trapping results in overinflation of the lung and development of an auto-PEEP. The danger of auto-PEEP formation is especially high in COPD patients, if the set expiratory time is not long enough.

33.1.4 What Are Typical Triggers for Bronchospasm?

Typical triggers include:

- Allergic reactions
- Pulmonary infections

- Exertion [2] (the so-called "Tour de France" asthma)
- Manipulations done in and around the airways (e.g., insufficient depth of anesthesia)
- Chemical stimuli (e.g., aspiration, irritating gases)
- Medications, e.g., noncardioselective β-blockers, cholinesterase inhibitors, and NSAIDS

Apart from that, bronchospasm may be seen as a result of psychological stress in susceptible patients.

>> *In the meantime, tech Roger helped Mr. Sanford to sit up and administered 2 puffs of albuterol. "I'm gonna be sick," Mr. Sanford said after he received the sprays.*

"*Hopefully it's not a myocardial infarction*!" *thought Dr. Spiegel. "Do you have any chest pain*?" *she asked. Mr. Sanford shook his head. "Oh, man," she thought. "Why does this always happen to the nicest patients*?" *She auscultated his lungs; breath sounds were normal. "Can I lie down again, doc*?" *was all she heard.*

"*Do you think it's a myocardial infarction*?" *Dr. Spiegel asked, turning to tech Roger. "We could give nitroglycerin and see if it helps*."

Tech Roger shook his head. "I don't think so," he said, as he started the blood pressure. The measurement appeared as the alarms went off, showing the following values:

- *Blood pressure 70/40 mmHg*
- *Heart rate 90/min*
- S_PO_2 *92 %*

33.1.5 What Would You Do Now?

The first suspected diagnosis, asthma attack, seems to have been incorrect. Mr. Sanford is now:

- Diaphoretic
- Short of breath
- Hypotensive

The diaphoresis and the dyspnea can be caused by the hypotension. In the presented case, before a search for the cause of hypotension is done, symptomatic therapy needs to be started, with volume administration and vasopressors.

>> *Mr. Sanford received phenylephrine and 1,000 ml of a crystalloid solution IV. His blood pressure stabilized at 110/55 mmHg. Mr. Sanford felt much better. "Thanks, Roger. I'll call the anesthesiologist-in-charge now," said Dr. Spiegel.*

33.1.6 What Is Your Differential Diagnosis?

Difficult, very difficult to determine. We just don't know enough at this time, but all too often, that's the way it is in real life.

33.1.6.1 Reflex Hypotension

Reflex hypotension and bradycardia can occur in very upset or nervous patients. Usually the patients are younger.

33.1.6.2 Cardiogenic Shock

Dr. Spiegel might have been right about her cardiac suspicions. An acute coronary syndrome could explain the symptoms. Pain is not necessarily present, especially when the patients are diabetic.

33.1.6.3 Hypovolemia

Hypovolemia may cause hypotension. Whether or not the preoperative fasting state in combination with the diuretics is sufficient to explain the symptoms is debatable.

33.1.6.4 Pulmonary Emboli

Thrombosis and pulmonary emboli are the most feared complications of every surgeon. Occurrence before an operation is not impossible, but unlikely in patients without additional risk factors.

33.1.6.5 Hypoglycemia

Hypoglycemia must be considered as the cause of the symptoms in diabetic patients.

33.1.6.6 Anaphylactic Shock

Even though Mr. Sanford didn't have bronchospasm, the symptoms could have been caused by an allergic reaction (see Sects. 14.2.1 and 14.2.2).

33.1.6.7 Septic Shock

Once you know the definition of sepsis, you can explain almost all symptoms as being due to sepsis (see Sect. 25.2.2).

33.1.6.8 Neurogenic Shock

An intracerebral mass can cause neurogenic shock. The possibility of this being the cause of the symptoms in the presented case is unlikely. Mr. Sanford is simply doing too well neurologically.

>> *Anesthesiologist-in-charge Dr. Eldridge walked into the preop holding area and listened to the recount of the situation. In the meantime, Roger had done a 12-lead ECG and a glucose. Both were normal/unchanged from the previous readings. Axillary temperature was 35.9 °C, and glucose was 105 mg/dl (reference 70–99 mg/dl).*

Dr. Eldridge examined Mr. Sanford, without making a new discovery. Then he reviewed the patient's medical record on the computer. "Have you seen the labs?" he asked Dr. Spiegel. "Everything is normal except the increased creatinine," she responded.

"Not exactly," said anesthesiologist-in-charge Dr. Eldridge. "The C-reactive protein (CRP) yesterday was 232 mg/dl. Maybe Mr. Sanford has an infection. My best differential diagnosis is an allergic reaction. Acute myocardial infarction and pulmonary emboli must be ruled out, even though I think they are unlikely. Put in an arterial line under local anesthesia so we can carefully monitor the blood pressure. I'll call the neurosurgeon and discuss postponing the surgery until we find out what is going on here."

33.1.7 Which Parameters Would You Determine?

33.1.7.1 To Rule Out Acute Myocardial Infarction

Lab values: troponin, creatine kinase (CK), CK-MB (heart specific portion of the CK), glutamate oxaloacetate transaminase (GOT), lactate dehydrogenase (LDH)

The lab values are initially negative in an acute myocardial infarction; additional measurements are required. Troponin is the first parameter to increase.

33.1.7.2 Pulmonary Emboli

Lab values: D-dimers, proBNP

Increased D-dimers could be a clue of a thrombosis, but can also be found in many other diseases like infections and tumors. proBNP is a marker of stress on the right side of the heart.

33.1.7.3 Infection/Sepsis

Lab values: complete blood count with differential, CRP, procalcitonin, blood culture

Procalcitonin is a precursor of the hormone calcitonin, which is formed in the C cells of the parathyroid gland. An increase of procalcitonin can have many causes. The value in the diagnosis of sepsis is debated [6]. High procalcitonin levels (>10 ng/ml) are a clue that sepsis is likely; low levels cannot exclude sepsis from the differential.

33.1.7.4 Anaphylaxis

Mast cells play a key role in anaphylactic shock. After activation, they release various mediators, the most important of which are histamine and tryptase (Fig. 33.1). Tryptase is an enzyme with many purposes, which are only partly understood. Known effects are:

- Activation of complement C3
- Sensitizing smooth muscle to the effects of histamine
- Activation of eosinophilic granulocytes
- Induction of kinin formation

After anaphylactic shock, there is a temporary increase in serum tryptase, which lasts from 15 min until 14 h after the attack. Persistently high tryptase values can be an indication of mastocytosis.

The increase of the histamine level which is observed after anaphylactic shock is difficult to diagnose, being that it is so short lived. The blood samples must be analyzed within the first hour – preferably within 30 minutes.

>> *Dr. Spiegel was not sure which tests Dr. Eldridge recommended. The arterial line was in, and arterial blood gas analysis was unremark-*

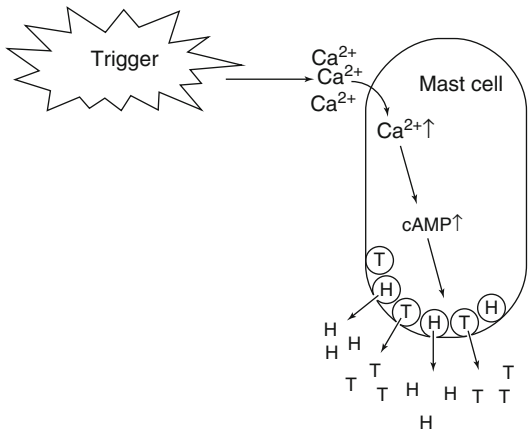

Fig. 33.1 Schematic representation of histamine and tryptase degranulation from mast cells. A trigger causes calcium to flow into the cell and activate cAMP, which causes histamine and tryptase to be released from the intravascular vesicles (*H* histamine, *T* tryptase)

able. "*We better repeat another CBC*," *Dr. Spiegel said to tech Roger*.

Tech Roger packed the order form and the tubes of blood into the transfer bag as the curtain in the preoperative holding area opened. Dr. Eldridge didn't look very happy. "*I spoke to the neurosurgeon, he said that he is not 100% sure about the glioblastoma diagnosis. His most important differential diagnosis was a cerebral abscess, which is why he didn't want to wait any longer. Start the anesthesia and let me know if you need any help*." *With that, Dr. Eldridge was gone. Roger shook his head*, "*I don't believe that either*."

"*Doctor, is everything OK with me*? "*asked the patient*. "*You don't need to worry about a thing, Mr. Sanford*," *replied Dr. Spiegel*. "*We'll take you to the OR and begin the anesthesia*." *After monitoring and preoxygenation in the OR, Dr. Spiegel pushed her induction drugs as she told the patient*, "*Think of your grandchildren. Dream wonderful dreams*."

Induction of general anesthesia and placement of a central vein catheter went without incident. "*That's a relief*," *Dr. Spiegel said to Roger*. "*I had a bad feeling about this – I got nervous*." "*I noticed*," *said tech Roger*, "*sometimes it is difficult when you want everything to go perfectly*."

Mr. Sanford received low doses of phenylephrine and some crystalloid solution. The blood pressure and heart rate were normal. The neurosurgeon entered the OR. "*The attending-in-charge called me about the problems. Everything OK now? There's not a high possibility of a brain abscess, but we won't know for sure until we get the tissue under the fluorescent microscope. Please wait with the dexamethasone and the antibiotic until I decide*."

33.1.8 What Do You Know About Fluorescence-Assisted Resection of Malignant Tumors?

The principal of fluorescence-assisted resection of malignant tumors requires administration of a pro drug which is absorbed by tumor cells and is then transformed into a fluorescing molecule. The resection is then done with a special light source, which fluoresces up the tumor cells, aiding in detection. The technique is used for identification of epithelial tumors and is well established in dermatology and urology (bladder tumors). In neurosurgery, the technique helps to remove more of the tumorous tissue, without removing healthy brain tissue. The technique increases chances of survival [7].

The florescent substance used is 5-aminolevulinic acid (5-ALA). 5-ALA is a natural byproduct of porphyrin metabolism, which is important for heme biosynthesis and for cellular respiration. If 5-ALA is present in excess, it accumulates as a fluorescent hemoglobin protoporphyrin IX.

33.1.9 Bet You've Been Waiting on This Question: What Are the Side Effects of 5-ALA?

Now you're probably thinking: Am I a pharmacologist – do I have to know that? What consequence could that possibly have for my anesthesia?

Our answer: No, of course not, you must not know the side effects of 5-ALA, unless, of course, you care for patients who have received the substance.

Our intention with this question was more than flat detail transfer; we want to sensitize you to the importance of looking over the sterile drapes and concerning yourselves with the surgical aspects of the operation, which may appear to have nothing at all to do with the anesthesia. Are you still interested in the answer? OK, here it comes:

A specific side effect of 5-ALA is a decrease in the systolic and diastolic blood pressure and reduced pulmonary arterial and peripheral resistance. Refractory tachycardia is to be expected [1]. The exact mechanism is not clear, but upregulation of heme oxygenase, a heme degradation enzyme, is known to cause hypotension due to CO_2 formation [8].

Apart from that, 5-ALA has additional nonspecific side effects such as an increase in liver enzymes, changes in the CBC, nausea, and diarrhea.

>> The surgery went as planned. Dr. Spiegel checked her meds reference for 5-ALA as soon as she had a free moment. Then she called the anesthesiologist-in-charge: "*It is all very interesting, and I think you might be right. We will wait until we have the lab results*." *Shortly thereafter, the labs came in*:

- *Leukocytes*: *6,800 cells/l* (*norm*: *3,600–9,800 cells/l*)
- *CRP*: *148 mg/dl* (*norm*: *<10 mg/l*)
- *Procalcitonin*: *<0.1 ng/ml* (*norm*: *<0.005 ng/ml*)
- *D-dimers*: *2,477 ng/ml* (*norm*: *negative*)
- *CK*: *0.32 µmol/l* (*norm*: *<3.17 µmol/l*)
- *CK-MB*: *0.13 µmol/l* (*norm*: *<0.41 µmol/l*)
- *Troponin*: *<0.001 µg/l* (*minimal value for a MI*: *0.10 µg/l*)
- *proBNP*: *12.9 pmol/l* (*norm*: *<14.5 pmol/l*)
- *ALAT* (*GPT*): *251 IU/l* (*norm*: *<17 IU/l*)
- *ASAT* (*GOT*): *108 IU/l* (*norm*: *<15 IU/l*)
- *γGT*: *217 U/l 3.62* (*norm*: *<71 IU/l*)
- *Tryptase*: *8.08 pg/l* (*norm*: *<11.4 pg/l*)

33.1.10 Considering the Suspected Diagnosis, How Would You Interpret the Lab Values (See Sect. 33.1.7)?

Acute myocardial infarction *can be ruled out*.

The lab values speak against this diagnosis. To be safe, the test should be repeated after 6 h.

33.1.10.1 Pulmonary Emboli

The D-dimers are increased, but this is very nonspecific. The low proBNP values exclude stress on the right side of the heart. Serious pulmonary embolism is highly unlikely.

33.1.10.2 Infection/Sepsis

Lack of leukocytosis, decreasing CRP, negative procalcitonin. A diagnosis of sepsis is also unlikely.

33.1.10.3 Anaphylaxis

The low tryptase makes an allergic reaction unlikely.

>> Intraoperatively, no cerebral abscess was found. Resection of the glioblastoma went well. "*Thank goodness*," *thought Dr. Spiegel. Having worked all day today in a darkened room had been stressful for Dr. Spiegel, even though she was a board-certified anesthesiologist.*

The surgery and the anesthesia ended without incident. The next day Dr. Spiegel surprised Roger with a cake. "*You were of such great assistance to me yesterday. You saved me from making some big mistakes*." *Roger was visibly pleased.* "*Always my pleasure. Now you must share a piece with me, or else I will consider this bribery*."

33.2 Case Analysis/Debriefing

33.2.1 Tryptase Values Have a Specificity of 92 % and a Sensitivity of 35 %. What Do Sensitivity and Specificity Mean Again?

33.2.1.1 Specificity

When a test has a high specificity, it means that the probability is high that patients without the disease will be identified. A specificity of 92 % means that out of 100 healthy people, 92 will be identified as healthy.

High specificity is desired when a disease should be identified with a high degree of certainty.

33.2.1.2 Sensitivity

Alternatively, high sensitivity is desired when a disease should be ruled out with a high degree of certainty.

High sensitivity means that the probability of identifying disease is high. A sensitivity of 35 % means that out of 100 people with a disease, 35 will be identified as having that disease.

33.2.2 What Does That Mean for Our Current Case?

Two things shouldn't be confused here: the specificity and sensitivity of the laboratory cases, and the clinical interpretation of the results.

A sensitivity of 35 % for a laboratory test would hardly be enough to be useful in a hospital. In other words, this would mean that out of 100 blood samples with elevated values, only 35 would be identified as having the elevation. The value of the specificity and sensitivity depends on its clinical meaning:

Read once again, the definition of specificity and sensitivity in Sect. 33.2.1. Calculated for our case, this means: of 100 patients without an allergic reaction, 92 will be correctly identified as having normal tryptase levels. Eight patients with elevated tryptase values do not have an allergy.

Now the sensitivity comes into play: 8 patients have elevated tryptase values, but only three (about 35 %) also have an allergic reaction.

>> *We will not dive deeper into statistics, but we want you to remember these clinical tips for dealing with sensitivity and specificity*:

- *If the sensitivity is low (as in this case), a positive test result barely helps at all.*
- *If the specificity is high (as in this case), a negative test result can be helpful to rule out a diagnosis. But it can't do anything else.*

33.2.3 Which Medical Errors Do You See in the Presented Case?

33.2.3.1 Physical Exam

As Mr. Sanford deteriorated clinically, Dr. Spiegel's first reaction was to give the β2-mimetic, which she did without auscultating the lungs. Auscultation would have been the fastest and easiest way to make a diagnosis.

33.2.3.2 Measurement of Glucose

Clammy skin together with vague feelings of malaise should always indicate the need for a glucose check – especially in diabetics.

33.2.3.3 What Are the Side Effects of 5-ALA?

Either Dr. Spiegel did not prepare herself for the specifics of the surgical procedure or she missed the part about what to watch for in patients who have received 5 ALA. A further topic was not even discussed here: photosensitivity.

33.2.4 Which Systems Failures Can You Find in the Presented Case?

33.2.4.1 Information Exchange Between Anesthesia and Neurosurgery

Having "brain abscess" in the differential diagnosis is very important. Unfortunately, the neurosurgery team neglected to inform the anesthesia colleagues of the possible abscess in advance. Unfortunately, there are very few hospitals that have an interdisciplinary discussion of scheduled surgery. Maybe we can't make it to happen, but it is desirable.

33.2.4.2 Consultation with Senior Anesthesiologists

In many cases, the anesthesiologist-in-charge Dr. Eldridge reacted correctly, but not in this one. He did not fully respond to Dr. Spiegel's request for help. Clear directions and an open discussion of the differential diagnosis as a team would have been desirable.

33.2.5 Have You Even Had Patients Who Grew Especially Close to Your Heart?

Then perhaps you were empathetic! **Empathy** is a behavior that makes it possible for us to share the feelings of another person and to better understand this person. By doing so we are not taking sides with the person or justifying their behaviors, we are just acknowledging their emotions in order to bring ourselves closer to understanding the person, in order to better react to the patient's needs and wants.

Feeling empathy is an important prerequisite for a relationship between two people, because many communication signals are sent nonverbally on an emotional level (see Appendix, the meaning of the name "Spiegel"). If we can recognize the emotions of others, then we can also understand them. It is good and it is important to be empathetic. Empathy is a sign of a good doctor: someone who understands another person.

It is also important to be aware of the emotions that are being elicited from us, since such emotions can immensely affect our decision-making.

The result is a deficiency in boundary setting. This is especially pronounced when our own personality traits are projected onto the patients. For example, one may imagine that the pediatric patient is one's own child, or one may realize that they would be very afraid in such a situation.

Patients expect, and have a right to expect, professional demeanor. Professionalism in this case means that we can understand the patient's feelings, without losing control of our impulses. How can we reach this?

33.2.5.1 Self-Reflection

Imagine the patients as the opposite of how they actually are. Change your perception of a young thin male patient into an old, fat white female patient. Are your feelings still the same? With this trick, you can expose your hidden motivations.

33.2.5.2 Professionalism as a Guiding Principle

Observe yourself from the outside and ask yourself the following questions:

- "Am I professional?"
- "Can I keep boundaries with my patients?"

A prerequisite to answer this question is that you have done some self-reflection.

Be observant, because a danger called reactance is lurking. Reactance behavior is often seen when one notices that one is caught emotionally in a positive view of another person and then one loses the ability to freely decide (e.g., the freedom to decide). The result is the tendency to change to the other extreme. For this reason, teachers should never teach their own children.

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Tips to Reduce Human Errors

34

Mike Hänsel and Matthias Hübler

Content

34.1 Can You Remember Each Question at the End of Each Case?

Nontechnical and psychological aspects have an important influence on the quality of our work and our job satisfaction. Therefore, we took one aspect per case and presented it as an example. Our goal was not to list out what the person had done wrong, but to give you tips to avoid similar mistakes, or to better master similar situations.

Table 34.1 summarizes the psychological factors presented in all 33 cases. On the left side, the human factors are listed in alphabetical order with a key word and a short description.

The third column adds examples from the cases. The right column lists examples of strategies to solve the problems. This listing is in no way complete, but perhaps you may remember one or two during your work day, which may help you to better manage a difficult situation.

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Table 34.1 Tips to reduce human error

Human factors	Description	Examples	Possible problem-solving strategies
Actionism	Spontaneous acts without consideration of goals Thereby neglecting to perform necessary steps and overlooking relevant information	**Case 24** Extubation of a patient during excitation phase with subsequent low pressure pulmonary edema	**Conscious formation of goals** Setting of primary and secondary goals and the criteria needed to fulfill each Priorities and goals must be communicated within the team
Activation level, low	Due to low level of personal activation, procedures were forgotten	**Case 21** Lack of oversight of volume administration during a colectomy	**Self-initiated stimulation during boring phases** For example: conscious anticipation of the worst-case scenario or preoccupation with solutions for fictitious problems
Attitude, overly optimistic outlook	Due to the readiness to take risks with the attitude "Nothing will go wrong," no serious consideration is given in advance to serious possible complications After an emergency arises, no alternative is available	**Case 19** No anticipation of nose bleeding in fiberoptic nasal intubation **Case 25** No consideration of the connection between the inguinal hernia and the sepsis	**Metacognition** Reflection on our own assumptions and motivations for the actions we take After recognizing a dangerous assumption → the antidote is to verbalize "Complications arise with my patients, too."
Attitude, state of resignation	Overwhelmed to the extent of mentally resigning from attempting to work anymore	**Case 29** Endangering the patient during transport to CT	**Critical self-reflection** Define and practice a stimulus for self-reflection: review the state of events out loud, ask for hints from the team members, pay special attention to advice
Availability heuristic	Conclusions are drawn from the diagnosis in most recent memory	**Case 20** Low S_pO_2 values were only associated with hypoxia	Conscious information search, to find details which go against your own assumptions
Cognitive economics	Due to the scanty information processing, medications were mixed up during a routine situation	**Case 10** The medication mix up lead to an overdose of ropivacaine, and consequently, infant CPR	Make sure you remain alert Work through checklists Check one another Question assumptions Read back medications before administration Don't make labels easy to confuse
Communication, unclear	Lack of shared language between various specialties leads to misunderstandings	**Case 26** Incorrect interpretation/meaning of the definition of infrarenal aortal aneurysm	**Establishment of a shared mental model** Communication of top priorities and delegation and coordination of tasks within "the big team" **Necessary requirements**: One person must be designated as being responsible, usually of their own initiative **Verbalization of findings out loud in front of the whole team** Thereby, possible recognition of various interpretations

Decision-making, emotional	Simple coherences are not comprehended due to fully emotional decision-making	**Case 23** The danger of a tube fire was not calculated, because the S_PO_2 decrease caused the O_2 to air ratio to be increased	**Step back** Consciously take a step back and bring yourself mentally out of the situation. Then examine the situation again, mentally stress free – this allows an emotional trigger to be recognized Practice the step-back ritual in advance **Metacognition after performing a step back** (thinking about your own thoughts) Question your own hypothesis Critically recheck your assumptions Search for information which could verify the worst-case scenario
Decision-making, under time pressure	When pressured for time, treatment options are forgotten or risks are not considered. The basis for the decision becomes incomplete/ incorrect	**Case 3** The hypertension was associated with the pheochromocytoma long after it should have been	**Assistance in decision-making** For example, the FORDEC model used in aviation → by working through all letters, complete reevaluation of the foundation for decision-making is possible
Empathy	Psychologically put yourself in another person's shoes	**Case 33** An anesthesiologist projects her father onto her patient	**1. Self-reflection** Imagine the patients as exactly the opposite of what they are (young and thin as opposed to old and obese). Are your feelings towards the patient still the same? **2. Professionalism as a guiding principle** Do I work professionally?
Goal setting	Due to uncoordinated goal setting between many specialties, there is divergence of those goals within the big team In emergency, lack of support from the other specialties	**Case 5** TURP syndrome is challenging for any anesthesiologist, but not for urologists **Case 11** The extent of the Palacos reaction is not shared with the orthopedic surgeons	**Establishment of a shared mental model** Communication of the top priorities as well as delegation and coordination of duties are aspects of "one big team" **Important requirement**: there must be one person designated as being responsible for the patient (usually designated through one's own initiative)
Information overload	Reduction of the amount of information comprehended due to the overload → whereby information, possibly essential, is lost	**Case 2** After transfer of an ICU patient, the information about the cerebrospinal fluid drain was forgotten	Repeat relevant information out loud as often as possible In transfer situations, summarize relevant information and obtain confirmation from the person doing the transfer Resource management: recruiting additional resources – more is heard by four ears than by two
Macho personality	"I have everything under control, regardless of what happens" leads to negligence and endangers the safety of patients	**Case 18** Carrying out peripheral regional anesthesia techniques without appropriate monitoring	Selection of staff members Only partly under our control: personal development (trainings, supervisor assessments, sanction)

(continued)

Table 34.1 (continued)

Human factors	Description	Examples	Possible problem-solving strategies
Memory loss, prospective	Treatment intentions are forgotten, which were decided upon at an earlier time point and should have been later carried out	**Case 17** Forgotten nerve stimulator for a patient with myasthenia gravis	**Open and clear communication of the planned steps to be carried out** (team members are then brought into the picture) Verbal elaboration, so that the intentions are kept fresh in mind Use external memory assists Continually reevaluate the situation External memory assists (checklists, etc.)
Perfectionism	Increased willingness to take risks, and refusal to accept assistance due to an elevated expectation of one's self, and fear of being seen negatively in another's eyes	**Case 6** The anesthesiologist wants to show off to the team during the pediatric anesthesia	**Establishing a culture of error** Fair, non-punitive acceptance of errors Norms and values must be known to all team members Supervising physician must function as a role model and educator
Presenteeism	A presentee is someone who goes to work sick	**Case 31** The anesthesiologist takes medications to be fit enough to continue working	**Employees** Stay home when sick! Don't take medications in order to make yourself capable of working! **Supervisors** Show a good example! Send your sick employees home! Pay attention to your sick statistics!
Protection of competency	The choice of information to be perceived and the treatments carried out are oriented around the feeling of security Information which threatens one's feelings of competency is suppressed	**Case 28** Avoidance of the diagnosis "malignant hyperthermia," therefore refusal to accept help	**Conscious attention paid to the worst-case scenario** Thereby reflection on one's tendencies to avoid
Representativeness heuristic	The more similar the situation is to prototype situation, the sooner the situation will be assumed to be a prototype situation, even though it may be very different	**Case 22** Hypertension was not associated with tourniquet pain	Conscious search for information which goes against personal assumptions
Responsibility, diffusion of	Necessary tasks are neglected due to unclear delegation of responsibility	**Case 9** Before beginning anesthesia in a high-risk patient, the anesthesiologist did not inform her attending physician	**First-line management** Targeted classification of responsibility and establishment of standards for use with concurring positions **Team level** Actively obtaining information in parallel responsibility areas Without double checking, never assume that the task was completed (e.g., that the supervisor was informed)

Role conflict	Fulfilling a new leadership position makes communication within the team much more difficult	**Case 14** Turning away suggestions due to the hierarchy of a lower ranking team member	**Answer the following questions**: What makes a good leader? How far does my role fulfill the definition? What do I have to do to fulfill the definition? Does my team know about my definition of my new role? Have I communicated to the team that I am still a part of the team and, as a team leader, do not take hierarchy into consideration when presented with medical questions?
Situational consciousness, lack of	Due to inadequate interpretation of information, incorrectly holding onto inaccurate treatment goals	**Case 16** One-sided intubation was not noticed, due to the misinterpretation of the end expiratory CO_2	**Metacognition following step back** (thinking about your own thinking) Questioning your own hypotheses Critically checking your assumptions Search for information to verify the worst-case scenario
Standards	Hold fast to set or self-made standards	**Case 32** The anesthesiologist in the PACU went along with the standard diagnosis of shivering and treated accordingly	**Ask why** Via continual questioning of why, the standard will be questioned, and simultaneously, a glimpse of exceptions to the standard comes into focus
Tunnel vision, selective search for information	Primarily a search for information which reinforces one's own assumptions	**Case 12** Fixation on a postoperative residual curarization **Case 7** The anesthesiologist forgot the option of intraosseous puncture in emergency **Case 15** Delayed opening of the intramaxillary fixation after a maxillofacial procedure	**Verbalization of the problem out loud** For example "Why doesn't the nerve stimulator work here?" Through the verbalization, thought processes are activated which bring the problem into new light Simultaneously, the problem is made public to the other team members. They can then involve themselves in the problem solving Alternatively, a review of the past events occurs, which helps to illuminate mistakes, which may include having been fixated too long on the wrong aspects **Step back – consciously going back a step** Mentally taking yourself out of the situation, and reevaluate, unstressed Through reflection, emotional triggers can be recognized In advance, create rituals for the step-back technique

Table 34.1 (continued)

Human factors	Description	Examples	Possible problem-solving strategies
Work motivation, minimal	Overtime hours running into personal time Depth of motivation and assumptions that endanger safety: "Everything is normal – nothing is going to happen!"	**Case 25** The planned movie after work blurred clear thinking	**Organization** Communicate overtime hours in advance **Individual** Operating room planner should ask for a replacement as soon as possible. Prerequisite: team culture, in which needs can be communicated
Work schedules, inflexible	Inflexible work schedules cause additional unnecessary time pressures	**Case 4** The PACU physician is overwhelmed by the amount of work to be done	Time management Recruiting additional resources Prioritization Setting goals together

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