Emergencies in obstetric anaesthesia

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Summary

The main factors of maternal and neonatal morbidity and mortality are peripartum haemorrhage, hypertensive pregnancy diseases and maternal sepsis as well as critical situations under labour and endocrine emergencies. Peripartum haemorrhage is the main reason of pregnancy-induced mortality; early detection of the life-threatening situation and interdisciplinary cooperation are imperative. During hypertensive pregnancy diseases, prolongation of pregnancy and prevention of maternal complications have priority; the anaesthetist is mainly challenged during delivery. Risk factors for maternal sepsis are divided in obstetrical and patient-related reasons; therapy is conducted in accordance with general guidelines. Stalled labour and umbilical cord prolapse are common indications for caesarean delivery. Amniotic fluid embolism is treated symptomatically. Endocrine emergencies like hyperthyroid and diabetic disorders are of rare occurrence.

Introduction

In emergency situations in obstetric anaesthesia, the anaesthetist and obstetrician must focus their attention on the welfare of both the mother and the unborn child, whereby the well-being of the child requires that the vital functions of the mother are consistently stable.

The main causes of maternal and neonatal morbidity and mortality are peripartum haemorrhage, hypertensive pregnancy diseases and sepsis [1,2], in addition, critical situations during delivery and endocrinological emergencies during pregnancy.

Peripartum haemorrhage

Epidemiology and general aspects

Peripartum haemorrhage (PPH) is the most frequent cause of pregnancy-related mortalities [1,2,3].

In Western Europe, seven women per 100,000 live births die of PPH, in Central Africa their number amounts to 1,570 [4]. In Germany, the incidence rate of PPH has increased to approx. One out of 250 deliveries, to which the increasing rate of caesarean deliveries (>30%) with consecutively increasing placenta implantation disorders and an increased risk of uterus rupture during subsequent pregnancies have made a contribution. In the Netherlands, 0.7% of all home births had to be admitted to hospital because of PPH [5].

The **blood loss** during a delivery proceeding without complications amounts to up to 500 ml; bleeding is terminated and/or compensated for by the contraction of the uterus and physiological hypervolemia. A blood loss of >500 ml within a period of 24 hours is referred to as **primary postpartum haemorrhage**;

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frequent causes are uterine atony and placental retention. Every larger bleeding after 24 hours and up to 12 weeks postpartum is considered as **secondary postpartum haemorrhage** [6,7]. In addition, the following definitions apply [6,7]:

- In German-speaking countries PPH exists if the loss of blood after either vaginal or caesarean delivery amounts to >500 ml and >1,000 ml, respectively. A serious case of PPH exists if the acute blood loss exceeds 1,500 – 2,000 ml.
- The WHO (World Health Organization) considers every blood loss greater than 500 ml as PPH irrespective of the birth modality.

Most young and healthy mothers are initially able to compensate for a greater blood loss for quite some time, before a **haemorrhagic shock** with agitation or clouding of consciousness, cold sweat, paleness, tachycardia, hypotension and hyperventilation sets in. The amount of blood captured in towels, compresses and sponges etc. is often underestimated, for which reason the use of calibrated blood collection bags or surgical drapes with blood collection pouches is recommended [7,8].

- From a blood loss of approx. 1,000 ml onward, the basic actions presented in more detail below must be immediately taken, e.g. retention of warmth, application of large-lumen indwelling venous cannulas, basic laboratory analyses, cross-matching of erythrocyte concentrates (EC) etc.
- From a blood loss of about 1,500 ml and more, regular antifibrinolysis and, if indicated, a transfusion of EC and coagulation factors will be necessary [3,7,8,9].

Next to recognising the life-threatening situation in time, interdisciplinary cooperation will be essential.

Risk factors and potential causes

A summary of the **risk factors** for PPH are shown Tab. 1. The medical case history, physical examination and sonography contribute to the diagnosis.

Tab. 2 shows the **potential causes**, their frequencies and the risk of PPH occurrences. In addition, PPH might develop as a consequence of disseminated intravascular coagulation (DIC) due to amniotic fluid embolism, septic abortion, intrauterine infection, eclampsia and HELLP syndrome (haemolysis, elevated liver enzymes, low platelet count).

Injuries of the genital tract

Injuries of the genital tract might be either unintentional (laceration of the perineum, cervical rupture) or iatrogenic (episiotomy).

- The best prophylaxis of PPH consists in rapid surgical treatment of the injury, whereby a high vaginal tear is

Tab. 1

Risk factors for peripartum haemorrhage (PPH) [1,7]. **HELLP syndrome** = haemolysis, elevated liver enzymes, low platelet count.

Factor	Prepartum	Intrapartum /Postpartum
Placenta	Placental abruption Placenta praevia Placenta accreta, percreta, increta	Placental retention
Uterus	Precedent uterine atony Precedent uterine surgery Uterus myomatosus Distension of the uterus (multiparity, polyhydramnios, transverse foetal position)	Uterine atony Uterine rupture Inversion of the uterus
Coagulation	Congenital or acquired haemostasis disorder	Consumptive coagulopathy due to pre-eclampsia /HELLP syndrome Placental abruption Amnion infection syndrome, sepsis Amniotic fluid embolism
Other	Haemorrhages prior to delivery Multipara Nicotine abuse Advanced age Non-Caucasian ethnicity PPH in the medical case history	Protracted delivery Induction of labour and prolonged administration of oxytocin Macrosomia (>4,500 g) Surgical vaginal delivery Injury of the genital tract Section, emergency section

Tab. 2

Potential causes and their frequencies as well as the odds ratio (OR) with 95% confidence interval (CI) for the occurrence of peripartum haemorrhage (PPH) [6,7,8,9,10].

Cause	Frequency	OR for PPH (95% CI)
Injury of the genital tract	1:8	1.4-4.7 (1.04-8.4)
Uterine atony	1:20	No data available
Pre-eclampsia	1:20	5 (3.0-8.5)
Multiparity	1:85	5 (3.0-6.6)
Emergency section	1:150	4 (3.28-3.95)
Premature detachment of the placenta	1:80 - 1:150	13 (7.61-12.9)
Placental retention	1:100 - 1:500	5 (3.36-7.87)
Placenta praevia	1:200	12 (7.17-23.0)
Placenta accreta	1:2,000 - 1:2,500	3.3 (1.7-6.4)
Uterine rupture	1:1,250 - 1:3,000	
Inversion of the uterus	1:6,400	

surgically very demanding and likely to be accompanied by great blood losses not to be underestimated.

Placental retention

In case of a placental retention without haemorrhage one can wait at maximum for 30 minutes for the placenta to detach spontaneously – however, it is a corroborated fact that the incidence rate of PPH can be reduced by about 60 percent through an active initiation of the placental stage of labour [11].

- Active procedures are – apart from a controlled traction on the umbilical cord (CCT) – the administration of uterotonic drugs, clamping and cutting of the umbilical cord and massage of the uterus. Recent studies indicated that only the administration of uterotonic drugs reduces the incidence rate of PPH [12].
- The expelled placenta must always be examined for completeness. Possible placental residues can be identified by ultrasound. Then, a manual or instrumental palpation done in proper time will prevent the development of a larger PPH.

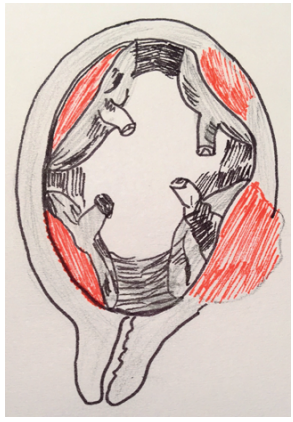
Placental implantation disorders

A placental retention might conceal a placental implantation disorder (Fig. 1).

If the placenta lacks the basal decidua, in part or fully, trophoblasts and thus placental villi may grow all the way into the uterine musculature (placenta accreta, 78%), (placenta increta, 17%), or even cross it into neighbouring organs (placenta percreta, 5%). The reason for this is cicatricial alterations of the uterus – e.g. after caesarean section, curettage and myoma removal surgery.

- The myometrium is injured when the placenta detaches and severe haemorrhages occasionally occur.
- In case of minor implantation disorders the firmly adhering placental residues can be loosened by means of a curettage, whereas hysterectomy will have to be carried out in case of placenta increta or placenta percreta.

Fig. 1



Placental implantation disorders.

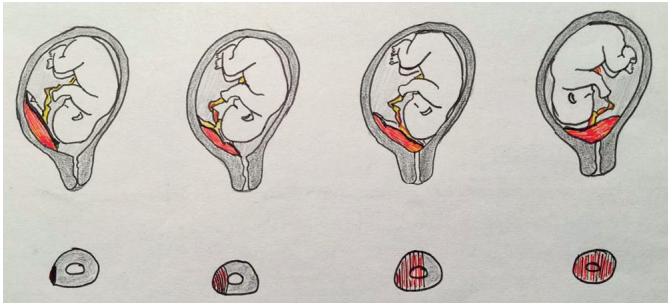
Placental malposition

A placenta praevia develops in 0.5% of all pregnancies – the placenta is either situated near the uterine cervix or obstructs the birth canal (Fig. 2).

We distinguish:

- **Low-lying placenta** – one part of the placenta lies in the lower uterine segment; vaginal delivery is possible.
- **Placenta praevia marginalis** – the placental tissue reaches the internal cervical os; in most cases vaginal delivery will still be possible.
- **Placenta praevia partialis** – the placental tissue overlies the cervical os to some extent; vaginal delivery should be avoided.

Fig. 2



Classification:				
- classic	- Typ I	- Typ II	- Typ III	- Typ IV
- modern	- low lying	- marginal	- partial	- total
- ultrasound	- minor	- minor	- major	- major

Classification of placenta praevia (placenta is represented in red) [13].

Top row: Vertical section through the uterus showing various types of placental insertion.
Bottom row: Cross-section at the level of the internal cervix.

Below the classifications. Instead of placenta praevia partialis/ totalis the terms placenta praevia incompleta / completa are not seldom used, respectively.

- **Placenta praevia totalis** – the placenta lies over the cervical os, vaginal delivery is impossible.

The cardinal symptom of placenta praevia is an **announcing vaginal haemorrhage**; it is painless, fresh and light-red in colour and occurs mostly in the middle of pregnancy. The high degree of vascularisation of the placenta and its malposition in a less contractile area explain the persistent bleeding; in addition, a placenta accreta often exists concomitantly [14]. The therapy depends on the type of the placenta praevia, the week of gestation, the intensity of bleeding and the condition of mother and child.

An emergency delivery under general anaesthesia is carried out in case of an already pronounced bleeding in order to develop the infant rapidly and achieve definitive surgical remediation.

Placental abruption

Placental abruption (abruptio placentae) is a prepartum emergency situation. As a result of the premature detachment of the placenta, the perfusion of the foetus will be disturbed or cease and the hematoma between placenta and uterus will rapidly produce a consumptive coagulopathy.

Risk factors are trauma, hypertensive diseases, premature rupture of the amnion, repeated pregnancies, smoking and uterine anomalies [15]. The hematoma might spread **undiscovered** in retroplacental direction or, as a marginal placental hematoma, be accompanied by – **now visible** – dark-red vaginal smear bleeding. Maternal mortality is nearly 1%, foetal mortality 20-60%.

- The cardinal symptom is a persistent abdominal pain in the mother, associated with hardening of the uterus; further unspecific symptoms are unrest, weakness, anxiety and nausea. In the event of a greater maternal blood loss (up to 3 litres intrauterine) signs of shock with disordered haemostasis will become noticeable. Smaller placental detachments also rapidly give rise to a DIC syndrome.
- Ultrasound will confirm the clinical diagnosis and reveal the dimensions of the hematoma. Cardiotocography (CTG) is applied to identify a condition of foetal bradycardia or already absent cardiac sounds.
- If vital signs of the foetus are detected an emergency section will be done under general anaesthesia (if indicated, accompanied by an adequate transfusion and haemostasis therapy). In the event of a lifeless foetus a vaginal delivery can be initiated provided that the mother's vital functions are stable.

Uterine rupture

A rupture of the uterus – incomplete with an intact peritoneum, or complete comprising the peritoneum – mostly occurs while delivery is in process.

In most cases it will be a **rupture of a scar** we are dealing with in this case, due to a preceding section or any other surgical operation previously carried out on the uterus [1,10,16], whereby an overstimulation of the uterus with uterotonic drugs might promote the occurrence of a uterine scar rupture. A **rupture due to distension** of tissues is possible in cases of protracted delivery, the child's presentation making delivery impossible, or macrosomia. The high section rate is of significance as a cause for the increasing incidence of uterine ruptures.

- Cardinal symptoms of an impending rupture are highly severe abdominal pain (uterine tetany) associated with foetal bradycardia as well as maternal anxiety and unrest. The pain suddenly subsides after both rupture and labour contractions stop; a condition of foetal hypoxia ensues. Symptoms of shock develop rapidly.
- An emergency section under general anaesthesia with adequate transfusion and haemostasis therapy has to proceed without delay.

Epidural analgesia to mitigate the pain of labour might not fully conceal the pain of an impending uterine rupture [17] – sustained labour pain under correctly applied epidural analgesia is therefore to be taken as an indication of an impending rupture of the uterus.

Uterine atony

Basics

Uterine atony is a condition of uterine contraction weakness, from which a severe to life-threating haemorrhage might result. The incidence has increased due to the growing number of caesarean sections in recent years [9].

Uterine atony internationally is the most common cause of PPH, whereby maternal mortality in developed countries with established atony prophylaxis is 200 times lower than in African countries [4]. A primary uterine inertia, a too high concentration of volatile anaesthetics – a MAC value (minimum alveolar concentration) above 0.8 – 0.9 reduces the action of oxytocin on the uterus – and other factors come into question as causes for uterine atony (cf. Tab. 1 and Tab. 2). Uterine atony occurs more often in cases of multigravidity, polyhydramnios, multipara, prolonged labour or chorionamnionitis.

The cause of an atonic uterine haemorrhage must be established and treated specifically, otherwise the PPH will not stop even after a massive transfusion.

Prevention of PPH due to uterine atony

The decisive prophylactic intervention is a slow IV administration or short infusion of 3 - 5 I. U. (international units) oxytocin (Syntocinon®) after delivery of the front shoulder or delivery of the child [7].

- A prophylactic administration of oxytocin decreases the risk of PPH by 50 percent and reduces the necessity of a therapeutic application of uterotonic drugs by about 50 percent as well [7,18].
- The synthetic oxytocin analogue carbetocin (Pabal®) acts longer than oxytocin and is currently approved exclusively for a single application (100 µg slow IV.), in cases of caesarean delivery carried out in spinal or epidural anaesthesia after development of the child [7]. Carbetocin should not be concomitantly administered with oxytocin, nor in cases of migraine, bronchial asthma, cardiovascular disease (including pre-eclampsia and eclampsia) or epilepsy.
- Prophylactic techniques to therapy an atony-dependent PHH without drugs includes uterus massage (with endogenous prostaglandin release) and bimanual uterus compression (Hamilton's manoeuvre).

Therapy of PPH due to uterine atony

If prophylactic measures fail, the therapeutic measures mentioned below should be applied immediately (Tab. 3) They are described in the pertinent Guideline of the German Society of Gynaecology and Obstetrics (DGGG) which refers to further guidelines with haemostaseological options [7].

- If not done prophylactically, 3-5 I. U. oxytocin are injected slowly IV (maximum IV bolus dose 6 I. U.); followed by an infusion dosed according to action consisting of 10-40 I. U. oxytocin dissolved in 500 ml balanced electrolyte solution (caution: side effects).
- In addition, a slow IV administration of up to 0.1 mg methylergometrine (Methergin®) might be taken into consideration; however, the substance is only officially approved for cases of very severe postpartum haemorrhages.

Typical side effects of oxytocin and methylergometrine are blood pressure rises/drops, tachycardia and further cardiac arrhythmias, flushes, headache and chest pain, nausea, vomiting, but also coronary spasms and myocardial infarction. Oxytocin should therefore be administered in doses as low as possible and IV application should be carried out slowly [8,19,20,21]. Methylergometrine is contraindicated in cases of high blood pressure, pre-eclampsia/eclampsia, ischemic vascular diseases, severe disorders of liver and kidney function and sepsis.

An oxytocin-resistant haemorrhage should be treated without much delay with prostaglandin derivatives, whereby oxytocin and prostaglandins should not be administered simultaneously [7].

- Sulprostone (Nalador®; 500 µg in 500 ml solution) is applied by an infusion pump. The initial dose amounts to 100 ml/h (if necessary: 500 ml/h at maximum), the maintenance dose is 100 ml/h, the maximum dose 1,000 µg in 10 hours, the daily maximum dose 1,500 µg. Side effects – e.g. epigastric and mesogastric spasms, bronchoconstriction, myocardial ischemia and blood circulation reactions all the way to pulmonary oedemas – must be relativised in a life-threatening situation. An injection into the myometrium is contraindicated.
- When initially administered, misoprostol is not more effective than oxytocin and was taken from the market in Germany; prostaglandin F2α (e.g. Dinoprost®) is no longer approved in Germany for the treatment of uterine atony or PPH.

Measures in case of sustained bleeding

Parallel to these drug-related measures, it is the responsibility of the obstetrician to exclude the typical causes of sustained bleeding such as placental residues (ultrasound) and injuries due to delivery (by speculum adjustment); in addition, the bladder should be emptied. The identification of the cause of bleeding (cf. Tab. 2) shall proceed in conformity with the DGGG guideline and the "PPH Consensus Group (Germany-

Tab. 3
Prophylactic and therapeutic measures in cases of peripartum haemorrhage [7,22].

Prophylaxis
• Slow administration IV or short infusion of 3-5 I. U. oxytocin
Drug therapy – escalating, parallel to surgical therapy
• Slow administration IV or short infusion of 3-5 I. U. oxytocin (if not already done) • Infusion von 10-40 I. U. oxytocin (in 500-1.000 ml balanced electrolyte solution) • Consider administration of 0.1 mg methylergometrine IV • Infusion of sulprostone (500 µg in 500 ml solution), initial dose 100 ml/h (up to 500 ml/h) • 1-2 g and/or 15-30 mg/kg b.w. tranexamic acid • 20-30 ml/kg b.w. frozen plasma; alternative or in addition to 30-60 mg/kg b.w. fibrinogen • Consider PPSB (initial dose 25 I. U./kg b.w. IV.) and factor XIII (15-20 I. U./kg b.w. IV.) • Heparin or antithrombin only after cessation of bleeding • In substantiated individual cases administration of desmopressin and recombinant factor VIIa
Surgical therapy – escalating, parallel to drug therapy
• Identification of the cause of bleeding according to the 4 T's • First uterine tamponade • Surgical or radiological-interventional haemostasis • If necessary, hysterectomy
Accompanying measures – parallel and early on
• Emptying the bladder • Application of efficient venous accesses • Blood sampling for cross-matching and emergency laboratory, information of blood bank /depository • Volume therapy, vasoactive substances • (Invasive) monitoring of blood circulation • Sequential laboratory analyses (differential blood-cell count, coagulation, blood gas analysis) • Seeking of the best-possible personal expertise • In case of regional anaesthesia or status post vaginal delivery, if indicated, endotracheal intubation to secure airways and oxygenation • Generous indication for a central venous catheter

I. U. = International units;
b.w. = body weight.

Austria-Switzerland)" according to the four T-s [7,22]:

- **Tonus and/or uterine atony** – uterine distension (multiparity, hydramnios, foetal macrosomia), tocolytic agents, rapid or delayed delivery, (long) oxytocin replacement, chorioamnionitis, uterine myomas.
- **Tissue and or placenta** – placental retention, placental implantation disorder, placental residues.
- **Trauma** – vulvovaginal injury, cervical rupture, episiotomy/laceration of the perineum, uterine rupture, uterine inversion.
- **Thrombin and/or coagulopathy** – pregnancy-induced (thrombocytopenia in case of HELLP syndrome, DIC in case of pre-eclampsia, intrauterine foetal death, placental abruption, or amniotic fluid embolism) or other disorder of haemostasis.

The guideline recommends consulting an anaesthetist in case of a PPH **at an early stage** [7]. An initial notification by the obstetrician should proceed at maximum 30 minutes after PPH has been diagnosed, after a maximum of further 30 minutes and sustained bleeding an anaesthetist should be consulted for further therapy and the surgical team alarmed [22].

The anaesthetist faces a professional challenge already at this stage and is responsible especially for establishing efficient venous accesses, (invasive) monitoring of blood circulation, volume therapy, sequential laboratory analyses (differential blood cell counts, coagulation, blood-gas analysis) as well as the provision and, if indicated, transfusion of blood components and coagulation factors [7,22].

Particularly in case of an unexpected occurrence of an emergency situation during a hitherto "uncomplicated" delivery the anaesthetist will have to convince himself of an existing blood group determination, otherwise an immediate blood sampling for cross-matching and emergency laboratory, information of the blood bank and cross-matching of erythrocyte concentrates and perhaps the provision of fresh frozen plasma (FFP) must proceed now.

As is the case in every serious bleeding, the anaesthetist must also in case of a PPH undertake all efforts to maintain and/or restore the following common target values [7,8,22]:

- body core temperature >34 °C (normothermia, if possible),
- pH value >7.2,
- ionised calcium (Ca^{++}) concentration >0.9 mmol/l (normocalcaemia, if necessary).

In case of a therapy-refractory bleeding, the obstetrician will now insert an initial tamponade into the cavity of the uterus in order to enable a permanent or temporary ("bridging") haemostasis with hemodynamic stabilisation and initiate further surgical and interventional-radiological measures [7,22]. Tamponade strips or balloon systems are used to achieve this end; in addition, also local haemostatic agents such as chitosan. Now, at the latest, the best-possible personal expertise must be acquired. Actual surgical haemostasis comprises laparotomy with eventeration of the uterus, traction in cranial direction and compression, in addition, clamping of the uterine arteries and applying compression sutures (e.g. B-Lynch sutures) with tamponade. Uterine compression sutures can be applied quickly and with a positive success rate (>90%) [7]. If the infrastructure is available an interventional-radiological embolisation of the uterine arteries may proceed.

Uterus-retaining measures are only reasonable as long as the patient remains hemodynamically stable and does not have a life-threating haemorrhage – a perhaps necessary hysterectomy should not be indicated too late [6,7].

Relative contraindications for uterus-maintaining measures are placental disorders (placenta increta, placenta percreta), an irreparable uterine injury (uterine rupture, intraabdominal haemorrhage in case of a caesarean section) and a septic uterus. An emergency hysterectomy must proceed in case of an uncontrollable PPH. After surgery, the patient must be monitored at an intensive care medical ward, for example, because of a relatively high relaparotomy rate.

The escalating indicated surgical measures must be flanked by a simultaneous and weighed-escalating therapy of the haemostasis disorder either now prevailing or yet to develop. In this context, tranexamic acid and fibrinogen have an important part to play in cases of PPH, for which reason both substances should be applied at an early stage [6,7,8,22]:

- **Tranexamic acid:** As a **hyperfibrinolysis** regularly exists in cases of PPH due to the massive release of plasminogen activators from the uterus, an IV administration of 1-2 g or 15-30 mg tranexamic acid /kg body weight (b.w.) (Cyklokapron®) is indicated and may also be repeated [7]. Alternatively, a short infusion of 1 g tranexamic acid is recommended after blood losses of approx. 1,000 ml [8,22]. Tranexamic acid reduces the postpartum blood loss (when administered in addition to uterotonic drugs) and prevents PPH as well as blood transfusions, both in cases of vaginal delivery and caesarean section [23]. The data currently available do not suffice to draw exact conclusions on the side-effect risk concerning thromboembolic incidents.
- Only after this is it recommendable to substitute coagulation factors in case of a persistently prevailing severe bleeding tendency [7]. To this end, the administration of **fresh frozen plasma** (FFP) at a dose of 20-30 ml/kg b.w. is recommended, either alternatively or in addition, the administration of **fibrinogen** (Haemocomplettan®) at a dose of 30-60 mg/kg b.w. IV (2-4-8 g) [7].
- **Fibrinogen:** In cases of PPH, the fibrinogen concentration in plasma will correlate best with the overall quantity of lost blood [24]. That a fibrinogen concentration of <200

mg/dl (<2 g/l) at the end of pregnancy is correlated with the occurrence of PPH has a likelihood of almost 100%. The determination of fibrinogen might therefore help to identify patients who are at risk of contracting PPH [7]. The target value for substitution is a concentration of ≥200 mg/dl [7]. 3 g fibrinogen elevate the plasma concentration by approx. 100 mg/dl.

- Furthermore, PPSB might be given intravenously in an initial dose of 1,000 – 2,500 I. U. (25 I.U./kg b.w.); under certain circumstances also factor XIII (Fibrogammin®P) at a dose of 1,250 I. U. (15-20 I.U./kg b.w.), whereby factor XIII is intended to stabilise soluble fibrin monomers.

In addition, the following aspects must be observed [7]:

- In order to avoid an enhancement of bleeding, **heparin** should not be administered when the haemorrhage is active.
- Neither should **antithrombin** (AT) be given in the course of active bleeding. After the bleeding has been stopped – especially after the administration of single factors or complex drugs (PPSB)-the activity of AT may be determined at the ICU and a target value of ≥ 80% is aspired.
- In case of patients under medication with platelet aggregation inhibitors, or patients with suspected thrombocytopathy, it may be endeavoured to improve platelet function by an enhanced release of factor VIII and von-Willebrand factor. Desmopressin (Minirin®) is applied IV to this end, at a dose of 0.3 µg/kg b.w. over a period of 30 minutes.
- In the individual case and if all other therapy options and optimisation of common factors fail (sufficient plasma coagulation potential, sufficient platelet counts and haemoglobin (Hb) concentration, lacking acidosis, normothermia and normocalcaemia), it may be endeavoured to stop a diffuse bleeding with an administration of **recombinant factor VIIa** (NovoSeven®) at an initial dose of 90 µg/kg b.w.

The following applies to therapy with blood components in cases of massive bleeding [7]:

- Erythrocyte concentrates with a target value of Hb 7-9 g/dl (4.3-5.6 mmol/l) are transfused in order to substitute for oxygen carriers. In case of a massive transfusion, it is recommeded to warm up the erythrocyte concentrates (using either a heating appliance or inline heating).
- Patients who require massive transfusions (if predictable) or who are suffering from a life-threatening shock might benefit from a high FFP: EC ratio (≥ 1:2) and from the combined administration of FFP and factor concentrates.
- Platelet concentrates (PC) with a platelet count target value of ≥ 100,000/µl are transfused in order to compensate for losses incurred during primary haemostasis.
- In cases of emergency erythrocyte concentrates derived from a 0 rhesus-negative blood groups must be applied without cross-matching as well as FFP derived from the blood group AB and/or coagulation factors, without waiting for the laboratory values. Holding a contingency depot in reserve is obligatory in obstetrics.
- In addition, an active heat supply as well as the application of a cell centrifuge (Cell-Saver) and rapid-transfusion systems are recommended [6,7,8,9].

Actions must be taken rapidly, with determination and interdisciplinary consensus. Monitoring of the patient should be extended at an early stage by arterial pressure measurement and a central venous catheter (CVC) and the ICU should be notified about the patient's transfer.

After PPH diagnosis, an adjusted monitoring for 24 hours and an interdisciplinary discussion of the case should take place. A pharmacological thrombosis prophylaxis will be obligatory, at the latest, 24 hours after the haemorrhage. A sufficient iron substitution is recommended [7].

Hypertensive pregnancy diseases

Incidence and definitions

Data pertaining to the **incidence** of pre-eclampsia vary between 3% and 10%; pregnancy-induced hypertension and pre-eclampsia are believed to be responsible for approx. 25% of perinatal morbidity and perinatal mortality [25, 26]. In combination with a peripartum lung oedema, the lethality of pre-eclampsia ranges at about 5% [27]. Risk actors are high age, adiposity, diabetes mellitus, pre-existing hypertension, pre-eclampsia in the medical case history of the patient and her family as well as kidney diseases and the existence of an antiphospholipid syndrome [28].

The following **definitions** apply according to the guideline of the DGGG [29]:

- **Gestational hypertension** exists whenever blood-pressure values of ≥140/90 mmHg without proteinuria appear in previously normotensive pregnant women after completion of the 20th week of gestation.
- In cases of **pre-eclampsia** (or EPH gestosis) proteinuria ≥300 mg/24 h appears; often resulting in foetal growth retardation.
- A **severe pre-eclampsia** exists whenever at least one of the following criteria are fulfilled in addition: blood pressure ≥ 160/110 mmHg, impaired kidney function (creatinine ≥79.6 µmoles/l or 0.9 mg/dl, or oliguria <500 ml/24 h), liver involvement (elevation of transaminase activities, persisting epigastric pain), pulmonary oedema, hematologic disorders (thrombocytopenia <100,000/µl, haemolysis), neurological symptoms (strong headaches, visual disorders), foetal growth restriction (estimated foetal weight <5th percentile and/or pathological Doppler of the umbilical artery).
- Tonic-clonic seizures that appear in the context of a pre-eclampsia not attributable to any other cause are referred to as **eclampsia**. Eclampsia is not always associated with severe hypertension and might also manifest itself as late as postpartum.

- A **HELLP syndrome** exists in case of the following triad: haemolysis, elevated liver enzymes and low platelet counts <100.000/µl. In 5-20% of HELLP syndrome cases there is neither proteinuria nor hypertension [30].

Pathophysiology

The aetiology of hypertensive pregnancy diseases is not fully known. As for pre-eclampsia, a disordered trophoblast invasion with consecutive placental insufficiency and generalised endothelial dysfunction are discussed as pathogenic events.

Furthermore, an imbalance of angiogenic factors (PIGF = placental growth factor) and antiangiogenic factors (sFlt-1-Protein; soluble fms-like tyrosinekinase-1) has been shown [30].

Consequences of **generalised vasoconstriction** and **endothelial dysfunction** are:

- Chronic placental insufficiency and foetal growth retardation, increased risk of a premature detachment of the placenta;
- Damage of the renal glomeruli associated with proteinuria and oedema tendency;
- Cerebral oedemas and intracranial microhaemorrhages associated with an increased risk of disordered vision, nausea, vomiting, headaches and generalised tonic-clonic seizures;
- Periportal haemorrhagic necrosis and capsule swelling of the liver with epigastric pain and elevation of transaminase activity;
- Thrombocytopenia and anaemia due to platelet activation and aggregation and microangiopathic haemolysis.

Diagnosis and therapy

The **diagnosis** already ensues from the stated definitions; in addition, a blood test which determines the quotient of sFlt-1/PIGF is capable of distinguishing healthy pregnant women from those having pre-eclampsia [31]. In patients with manifest pre-eclampsia, the test permits drawing conclusions on the severity, dynamics and short-term prognosis of the disease [32].

The primary objective of therapy between the 24th and 34th week of gestation consists in the prolongation of pregnancy in order to prevent premature delivery and to induce foetal pulmonary maturity. All other measures focus on the prevention of maternal complications.

The only **causal therapy** is delivery [29]:

- In cases of **pre-eclampsia** delivery is regularly indicated after the 37th week of gestation.
- In cases of **severe eclampsia** the patient should soon deliver starting with the completed 34th week of gestation, the same applies in case of a severe foetal growth restriction.
- A primarily conservative procedure is to be recommended if pre-eclampsia either starts in the completed 24th to 34th week of gestation or appears in connection with the HELLP syndrome. Maternal indications to terminate pregnancy in this interval are therapy-refractory severe hypertension, therapy-refractory kidney insufficiency, cardiac decompensation, acute pulmonary oedema, DIC, persistent epigastric pain, new occurrence of serious central nervous symptoms and eclampsia.

The following recommendations apply to **drug therapy**:

- Antihypertensive drug therapy (Tab. 4) should be initiated at the latest if blood pressure values are at ≥160/110 mmHg. It should proceed in the hospital setting under close blood-pressure monitoring of the mother and – if the infant is able to survive – under CTG monitoring. It serves the purpose of preventing cerebrovascular and cardiovascular complications (especially cerebral haemorrhages), whereas an additional administration of magnesium IV is required to establish an effective eclampsia prophylaxis. As a too strong blood-pressure decrease increases the rate of growth-retarded newborns, a systolic pressure of <150 mmHg and a diastolic pressure of 80-100 mmHg are considered to be recommendable target values [29,33].
- α-methyldopa (Dopegyt®) is the drug of first choice for long-term oral therapy. Suitable to a limited extent are nifedipine retard as well as selective β_1-receptor blockers (preferentially metoprolol). Not appropriate because of their pronounced foetal side effects are diuretics, angiotensin-converting-enzyme (ACE) inhibitors, angiotensin-II-receptor-subtype-1 (AT_1)-antagonists and all other antihypertensive agents [29].
- Recommended for acute therapy in case of an hypertensive emergency are nifedipine (e.g. Adalat®), urapidil (e.g. Ebrantil®) and dihydralazine (Nepresol®). Furosemide (e.g. Lasix®) is injected in case of a pulmonary oedema [29].

Anticonvulsive therapy proceeds with magnesium sulphate.

- Initially, 4 g (up to 6 g) **magnesium sulphate** are administered as a short infusion over 15 minutes, followed by a maintenance infusion of 1 g/h to 48 hours postpartum [28,29,30]. The targeted level is 2-4 mmol/l; in addition, respiration rate (respiratory depression at 5 mmol/l), patellar tendon reflex (disappears at 4 mmol/l) and diuresis (at least 100 ml/4 h) should be monitored. Calcium gluconate is available as an antidote.
- A cerebral convulsive seizure must be broken with diazepam (10-20 mg IV, if indicated, repeatedly) or midazolam (5-10 mg IV, if indicated, repeatedly). Thiopental or phenytoin are second-choice drugs.

Glucocorticoids are increasingly applied in order to prolong pregnancy in case of a **HELLP syndrome** (e.g. daily 2-3x 10 mg dexamethasone IV). Apart from an induction of foetal pulmonary maturity, positive maternal effects have also been demonstrated (increase in platelet counts), so that ultimately up to 50 percent of the mothers could receive regional anaesthesia for delivery [34]. However, a decline of maternal and foetal morbidity and mortality has not been demonstrated, wherefore this

Tab. 4
Antihypertensive drugs in cases of pre-eclampsia [29].

Active substance / product	Dosage	Comment
Long-term oral therapy		
α-Methyldopa / e.g. Dopegyt®	4x500 mg/d	First-choice therapy
Nifedipine retarded / generics	2-6x 10 mg/d, max. 120 mg/d	No teratogenic effects determined
Metoprolol / generics	2x 25-100 mg/d	Elevated risk of foetal growth restriction
Acute therapy		
Nifedipine / e.g. Adalat®	Initial oral dose of 5 mg (if required, repeatedly); syringe pump (5 mg/50 ml) after effect, at maximum 15-30 mg/24 h	Off-label-use
Urapidil / e.g. Ebrantil®	Initial 6.25 mg IV (if required, repeatedly); syringe pump 3-24 mg/h	Off-label-use
Dihydralazine / Nepresol®	Initially 5mg IV; syringe pump 2-20 mg/h	Multiple side effects, second-choice therapy
Furosemide / e.g. Lasix®	10-20 mg IV	In case of pulmonary oedema

option is discussed controversially [29, 35]. Therapy of thrombocytopenia and coagulopathy follows the principles described for PPH.

Anaesthesiological treatment

Spinal or epidural anaesthesia is the method of choice in case of a caesarean delivery and patients with pre-eclampsia, because there is a risk of cerebral oedemas, intracranial haemorrhages, acute left-heart insufficiency and pulmonary oedema due to blood-pressure rises during laryngoscopy as well as intubation and extubation whenever general anaesthesia is applied [36,37].

- In case of vaginal delivery, epidural analgesia applied in proper time contributes to a moderate blood-pressure decrease and an improvement of uteroplacental perfusion (decrease of pain-dependent maternal catecholamine levels, increase of the intervillous blood flow and decrease of peripheral vascular resistance).
- The low dose of acetylsalicylic acid (ASA; 100 mg/d) often applied to patients with pre-eclampsia is not a contraindication for an intervention near the spinal cord.
- In female patients with HELLP syndrome, spinal or epidural anaesthesia have been carried out after the administration of glucocorticoids [34] – however, in cases of HELLP syndrome it must be considered that the platelets are not only reduced in number, they are also functionally impaired, for which reason general anaesthesia is preferred in the hospitals of the authors.
- In cases of eclampsia and after initial therapy, an emergency caesarean section under general anaesthesia will mostly be inevitable. In order to prevent blood-pressure peaks caused by intubation an opioid is applied in this case before administration of the hypnotic agent and before severing the umbilical cord of the foetus. When applying remifentanil (1 µg/kg b.w. IV) as described in the literature [38, 39], respiratory depression and chest rigidity in both mother and child must be taken into account – such procedure must therefore be communicated to the neonatologist.
- A pre-existing vasoconstriction with relative hypovolemia affecting the patients makes anaesthesia more difficult, wherefore the indication for invasive arterial pressure measurement and CVC should be generous in cases of severe pre-eclampsia. Vasopressor agents must be carefully titrated as overshooting reactions are often induced.
- Pregnant women with pre-eclampsia have a high risk of contracting a peripartum lung oedema [40]. Main reasons are the disease-related high hydrostatic and low colloid-osmotic pressure (COP), in addition to the often applied tocolysis and a perhaps iatrogenic volume loading. Cerebral convulsive seizure and pulmonary oedema can manifest themselves even hours or days after delivery, for which reason patients with eclampsia and HELLP syndrome should be first monitored in the ICU and subsequently closely monitored on peripheral ward level.

Sepsis during pregnancy and lactation

Incidence

Infections appearing during pregnancy might result in premature delivery, premature rupture of membranes, premature uterine contractions and in breastfeeding difficulties. According to cohort studies conducted in the United States, the incidence rate of **sepsis** is at 3-10: 10,000 births [41,42]; the incidence rate of a sepsis-dependent case of death at 1:100.000 births [2,42]. On an international scale, sepsis is responsible for 15 percent of all maternal deaths.

Risk factors

The risk factors for developing maternal sepsis can be divided to obstetric-related and patient-related factors:

- **Obstetric-related factors** are cerclage, caesarean section, premature rupture of the membranes, placental retention after delivery or abortion, vaginal trauma, wound infection and amniocentesis.
- **Patient-related factors** are high age, group B streptococcal infection in the medical case history, group A streptococcal infection in the family, perturbed vaginal flora, immunosuppressive medication, cardiac insufficiency, chronic hypertension, chronic liver and kidney disease,

infection with human immunodeficiency virus (HIV), malaria, sickle-cell anaemia, systemic lupus erythematosus as well as Afro-American or Asian ethnicity and low social status [41,42].

- In case of PPH, the odds ratio (OR) for the development of sepsis is at 4.2, with a 95% CI of 2.5-7.1; whereas in case of pre-eclampsia, the OR is at 3.7, with a 95% CI of 2.5-4.4 [41].

The most common causes of sepsis during pregnancy and lactation are infections of the genital tract (chorioamnionitis, endometritis, septic abortion), pneumonia, pyelonephritis, peritonitis, mastitis and wound infections. An antibiotic prophylaxis is therefore recommended in cases of caesarean delivery, premature rupture of membranes and amniotic fluid containing meconium [43,44,45]. In case of caesarean delivery, antibiotic prophylaxis significantly reduces the rate of wound infections, endometritis and serious maternal infections, irrespective of the type of section (elective, non-elective) and administration before or after clamping the umbilical cord [43,46].

Spectrum of pathogens

Numerous pathogens are potential candidates for casing sepsis [47,48]. In Great Britain, β-haemolysing streptococci belonging to Lancefield group A were responsible for almost one-half of all sepsis-related maternal deaths in the years from 2006 to 2008 [49]. Next to staphylococci they are the major cause of puerperal sepsis ("childbed fever") and mostly transmitted by droplet infection by asymptomatic carriers (children) or persons with acute upper respiratory tract infections [50]. β-haemolysing streptococci belonging to Lancefield group B are representatives of the vaginal flora and can cause neonatal infections and sepsis by means of "vertical" transmission. Prophylaxis with antibiotics fails to reduce the incidence of puerperal sepsis in cases of colonisation with group B streptococci; the evidence regarding a reduction of neonatal infections is unclear [51]. In case of wound infections, peritonitis and endometritis, mixed infections with Staphylococcus aureus, Enterobacteriaceae and anaerobic bacteria often prevail. E. coli is dominant in cases of pyelonephritis, whereas in case of pneumonias pneumococci, Haemophilus spp., staphylococci, chlamydia, mycoplasmas and varicella must be taken into account [42,48,49]. Pregnant women are considered to represent a risk population during outbreaks of influenza-A virus H1N1, whereas HIV is the main factor for sepsis-dependent maternal mortality in developing countries [47].

Therapy

Maternal sepsis is categorically treated according to the applicable Guideline on the Prevention, Diagnosis, Therapy and Aftercare of Sepsis [52].

- Consequently, the same target values are applicable to initial **hemodynamic stabilisation** as apply to other patients (mean arterial pressure ≥65 mmHg), whereby the risk of a peripartum pulmonary oedema must be observed.
- **Microbiological diagnostics** requires a blood culture, urinalysis and a vaginal swab [50].
- As only scarce data are available on the side effects of an **antibiotic therapy** on mother and child, absolute contraindications in cases of severe maternal sepsis after childbirth do not exist. The immediately necessary, initially still calculated antibiotic therapy starts with broad-spectrum antibiotics IV [47,49,52]. As the penicillins and cephalosporins recommended for common bacterial infections during pregnancy and lactation (Tab. 5) [53] – and/or ma-

Tab. 5
Common antibiotic therapy during pregnancy and lactation; contraindications and limitations must be overruled in case of sepsis [53,54]

Antibiotic	Comment
Recommended	
Penicillins, amoxicillin	Ampicillin/sulbactam is safe
Cephalosporins	Ceftriaxone assumes bactericidal concentrations in the amniotic fluid; antibiotic of choice during lactation
Macrolides	in case of penicillin hypersensitivity and infection with Chlamydia
Contraindicated	
Tetracyclines	14th week of gestation until 7th year of life
Quinolones	Cartilage damage
Rifampicin	Bleeding tendency in the newborn due to vitamin K antagonism, but drug of choice in cases of tuberculosis applicable during pregnancy and lactation
Allowed with restrictions	
Aminoglycosides	30% cross the placental barrier, ototoxicity and nephrotoxicity; applicable during lactation
Metronidazole	Experimentally cancerogenous and mutagenic; applicable in cases of infection with anaerobic bacteria during pregnancy and lactation
Clindamycin	Diarrhoeas in neonates , more effective than penicillin against group A streptococci
Cotrimoxazole	Neural tube defect possible, passes into breast milk only to a limited extent
Piperacillin/tazobactam	No data available, applicable in cases of sepsis
Carbapenemes	Crosses the placental barrier and passes into breast milk, applicable in cases of sepsis
Linezolid	Reproduction toxicity possible, applicable in cases of MRSA sepsis
Vancomycin	Second choice during pregnancy and lactation

MRSA = methicillin-resistant Staphylococcus aureus.

crolides in case of an allergy against β-lactam antibiotics or infection with chlamydia – have a too narrow action spectrum in cases of maternal sepsis, piperacillin-tazobactam and carbapenems are recommended [47, 49], or an initial triple combination of β-lactam, aminoglycoside and metronidazole is used because of the mixed infection often prevailing [48]. Antibiosis should be tested on a daily basis and continued for the duration of at least 7 to 10 days [47, 49].

- As is the case in every sepsis, the rapid detection and, if indicated, surgical **elimination of the focus of infection** is decisive. This might span from the curettage of a residual placenta over exploratory laparotomy to hysterectomy. A septic uterus must be extirpated, otherwise it is likely to trigger an uncontrollable PPH. Hysterectomy in an instable patient is fraught with risk, however, it might also be the only chance for maternal survival [49].

In addition, it should be mentioned that for the macrolides and β-lactams, which are generally regarded as safe, an increased neonatal mortality (relative risk 1.5; 95%-CI 1.05-2.15) has been discussed when applied in cases of impending premature delivery without breaking of waters and without signs of maternal inflammation, despite a significantly reduced maternal infection rate; whereas there had been an increased risk for infantile cerebral paresis (relative risk 2.8; 95%-CI 1.02-7.9) in cases of simultaneous applications [55].

Other emergency situations

General considerations

In some obstetric emergencies the anaesthetist can only contribute to solving the problem by means of adequate epidural analgesia (subsequent injections always only upon request and/or after conferring with the midwife or obstetrician) or by immediate realisation of an emergency or urgent section.

Obstructed labour

Reasons for obstructed labour might be uterine inertia, positions obstructing delivery (transverse position, oblique position, mentoposterior face position), presentation abnormalities (high foetal station, occiput posterior presentation), or a shoulder dystocia.

- Obstructed labour occurring in the **dilation stage** is usually terminated by caesarean section.
- In the **expulsion stage** vaginal delivery is attempted by applying specific measures. In case of bradytocia, this might consist in an administration of oxytocin or vaginal-surgical delivery; in case of abnormal presentations, episiotomy and special manoeuvres are carried out by the obstetrician.
- **Shoulder dystocia** occurs in about 0.5 percent of all births and merges rapidly into state of infantile hypoxia. Risk factors are foetal macrosomia, diabetes mellitus and maternal adiposity. In cases of suspected macrosomia (estimated weight >4,500 g) and preceding shoulder dystocia the indication for primary section should be generous. In case of a manifest shoulder dystocia the obstetrician will make an attempt to solve the critical situation by episiotomy, McRoberts manoeuvre, acute tocolysis and other specific manoeuvres (e.g. after Woods).

Umbilical cord prolapse

An umbilical cord prolapse occurs in approx. 0.3 percent of all births. After rupture of the amnion the umbilical cord falls in front of the presenting part of the foetus, the foetus becoming hypoxic due to an ensuing compression of the umbilical cord.

- Initial actions taken are elevation of the mother's pelvis and pushing foetus and umbilical cord back into the uterine cavity – afterwards an emergency section must be performed.

Peripartum pulmonary oedema

The peripartum pulmonary oedema (PPE) has an incidence rate of 0.05-0.2% and belongs to the rare obstetric emergencies. The most relevant predisposing alterations [56] during pregnancy are haematocrit decline, enhanced cardiac output and a COP decrease. A COP of 13-16 mmHg [57] is considered to be the threshold limit value for the development of a pulmonary oedema; the most critical decrease in COP [58,59] occurs up to 48 hours postpartum (from 22 to 15 mmHg) and in pre-eclampsia patients (from 18 to 14 mmHg). Other risk factors are the application of tocolytic drugs, the existence of maternal infections, pre-eclampsia or a HELLP syndrome as well as an excessive volume supply under tocolysis, and cases including general or regional anaesthesia for caesarean delivery [56].

- Therapy will depend on the respective causes and will be flanked by liquid balancing, forced diuresis and, if indicated, ICU circulation therapy and (non)invasive ventilation.

Paracolpium tear, paracolpium hematoma

A tear of the paracolpium or a hematoma after spontaneous or vaginal-surgical delivery is of rare occurrence. The cause is a venous, externally invisible haemorrhage from the paracolpial plexus with blood flowing into the parametrium. The patients are distinguished postpartum by a uterine fundus persisting over the umbilicus and lacking recovery. The cardinal symptoms are pain, urinary retention and a pressure sensation on the colon. The diagnosis proceeds by means of a vaginal examination in which the hematoma is palpated as a bulging tumour. After reaching a certain size, or if diagnosed at a later event, a haemorrhagic shock with cloudy consciousness or mental agitation, pale skin, cold sweat as well as hypotension and tachycardia is likely to develop.

- Therapy consists in the surgical treatment by a vaginal approach for making an incision into the hematoma, suturing and, if indicated, drainage. Spreading of the hematoma in cranial direction into the retroperitoneal space with the necessity of laparotomy is feared.

Ogilvie's syndrome and colon perforation

In case of an **Ogilvie's syndrome** – an acute pseudo-obstruction of the colon – there is a massive dilation of the colon without any mechanical obstruction; the incidence rate amounts to 1:1,500 births.

- Therapy is primarily conservative (neostigmine, erythromycin, endoscopic suction); only one-fourth of the patients require laparoscopic intervention [60].

A perforation of the colon after caesarean section has been described to occur in individual cases [61]; causes were paralytic ileus or an Ogilvie's syndrome with ischemia after section. As the mortality rate amounts to 30-50%, this serious complication must be borne in mind early on after an "obstetric routine intervention". Indications are increasing abdominal pain (despite analgesia), defensive tension and lacking peristalsis and/or discharge of gas. A perforation of the colon might rapidly result in a peritonitis and sepsis; most frequently the caecum will be affected.

- Therapy consists in laparotomy with oversewing or caecostomy all the way to hemicolectomy.

Amniotic fluid embolism

A case of amniotic fluid embolism – or anaphylactic pregnancy syndrome – occurs in about 1:50,000 births [56,62,63]. Maternal mortality ranges at 10-40% [63].

Amniotic fluid embolism (AFE) appears particularly during caesarean delivery, in case of a high cervical tear, placenta praevia, or placental abruption. Risk factors include high age and initiation of pregnancy with drugs. An obstruction of pulmonary vessels by cellular and humoral foetal factors with abrupt pulmonary hypertension and pulmonary heart are discussed to be involved in the pathogenesis of the disease [64], but increasingly also an anaphylactoid reaction in the sense of an anaphylactic pregnancy syndrome.

- Amniotic fluid embolism is a diagnosis by exclusion with the symptom triad of hypoxia, hypotension and DIG; early symptoms are unrest, confusion and dyspnoea. The course is mostly biphasic; after a period of 30 minutes to 9 hours, the initial cardiorespiratory insufficiency is followed by a coagulation disorder [7,62].
- Therapy is symptomatic, cardiopulmonary resuscitation is often initially required. With regard to anaphylaxis, an early high-dosed application of glucocorticoids is recommended next to an initial catecholamine and volume therapy [62]. Therapy of the coagulation disorder and (multiple) organ failure follow the general criteria of intensive medical care.

Endocrinological emergencies during pregnancy

Endocrinological emergencies during pregnancy are seldom. They are mostly imbalances of hyperthyroid or diabetic metabolism [65].

A state of **hyperthyroidism** appears in approx. 1:500 pregnancies [66]. The growth of the foetus, associated with an increased requirement of thyroid gland hormone, is physiologically accounted for by a slight hypertrophy of the maternal thyroid gland. Hypothyroidism might result in premature birth, growth retardation, low foetal weights, or stillbirths, whereas hyperthyroidism leads to uncontrollable vomiting of pregnancy, hypertension, cardiac insufficiency or thyrotoxic crisis.

- Propylthiouracil is the thyreostatic agent of choice in the first trimester, as carbimazole and thiamazole are teratogenic [66]; from the second trimester onward, thiamazole is preferred due to the risk of potential liver damage. The lowest-possible dose is aspired because thyreostatic drugs are known to cross the placental barrier and thus might induce foetal hypothyroidism.
- A thyrotoxic crisis is treated with glucocorticoids (dexamethasone, hydrocortisone), propranolol and, in case of cardiac insufficiency, with diuretics [65], if indicated, with plasmapheresis or with resection of the organ as the ultimate rationale.
- After delivery, the metabolic situation of hyperthyroidism frequently reappears; carbimazole and thiamazole are drugs considered to be safe during lactation [66].

Pregnant women with **type 1 diabetes mellitus** should be attended at a specialised obstetric centre. Maternal insulin sensitivity declines in the second and third trimester of pregnancy, so that the insulin dose has to be increased in most cases. About 2-3% of the affected women develop diabetic ketoacidosis, which may also appear at the end of pregnancy despite nearly normoglycaemic values prevailing [65].

- The therapy of diabetic ketoacidosis is the same as in non-pregnant women and consists of an adequate liquid, electrolyte and insulin substitution. As ketones are capable of crossing the placental barrier the foetus might develop an acidosis which will disappear once the maternal hyperglycaemia is being treated.

References

1. Waterstone M, Bewley S, Wolfe C: Incidence and predictors of severe obstetric morbidity: Case-control study. BMJ 2001;322:1089-1093
2. McClure J, Cooper G; Centre for Maternal and Child Enquiries (CMACE): Saving Mothers' Lives: Reviewing maternal deaths to make motherhood safer: 2006-08. The eighth report on confidential enquiries into maternal deaths in the United Kingdom. Chapter 8: Anaesthesia. BJOG 2011;118: Suppl.1, 102-108
3. Cooper GM, McClure JH: Anaesthesia chapter from Saving mothers' lives; reviewing maternal deaths to make pregnancy safer. Br J Anaesth 2008;100: 17-22
4. Hogan MC, Foreman KJ, Naghavi M, Ahn SY, Wang M, Makela SM, et al: Maternal mortality for 181 countries, 1980-2008: A systematic analysis of progress towards Millennium Development Goal 5. Lancet 2010;375:1609-1623
5. Smit M, Dijkman A, Rijnders M, Bustraan J, van Dillen J, Middeldorp J,

et al: Haemorrhage after home birth: Audit of decision making and referral. Pract Midwife 2013;16:12-15
6. Mavrides E, Allard S, Chandraharan E, Collins P, Green L, Hunt BJ et al; on behalf of the Royal College of Obstetricians and Gynaecologists: Prevention and management of postpartum haemorrhage. BJOG 2016; DOI: .10.1111/1471-0528.14178. https://www.rcog.org.uk/en/guidelines-research-services/guidelines/gtg52/ (am 16.01.2017)
7. Deutsche Gesellschaft für Gynäkologie und Geburtshilfe 2016: Peripartale Blutungen, Diagnostik und Therapie. (S2k-Leitlinie, AWMF Nr. 015-063, Stand 01.04.2016). http://www.awmf.org/leitlinien/detail/ll/015-063.html (am 27.12.2016)
8. Knapp J, Hofer S, Lier H: Anästhesiologisches Vorgehen bei peripartaler Blutung. Anaesthesist 2016;65:225-240
9. Durchführung von Analgesie- und Anästhesieverfahren in der Geburtshilfe. 2. überarbeitete Empfehlungen der Deutschen Gesellschaft für Anästhesiologie und Intensivmedizin und des Berufsverbandes Deutscher Anästhesisten in Zusammenarbeit mit der Deutschen Gesellschaft für Gynäkologie und Geburtshilfe. Anästh Intensivmed 2009;50:S490-S495
10. Crochetière C: Obstetric emergencies. Anesthesiology Clin N Am 2003;21:111-125
11. Begley CM, Gyte GM, Devane D, McGuire W, Weeks A: Active versus expectant management for women in the third stage of labour. Cochrane Database Syst Rev 2011; CD007412
12. Rath W: Aktive Leitung der Nachgeburtsperiode – Das Ende eines 50-jährigen Dogmas? Z Geburtshilfe Neonatol 2013;217:173-176
13. Chamberlain G, Steer P: ABC of labour care: obstetric emergencies. BMJ 1999; 318:1342-1345
14. Palacios-Jaraquemada JM: Caesarean section in cases of placenta praevia and accreta. Best Pract Res Clin Obstet Gynaecol 2013;27:221-232
15. Gleeson C, Scrutton M: Obstetric emergencies. Anaesth Intensive Care Med 2008;9:115-121
16. Torbé A, Mikołajek-Bedner W, Kałużyński W, Gutowska-Czajka D, Kwiatkowski S, Błogowski W, et al: Uterine rupture in the second trimester of pregnancy as an iatrogenic complication of laparoscopic myomectomy. Medicina (Kaunas) 2012;48:182-185
17. Deutsche Gesellschaft für Gynäkologie und Geburtshilfe 2007: Schwangerenbetreuung und Geburtsleitung bei Zustand nach Kaiserschnitt. http://www.awmf.org/leitlinien/detail/ll/015-021.html (Stand August 2010)
18. Westhoff G, Cotter AM, Tolosa JE: Prophylactic oxytocin for the third stage of labour to prevent postpartum haemorrhage. Cochrane Database Syst Rev 2013; CD001808
19. Butwick AJ, Coleman L, Cohen SE, Riley ET, Carvalho B: Minimum effective bolus dose of oxytocin during elective Caesarean delivery. Br J Anaesth 2010; 104:338-343
20. Carvalho JCA, Balki M, Kingdom J, Windrim R: Oxytocin requirements at elective caesarean delivery: A dose-finding study. Obstet Gynecol 2004; 104:1005-1010
21. Thomas JS, Koh SH, Cooper GM: Haemodynamic effects of oxytocin given as i.v. bolus or infusion on women undergoing Caesarean section. Br J Anaesth 2007;98:116-119
22. Postpartale Blutung. Handlungsalgorithmus nach vaginaler Geburt oder in der postoperativen Überwachungsphase nach Sectio caesarea 2012: PPH-Konsensus-Gruppe (D-A-Ch) http://www.postpartum-hemorrhage.com/downloads/algorithmus_a6-20120521.pdf (am 27.12.2016)
23. Novikova N, Hofmeyr GJ, Cluver C: Tranexamic acid for preventing postpartum haemorrhage. Cochrane Data-base Syst Rev 2015: CD007872
24. de Lloyd L, Bovington R, Kaye A, Collis RE, Rayment R, Sanders J, et al: Standard haemostatic tests following major obstetric haemorrhage. Int J Obstet Anesth 2011;20:135-141
25. Steinhard J, Klockenbusch W: Schwangerschaftsinduzierte Hypertonie und Präeklampsie: Risikofaktoren und Vorhersagemöglichkeiten. Gynäkologe 1999; 32:753-760
26. Walker JJ: Severe pre-eclampsia and eclampsia. Baillieres Best Pract Res Clin Obstet Gynaecol 2000;14:57-71
27. Huang WC, Chen CP: Pulmonary edema in pregnancy. Int J Gynecol Obstet 2002; 78:241-243
28. Arulkumaran N, Lightstone L: Severe pre-eclampsia and hypertensive crises. Best Pract Res Clin Obstet Gyn 2013; 27:877-884
29. Deutsche Gesellschaft für Gynäkologie und Geburtshilfe 2014: Diagnostik und Therapie hypertensiver Schwangerschaftserkrankungen. http://www.awmf.org/leitlinien/detail/ll/015-018.html (am 27.12.2016)
30. Tallarek AC, Stepan H: Präeklampsie und HELLP-Syndrom als geburtshilfliche Notfälle. Med Klin Intensivmed Notfmed 2012; 107: 96-100
31. Verlohren S, Galindo A, Schlembach D, Zeisler H, Herraiz I, Moertl MG, et al: An automated method for the determination of the sFlt-1/PIGF ratio in the assessment of preeclampsia. Am J Obstet Gynecol 2010;202:161.e1-161.e11
32. Stepan H, Schaarschmidt W: Bluttest auf Präeklampsie – reif für den klinischen Einsatz? Geburtshilfe Frauenheilkd 2010; 70:6-9
33. Abalos E, Duley L, Steyn DW, Henderson-Smart DJ: Antihypertensive drug therapy for mild to moderate hypertension during pregnancy. Cochrane Database Syst Rev 2007; CD002252
34. O'Brien JM, Shumate SA, Satchwell SL, Milligan DA, Barton JR: Maternal benefit of corticosteroid therapy in patients with HELLP (hemolysis, elevated liver enzymes, and low platelet count) syndrome: Impact on the rate of regional anesthesia. Am J Obstet Gynecol 2002; 186:475-479
35. Woudstra DM, Chandra S, Hofmeyr GJ, Dowswell T: Corticosteroids for HELLP syndrome in pregnancy. Cochrane Database Syst Rev 2010; CD 008148
36. Gremm B, Sohn C: Spezifische geburtshilfliche Risiken. In: Osswald PM, Hartung HJ, Wulf H, Neises M (Hrsg): Geburtshilfliche Anästhesie und Analgesie. Stuttgart: Wissenschaftliche Verlagsgesellschaft; 2003:120-130
37. Stamer UM, Stuber F: Spinal- oder Epiduralanästhesie zur Sectio caesarea bei Präeklampsie. Anasthesiol Intensivmed Notfallmed Schmerzther 2007; 42:200-207
38. Girard T, Brugger S, Hössli I: Neues aus der geburtshilflichen Anästhesie. Anaesthesist 2013;62:963-972
39. Ngan Kee WD, Khaw KS, Ma KC, Wong AS, Lee BB, Ng FF: Maternal and neonatal effects of remifentanil at induction of general anesthesia for cesarean delivery: A randomized, double-blind, controlled trial. Anesthesiology 2006;104:14-20
40. Wallenborn J, Rohrbach A, Stepan H, Kühnert I, Olthoff D: Das peripartale Lungenödem – Anästhesiologische Aspekte eines geburtshilflichen Notfalls. Anästh Intensivmed 2004;45:341-350

41. Acosta CD, Knight M, Lee HC, Kurinczuk JJ, Gould JB, Lyndon A: The continuum of maternal sepsis severity: Incidence and riskfactors in a population-based cohort study. PLoS One 2013; 8(7):e67175
42. Bauer ME, Bateman BT, Bauer ST, Shanks AM, Mhyre JM: Maternal sepsis mortality and morbidity during hospitalization for delivery: Temporal trends and independent associations for severe sepsis. Anesth Analg 2013; 117:944-950
43. Smaill FM, Gyte GM: Antibiotic prophylaxis versus no prophylaxis for preventing infection after cesarean section. Cochrane Database Syst Rev 2010; CD007482
44. Kenyon S, Boulvain M, Neilson JP: Antibiotics for preterm rupture of membranes. Cochrane Database Syst Rev 2013; CD001058
45. Siriwachirachai T, Sangkomkamhang US, Lumbiganon P, Laopaiboon M: Antibiotics for meconium-stained amniotic fluid in labour for preventing maternal and neonatal infections. Cochrane Database Syst Rev 2010; CD007772
46. Heesen M, Klöhr S, Rossaint R, Allegeaert K, Deprest J, Van de Velde M, et al: Concerning the timing of antibiotic administration in women undergoing caesarean section: A systematic review and meta-analysis. BMJ Open 2013; pii:e002028. doi: 10.1136/bmjopen-2012-002028
47. Lucas DN, Robinson PN, Nel MR: Sepsis in obstetrics and the role of the anaesthetist. Int J Obstet Anesth 2012;21:56-67
48. Timezguid N, Das V, Hamdi A, Ciroldi M, Sfoggia-Besserat D, Chelha R, et al: Maternal sepsis during pregnancy or the postpartum period requiring intensive care admission. Int J Obstet Anesth 2012;21:51-55
49. Harper A; Centre for Maternal and Child Enquiries (CMACE): Saving Mothers' Lives: Reviewing maternal deaths to make motherhood safer: 2006-08. The eighth report on confidential enquiries into maternal deaths in the United Kingdom. Chapter 7: Sepsis. BJOG 2011; 118: Suppl.1, 85-101
50. Arulkumaran N, Singer M: Puerpal sepsis. Best Pract Res Clin Obstet Gynecol 2013;27:893-902
51. Ohlsson A, Shah VS: Intrapartum antibiotics for known maternal group B streptococcal colonization. Cochrane Database Syst Rev 2013; CD007467
52. Deutsche Sepsis-Gesellschaft und Deutsche Vereinigung für Intensiv- und Notfallmedizin 2010: S2k-Leitlinie Sepsis – Prävention, Diagnose, Therapie und Nachsorge. http://www.awmf.org/leitlinien/detail/ll/079-001.html. In Überarbeitung (am 15.01.2017)
53. Gabler-Sandberg E: Schwangerschaft und Stillzeit – Antibiotikawahl bei Infektionen. Deutsches Ärzteblatt 2000; 97:A-1158-1159
54. http://www.embryotox.de/einfuehrung.html (am 15.01.2017)
55. Flenady V, Hawley G, Stock OM, Kenyon S, Badawi N: Prophylactic antibiotics for inhibiting preterm labour with intact membranes. Cochrane Database Syst Rev 2013; CD000246
56. Wallenborn J, Kühnert I: Das peripartale Lungenödem: Anästhesiologische Aspekte. In: Eckart J, Jaeger K, Möllhoff T (Hrsg): Anästhesiologie – Kompendium und Repetitorium zur Weiter- und Fortbildung. Landsberg/Lech: ecomed Medizin 2007
57. Davison JM: Edema in pregnancy. Kidney International 1997;51:90-96
58. Stewart LS: Pulmonary edema during pregnancy. NAACOGS Clin Issu Perinat Womens Health Nurs 1992;3:454-460
59. Phelan JP: Pulmonary edema in obstetrics. Obstet Gynecol Clin North Am 1991;18:319-331
60. Reeves M, Frizelle F, Wakeman C, Parker C: Acute colonic pseudo-obstruction in pregnancy. ANZ J Surg 2015;85:728-733
61. Shakir AJ, Sajid MS, Kianifard B, Baig MK: Ogilvie's syndrome-related right colon perforation after cesarean section: A case series. Kaohsiung J Med Sci 2011;27: 234-238
62. Clark SL, Hankins GD, Dudley DA, Dildy GA, Porter TF: Amniotic fluid embolism: Analysis of the national registry. Am J Obstet Gynecol 1995;172: 1158-1169
63. Knight M, Berg C, Brocklehurst P, Kramer M, Lewis G, Oats J, et al: Amniotic fluid embolism incidence, risk factors and outcomes: A review and recommendations. BMC Pregnancy Childbirth 2012;12:7
64. Locksmith GJ: Amniotic fluid embolism. Obstetrics and Gynecology Clinics of North America 1999;26:435-444
65. Khoo CM, Lee KO: Endocrine emergencies in pregnancy. Best Pract Res Clin Obstet Gynaecol 2013;27:885-891
66. Franklin JA, Boelaert K: Thyrotoxicosis. Lancet 2012;379:1155-1166.

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