Shock in pregnancy - recommendations of the German Interdisciplinary Association for Intensive and Emergency Medicine (DIVI –Section Shock) and the Working Group on Obstetrics and Prenatal Medicine (AGG –Section on Maternal Disorders)

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Thieme

Shock in Pregnancy – Recommendations of the German Interdisciplinary Association for Intensive and Emergency Medicine (DIVI – Section Shock) and the Working Group on Obstetrics and Prenatal Medicine (AGG – Section on Maternal Disorders)

Schockzustände in der Schwangerschaft – Empfehlungen der Deutschen Interdisziplinären Vereinigung für Intensiv- und Notfallmedizin (DIVI – Sektion Schock) und der Arbeitsgemeinschaft Geburtshilfe und Pränatalmedizin (AGG – Sektion Maternale Erkrankungen)

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Bibliography

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ABSTRACT

Objective The recommendations of the Shock Section of the German Interdisciplinary Association for Intensive and Emergency Medicine (DIVI) and the Maternal Disorders Section of the Working Group on Obstetrics and Prenatal Medicine (AGG) aim to improve the diagnosis and management of pregnant patients in shock. In 2018, the DIVI Shock Section published a revised classification of shock types. Given that pregnancy involves extensive physiological changes affecting

all organ systems – with direct implications for the development and progression of shock – specific characteristics of shock in pregnancy were analyzed.

Methods A selective literature review and iterative consensus process were conducted within the DIVI Shock Section and the Maternal Disorders Section of the AGG.

Results Shock, defined as a state of circulatory failure characterized by a critical mismatch between oxygen delivery (DO_2) and consumption (VO_2), is common to all shock types, including in pregnant women. Unique features of pregnancy include altered sensitivity to triggering factors, modified classical shock symptoms, and specific diagnostic and therapeutic approaches to optimize outcomes for both mother and child.

Conclusions The statements and recommendations facilitate the identification of underlying causes across the different forms of shock (hypovolemic, distributive, cardiogenic, and obstructive) and support the initiation of appropriate management strategies.

ZUSAMMENFASSUNG

Ziel Die Empfehlungen der Sektion Schock der Deutschen Interdisziplinären Vereinigung für Intensiv- und Notfallmedizin (DIVI) und der Sektion Maternale Erkrankungen der AGG (Arbeitsgemeinschaft Geburtshilfe und Pränatalmedizin) der DGGG haben das Ziel der Verbesserung der Diagnostik und des Managements von Schwangeren in einer Schocksituation. Im Jahr 2018 wurde von der Sektion Schock der DIVI eine veränderte Definition der Schockformen publiziert. Da eine Schwangerschaft zu umfangreichen physiologischen Veränderungen, die jedes Organsystem betreffen und unmittelbare Auswirkungen auf Schockentstehung und -verlauf haben können, führt, wurden die Spezifika der Schockformen in der Schwangerschaft herausgearbeitet.

Methode Es erfolgte eine selektive Literaturrecherche und iterative Konsensbildung innerhalb der Sektion Schock der DIVI und der Sektion Maternale Erkrankungen der Arbeitsgemeinschaft Geburtshilfe und Pränatalmedizin (AGG).

Ergebnisse Schock als Kreislaufinsuffizienz mit einem gravierenden Missverhältnis von O_2-Angebot (DO_2) und Bedarf (VO_2) bleibt allen Schockformen, auch bei der Schwangeren, gemeinsam. Als Besonderheiten der Schwangerschaft werden eine veränderte Empfindlichkeit gegenüber auslösenden Ursachen, veränderte klassische Schocksymptome und die speziellen diagnostischen und therapeutischen Ansätze zur Rettung von Mutter und Kind behandelt.

Schlussfolgerungen Die Statements und Empfehlungen helfen, die zwischen den verschiedenen Schockformen (hypovolämischer Schock, distributiver Schock, kardiogener Schock und obstruktiver Schock) vorhandenen Ursachen leichter zu erkennen und das entsprechende Management einzuleiten.

Introduction

STATEMENT

There are four main categories of shock, each of which is mainly related to one of four organ systems:

- hypovolemic shock relates to the blood and fluids compartment
- distributive shock relates to the vascular system
- cardiogenic shock relates to the heart
- obstructive shock relates to the circulatory system

With the start of pregnancy, the female organism undergoes many different physiological changes and adaptations which affect all organ systems (▶ **Table 1**) and are aggravated in the event of illness. The revision of the nomenclature, definition, and classification of different forms of shock into four main categories relates each one to one of four organ systems systems according to the predominant aspects [1]:

- hypovolemic shock relates to the blood and fluids compartment
- distributive shock relates to the circulatory system
- cardiogenic shock relates to the heart
- obstructive shock relates to the circulatory system

Hypovolemic shock results from a loss of volume in absolute terms. Distributive shock leads to relative hypovolemia resulting from a pathological redistribution of intravascular volumes. Cardiogenic shock is caused by inadequate cardiac function; obstructive shock is the result of a resistance-related pathology leading to malperfusion. This analysis of different forms of shock with a focus on particular aspects occurring in pregnancy aims to facilitate targeted diagnosis and improve the preclinical and clinical management of shock in pregnant women.

Method

These recommendations were compiled by a working group from the Shock Section of the German Interdisciplinary Association for Intensive and Emergency Medicine (DIVI) and the Maternal Disorders Section of the Working Group on Obstetrics and Prenatal Medicine of the German Society for Gynecology and Obstetrics (AGG i. d. DGGG). It was based on a selective literature search of the PubMed database up to and including May 2024. Search terms included “pregnancy AND shock”, “obstetric hemorrhagic shock”, “cardiogenic shock AND pregnancy”, “septic shock AND pregnancy”, and “obstructive shock AND pregnancy”. National and international guidelines (e.g., AWMF, NICE, SCCM, Surviving Sepsis Campaign) were additionally consulted.

The contents of relevant articles identified in the literature were evaluated by the working group. When the data was contradictory, it was classified according to clinical relevance, plausibility, and transferability in an obstetric intensive medicine context. Recommendations and statements were iteratively discussed at several digital consensus meetings and adopted by consensus. The aim was to formulate practical recommendations for the dif-

▶ **Table 1** Physiological changes of organ function during pregnancy.

Respiratory system	Progesterone-induced increased CO_2 sensitivity
	Increase in respiratory volume and respiratory rate by up to 40 %
	Increase in respiratory minute volume by up to 50 %
	Reduction of FRC by 20 %
	Increase in O_2 consumption by 20–25 %
	Mild chronic hyperventilation and respiratory alkolosis pO_2 + 10 mmHg, pCO_2 − 10 mmHg, maternal pH between 7.40–7.45
	Estrogen-induced increase in perfusion and edema formation in the upper respiratory tract
Circulatory system	Synthesis of progesterone, prostacyclin PGI2, increased NO
	Decreased vascular smooth musculature tone with SVR − 21 % and PVR − 34 %
	Decreased effective circulatory blood volume (relative hypovolemia) at the start of pregnancy
	Activation of the renin-angiotensin-aldosterone system with increased sodium and water retention (2nd + 3rd trimester of pregnancy)
	Increase of SV + 30 %, HR + 17 %, CO + 40 %, uterine blood flow at the time of delivery up to 900 ml/min (≈ 14 % of CO)
Blood system	Increase in total blood volume by up to 40 %, plasma volume by up to 50 %, erythrocyte volume by up to 30 % with or 20 % without iron substitution
	Anemia (dilution and iron deficiency due to fetal requirements), Hct − 5 to − 10 % (with or without iron substitution, respectively)
	Hypoalbuminemia with tendency to edema formation
Coagulation system	Increase of Factors I, VII, VIII, IX, X, XII
	Decrease of proteins C and S, physiological APC resistance
	Decreased tPA, PTT and clotting time
	Thromboembolism rate in pregnancy 0.05–1.8 % (~ six times higher)
	Thromboembolism rate peripartum increased up to 14.4-fold
Gastrointestinal system	Gastric emptying is not delayed, not even with obesity
	Gastric acid secretion unchanged during pregnancy
	Decreased lower esophageal sphincter pressure due to effect of progesterone, increased intraabdominal pressure, stomach displacement
	Gastric emptying is delayed after the start of labor, especially under opioid therapy (IV, s. a. and epidural)
	A light meal 2–4 h prior to cesarean section increases the intragastric volume and reduces pH
Renal function	Increased renal blood flow by up to + 60 %
	Increased glomerular filtration rate + 60 %
	Elevated aldosterone levels promote Na^+ and water retention and edema formation (see above)
Central and peripheral nervous system	Sedative effect of progesterone
	Reduced MAC for volatile anesthetics − 40 %
	Increased sensitivity to local anesthetics (respiratory alkalosis increases diffusion of non-ionized LA to the nerves)
	Increased sensitivity to LA intoxication (relative hypalbuminemia, lower seizure threshold)

FRC: functional residual capacity; SVR: systemic vascular resistance; PVR: pulmonary vascular resistance; CO: cardiac output (l/min); MAC: minimum alveolar concentration (volatile anesthetics)

ferentiated diagnosis and therapy of different forms of shock in pregnancy which can be applied in both an obstetric and in an intensive medical care setting.

RECOMMENDATION

Treatment of hypovolemic shock should consist of rapid hemostasis and intravascular volume substitution (balanced crystalloid solutions).

RECOMMENDATION
A vasoconstrictor (e.g., noradrenaline) should be administered to pregnant patients with hypovolemic shock and persistent hypotension to achieve a systolic arterial blood pressure (SAP) of ≥ 100 mmHg.

RECOMMENDATION
Tranexamic acid (1 g) with the addition of fibrinogen (2–4 g) in cases with persistent bleeding should be administered early to patients in hypovolemic shock caused by traumatic or peripartum hemorrhage.

Hypovolemic shock is characterized by critically reduced cardiac preload due to volume loss [1]. Due to the increase in plasma and erythrocyte volumes, pregnant women can tolerate a greater loss of blood compared to non-pregnant women.

Obstetric hemorrhagic shock is mainly caused by peripartum bleeding arising from uterine atony, placental remnants, and childbirth injuries. The risk of bleeding is especially high when bleeding occurs in the context of placenta accreta spectrum disorders [2]. A critical decrease in circulating blood volume triggers shock; massive erythrocyte loss intensifies tissue hypoxia.

Traumatic hemorrhagic shock is additionally characterized by significant tissue trauma, early clotting disorder, and endothelial damage, e.g., arising in the context of polytrauma [3–6]. In pregnant women it can be caused by preterm placental abruption, with hemorrhage and uterine tissue trauma (Couvelaire uterus) leading to hypovolemia [7].

Hypovolemic shock in its narrow defintion and traumatic hypovolemic shock are both characterized by a relevant loss of fluid (external or internal fluid loss) without bleeding. Causes can include persistent vomiting, for example due to hyperemesis gravidarum, and diarrhea as well as uncompensated renal loss (diabetes insipidus, hyperosmolar diabetic coma). Extravascular sequestration of large fluid volumes due to ileus, acute pancreatitis, or eclampsia also results in a significant reduction of circulating plasma volumes.

Hypovolemic shock treatment consists of rapid hemostasis and intravascular volume substitution. Substitution is done with balanced crystalloid solutions. If hypotension persists, a vasoconstrictor (e.g., noradrenaline) must be administered to achieve a systolic arterial blood pressure (SAP) of ≥ 100 mmHg. In cases with traumatic or excessive peripartum bleeding, tranexamic acid (1 g) must be additionally administered at an early stage followed by the administration of fibrinogen (2–4 g) if bleeding persists [8]. The treatment of hemorrhagic shock arising in the context of peripartum hemorrhage has been summarized in the AWMF guideline "Peripartum Haemorrhage, Diagnosis and Therapy" (S2k, AWMF Registry No. 015–063) [2]. The prepartum treatment algorithm must always take the decrease in fetal supply into account.

Distributive Shock

STATEMENT
Distributive shock arises from a pathological redistribution of initially unchanged absolute intravascular volumes into the interstitium. Distributive shock is differentiated into septic shock and neurogenic shock.

Relative hypovolemia is present in distributive shock as a consequence of the pathological redistribution of initially unchanged absolute intravascular volumes. Causes of distributive shock are a loss in the regulation of vasotonus followed by a permeability disorder of the vascular system with redistribution of intravascular volumes into the interstitium [1].

Septic shock

STATEMENT
Septic shock is defined as a lactate value of > 2 mmol/l with persistent hypotension which necessitates the administration of vasopressors to maintain a mean arterial blood pressure of (MAP) > 65 mmHg.

RECOMMENDATION
Early recognition of sepsis in patients is important. Scores adjusted to the changes in pregnant women could be useful to facilitate diagnosis in pregnancy.

RECOMMENDATION
The treatment of septic shock in pregnant patients must reconcile concerns about maternal safety with fetal interests.

Sepsis is a dysregulated bodily response to an infection which results in life-threatening organ dysfuction. Organ dysfunctions are quantified by increases in the SOFA (sequential organ failure assessment) score by ≥ 2 points [9]. Septic shock is defined as a lactate value of > 2 mmol/l with persistent hypotension which necessitates the use of vasopressors to maintain a mean arterial blood pressure (MAP) of > 65 mmHg [10]. Pathophysiologically, sepsis is the result of immune pathology-triggered endothelial dysfunction with vasodilation, hypotension, and pathological vascular permeability which leads to intravascular volume loss (capillary leak syndrome). Sepsis-related cardiac depression may occur and exacerbate hypotension [11]. Early identification of patients with sepsis is key with regard to the further course of disease [12].

When caring for pregnant women it is important to be aware that physiologically, tachycardia > 90/min and tachypnea with leukocyte values of up to 16 000/µl may be classified as normal findings, which greatly limits the validity of standard scoring systems. Scores adapted to physiological pregnancy-related changes such as the Maternal Early Warning Score (MEWS) are useful when making a diagnosis [13]. MEWS includes an assessment and evaluation of blood pressure, heart rate, oxygen saturation, temperature, and consciousness using a point-based scale, with higher scores indicating a greater risk. Alternatively, scores with appropriately adapted thresholds such as the obstetrically modified SOFA score (omSOFA) may be used [14].

Sepsis with *Group A Streptococci* is a typical complication of puerperium and is associated with an extremely serious course, especially when the diagnosis is made too late. Infants, older and immune-supressed persons, and pregnant women all have a high risk of influenza complications and a fourfold higher risk of requiring admission to hospital, intensive care, or death. The pregnancy-related changes to the cardiovascular and immune systems result in delayed recovery after viral infection and a longer and more severe course of disease. Pregnant women are more affected in the 2nd and 3rd trimester of pregnancy. The most common comorbidity is bronchial asthma [15]. Cleft lip and palate, neural tube defects, congenital heart defects, neurological disorders and schizophrenia have all been reported in fetuses after maternal infection with influenza [16]. Early antiviral therapy is recommended because of the increased maternal and fetal risk [15]. The majority of pregnant or postpartum patients who require treatment in an intensive care unit due to infection with influenza A or who died from the infection are not vaccinated, which is why vaccination is recommended to pregnant women during flu season [15].

Pregnant women have a higher risk of admission to hospital, treatment in an intensive care unit, and death compared to non-pregnant women and have a high risk of preterm or stillbirth following SARS-CoV-2 infection [17]. A retrospective study reported that 5.5 % of 793 pregnant women positive for COVID-19 were admitted to an intensive care unit and 1.3 % died. Diagnosis of shock in the 3rd trimester, maternal age, and high maternal BMI have been identified as risk factors for a severe course of disease [18, 19]. Delivery of the infant only improves oxygenation. Preterm delivery is mainly initiated for maternal indications. Risk factors for maternal death are high BMI and comorbidities; risk factors for fetal or neonatal mortality are gestational age at delivery and the SOFA score in the first 24 hours [18].

In addition to repeat measurements of blood lactate values, culturing blood samples, and the administration of broad-spectrum antibiotics [20, 21], infusion of balanced crystalloid solutions (30 ml/kg body weight) and the administration of vasopressors until a MAP of 65 mmHg is reached are the most important therapeutic measures which should be initiated in the first hour to achieve initial stabilization of the patient [14]. Vasopressors can result in a decrease in uterine perfusion with reduced fetal blood supply. But untreated maternal hypotension also has this effect, meaning that the treatment of pregnant women with septic shock must always try to reconcile concerns about maternal safety with fetal interests [22].

Neurogenic shock

STATEMENT

Neurogenic shock is characterized by an acute decrease in systolic arterial blood pressure to less than 100 mmHg and a decreased heart rate of < 60 beats per minute and is associated with clouding of consciousness and a loss of spinal reflexes in cases with high spinal cord injury.

STATEMENT

Treatment of the cause is decisive when treating neurogenic shock.

An imbalance between the vegetative regulation of cardiac activity and the vascular musculature can lead to neurogenic shock. It is characterized by an acute decrease of the SAP to < 100 mmHg and of the heart rate to < 60/min and is associated with clouding of consciousness and a loss of spinal reflexes in cases with high spinal cord injury [1].

The pathomechanisms of neurogenic shock are divided into

- *direct damage to the centers for circulatory control* caused by trauma (brainstem trauma) or stroke. The most common causes in pregnancy or puerperium are preeclampsia/eclampsia, cerebral venous sinus thrombosis, and reversible cerebral vasoconstriction syndrome (postpartum angiopathy). In addition to excluding clotting disorders, it is also important to exclude cerebral aneurysm, arteriovenous malformations, and moyamoya syndrome [23, 24].
- *Afference changes to the cardiovascular regulatory center* in the medulla oblongata triggered by fear, stress, or pain, e.g., during delivery, or
- *interruption of the descending connection* between the bulbar region and the spinal cord (quadriplegia syndrome). Neurogenic shock can also be caused by meningitis (infection and septic shock), seizures (preeclampsia), or rapidly ascending Guillain-Barré syndrome [25, 26].

Treatment of the cause is decisive when treating neurogenic shock. Initial treatment must focus on hemodynamic stabilization. In addition to rapid volume therapy, treatment also consists of administration of noradrenaline which can impair placental perfusion and may induce fetal bradycardia. In the late stage of pregnancy there may be a risk of fetal asphyxia due to uterine contractions. Bradycardia is treated with atropine or glycopyrrolate. Although the overall data are limited, there are no indications of teratogenicity, and administration of these medications in pregnancy is possible after careful reviewing the indications [27, 28]. Direct or indirect sympathomimetic drugs such as phenylephrine, cafedrine, theodrenaline, and droxidopa may be used to restore vascular tone [27]. Mineralocorticoids such as prednisolone and prednisone to increase plasma volume and prevent sodium loss are additionally recommended [28].

Cardiogenic Shock

STATEMENT
Clinically, cardiogenic shock is defined as systolic arterial blood pressure < 90 mmHg or a mean pressure of 30 mmHg below baseline and a cardiac index (CI) < 1.8 l/min/m^2 without pharmacological or mechanical support.

STATEMENT
Common peripartum causes of cardiogenic shock are peripartum cardiomyopathy and amniotic fluid embolism.

RECOMMENDATION
If stress apnea is present, it is important to watch for clinical signs of (cardiogenic) shock as cardiac decompensation is often not recognized.

Cardiogenic shock is primarily a disorder of cardiac function which takes the form of a critical reduction of cardiac pumping capacity. Clinically, cardiogenic shock is defined as SAP < 90 mmHg or a mean blood pressure of 30 mmHg below baseline and a cardiac index (CI) < 1.8 l/min/m^2 without pharmacological or mechanical support [1].

One study found that 59% of cases occurred postpartum, 23% during the second stage of labor, and 18% developed antepartum [29]. The most common cause with an incidence of 1/3000 was peripartum cardiomyopathy, although 25% of cases developed prepartum. Amniotic fluid embolism is a relatively common cause of combined forms of shock with a cardiogenic aspect and can develop both antepartum and peripartum [29]. Shock resulting from acute coronary syndrome develops in around 1/150 000 pregnancies. In rare cases, mechanical problems can lead to cardiogenic shock in cases with pre-existing (rheumatic) valvular disease or acute endocarditis.

Physiologically, pregnant women have a higher cardiac output (CO) and lower peripheral resistance and lower blood pressure. The first peak CO is often reached around the 24th week of gestation: cardiac decompensation therefore often occurs in this period or immediately postpartum. Pregnancy is often referred to in this context as a form of "cardiac stress test" [30].

Symptoms are often trivialized when stress apnea is present, and cardiac decompensation is often not recognized. Dyspnea, a systolic blood pressure < 90 mmHg, a heart rate > 130/min or < 45/min, a breathing rate > 25/min, oxygen saturation < 90%, lactate > 2.0 mmol/L or central venous oxygenation saturation < 60% are clinical signs of (cardiogenic) shock in pregnant women.

After a diagnosis has been made, the cause must be identified. In addition to taking the patient's medical history and a clinical examination, ECG, echocardiography, and BNP testing are useful for diagnosis. The threshold before carrying out radiological tests in pregnant women is high. It would therefore be useful to assess the status of patients with pre-existing cardiac conditions prior to pregnancy. Close cooperation between the involved medical specialties is always necessary. The threshold before carrying out invasive coronary diagnostic tests is very high.

Treatment of a pregnant woman must always also take the unborn child and adequate perfusion of the placenta into consideration. Treatment and care should therefore be primarily provided by specialized centers where possible; if this is not possible, a transfer should be requested [31]. Treatment of peripartum cardiomyopathy often consists of timely induction of labor depending on the fetal development status. Specific treatment of acute coronary syndrome in pregnant women corresponds to the treatment provided to non-pregnant women [32, 33], including the administration of dual antiplatelet therapy.

Supportive treatment of cardiogenic shock requires the initial optimization of intravascular volumes as well as the optimization of oxygenation using non-invasive ventilation or ventilation. Catecholamine therapy in pregnant women can include the administration of dobutamine, adrenaline or noradrenaline. Data on the use of phenylephrine in pregnant women is good, especially in the context of cesarean sections [34]. Levosimendan infused over 24 hours can also improve cardiac pumping function [31]. Fetotoxic substances such as ACE inhibitors, AT1 blockers and atenolol should be avoided. However, loop diuretics, thiazide diuretics, hydralazine, nitrates, and beta blockers are well tolerated. Bromocriptine may be used to treat peripartum cardiomyopathy but it must be accompanied by at least prophylactic heparinization [31]. If these specific measures and supportive pharmacological therapy are unable to terminate cardiogenic shock, mechanical circulatory support such as use of an Impella pump or extracorporeal membrane oxygenation may be initiated [35].

Obstructive Shock

STATEMENT
Obstructive shock is a consequence of mechanical intravascular or extravascular factors obstructing blood flow in the great vessels or cardiac outflow.

Obstructive shock is the result of mechanical intravascular or extravascular factors obstructing blood flow in the great vessels or cardiac outflow [1]. The most common causes are

- pericardial tamponade
- tension pneumothorax
- inferior vena cava syndrome
- thromboembolism with pulmonary embolism
- amniotic fluid embolism

The first two causes have already been discussed in detail in earlier articles [1, 36].

Inferior vena cava syndrome is caused by acute pressure created by the fetus which results in a reduction of venous backflow to the right side of the maternal heart, resulting in low cardiac output

syndrome. This syndrome mostly occurs in the last trimester of pregnancy if the mother has spent a lot of time lying on her back; the resultant hypotension or shock may also significantly decrease placental perfusion. Treatment consists of rapid repositioning of the pregnant woman onto her left side. If cardiopulmonary resuscitation is required, it should be carried out with the patient in a supine position because resuscitation in this position is more effective but an assistant must shift the pregnant uterus to the left during resuscitation (left uterine displacement).

Pulmonary embolisms are relatively common in pregnant women with an incidence of around 1/20 000. As D-dimer levels are higher in pregnancy and can therefore not be assessed, diagnosis must be based on arterial blood gas analysis, duplex sonography, a modified Wells score, and also on low-dose computed tomography or magnetic resonanice imaging if necessary [31]. In pregnant women, heparin, low molecular weight heparin, or fondaparinux may be used for specific anticoagulation of pulmonary embolisms.

Amniotic fluid embolism is uncommon and occurs during labor or immediately postpartum within 30 min after delivery of the placenta with disruption of the fetomaternal barrier and an ingress of amniotic fluid into the maternal circulation [37]. In addition to obstruction of the pulmonary circulation, the immune system is activated, resulting in the release of vasoactive, cardiodepressive and procoagulant mediators. The early stage of this disorder is characterized by pulmonary vasospasm and hypertension as well as right heart failure. The consumption of coagulation factors directly results in acute coagulopathy with bleeding (hemorrhagic shock). During the further course of the disorder, the symptoms “left ventricular insufficiency” and “immune-mediated vasodilation” (distributive shock) often lead to multiorgan failure. The diagnosis is based on the sequence of symptoms, beginning with sudden cardiovascular collapse, followed by acute coagulopathy, even before any relevant bleeding has occurred. Amniotic fluid embolism is diagnosed by exclusion. There is currently no causal therapy for amniotic fluid embolism; treatment of this form of combined shock is purely symptomatic and ranges from the administration of catecholamines to replacement of clotting factors and cardiopulmonary resuscitation [38, 39].

Preeclampsia, Eclampsia and HELLP Syndrome

STATEMENT
Different pathophysiological changes occurring in the context of preeclampsia, eclampsia or HELLP syndrome may lead to shock.

Different pathophysiological changes arising in the context of hypertensive disorders of pregnancy and their complications can lead to shock. Progression of preeclampsia to severe eclampsia results in fluid sequestration in the interstitium (hypovolemic shock) and often additionally leads to left ventricular insufficiency with pulmonary edema (cardiogenic shock). Renal vasoconstriction can lead to acute renal failure. Eclampsia can cause seizures, and intracranial bleeding may develop when blood pressure increases to > 160/110 mmHg (neurogenic shock).

HELLP syndrome with hemolysis (H), elevated liver enzymes (EL) and low platelets (LP) occurs in 10–20 % of cases. The typical symptom of epigastric pain is caused by impaired hepatic perfusion which may lead, in rare cases, to hepatic rupture with life-threatening bleeding (hemorrhagic shock). The differential diagnosis of thrombocytopenia must exclude aHUS (atypical hemolytic uremic syndrome), acute fatty liver of pregnancy, and TTP (thrombotic thrombocytopenic purpura) [6].

Premature placental abruption can occur in the context of preeclampsia/eclampsia, leading to impaired fetal supply. Just like hepatic rupture caused by HELLP syndrome, placental abruption requires immediate emergency cesarean section.

Conclusion

The (patho-)physiological changes of pregnancy result in specific diagnostic and therapeutic features, even though the basic measures used to rectify the effects of shock do not differ much from those used when treating non-pregnant women. With all forms of shock in pregnant women, attention must focus on the fetal outcome. Depending on gestational age and estimated fetal weight, (emergency) cesarean section may improve the mother’s condition and may (also) save the child’s life.

Conflict of Interest

The authors declare that they have no conflict of interest.

References

[1] Standl T, Annecke T, Cascorbi I et al. The Nomenclature, Definition and Distinction of Types of Shock. Dtsch Arztebl Int 2018; 115: 757–768. DOI: 10.3238/arztebl.2018.0757

[2] Schlembach D, Annecke T, Girard T et al. Peripartum Haemorrhage, Diagnosis and Therapy. Guideline of the DGGG, OEGGG and SGGG (S2k, AWMF Registry No. 015–063, August 2022). Geburtshilfe Frauenheilkd 2023; 83: 1446–1490. DOI: 10.1055/a-2073-9615

[3] Gänsslen A, Adams HA, Baumann G et al. Haemostasis in shock Part 4: Special pathophysiological aspects. Anästh Intensivmed 2016; 57: 58–67

[4] Deitch EA, Condon M, Feketeova E et al. Trauma-hemorrhagic shock induces a CD36-dependent RBC endothelial-adhesive phenotype. Crit Care Med 2014; 42: e200–e210. DOI: 10.1097/CCM.0000000000000119

[5] Slaughter AL, Nunns GR, D’Alessandro A et al. The Metabolopathy of Tissue Injury, Hemorrhagic Shock, and Resuscitation in a Rat Model. Shock 2018; 49: 580–590. DOI: 10.1097/SHK.0000000000000948

[6] Pourrat O, Coudroy R, Pierre F. Differentiation between severe HELLP syndrome and thrombotic microangiopathy, thrombotic thrombocytopenic purpura and other imitators. Eur J Obstet Gynecol Reprod Biol 2015; 189: 68–72. DOI: 10.1016/j.ejogrb.2015.03.017

[7] Jackson J, George V, McKinney J et al. Multimodal postpartum imaging of a severe case of Couvelaire uterus. Case Rep Perinat Med 2022; 11: 20210013. DOI: 10.1515/crpm-2021-0013

[8] WOMAN Trial Collaborators. Effect of early tranexamic acid administration on mortality, hysterectomy, and other morbidities in women with post-partum haemorrhage (WOMAN): an international, randomised, double-blind, placebo-controlled trial. Lancet 2017; 389: 2105–2116. DOI: 10.1016/S0140-6736(17)30638-4

[9] Singer M, Deutschman CS, Seymour CW et al. The Third International Consensus Definitions for Sepsis and Septic Shock (Sepsis-3). JAMA 2016; 315: 801–810. DOI: 10.1001/jama.2016.0287

[10] Shankar-Hari M, Phillips GS, Levy ML et al. Developing a New Definition and Assessing New Clinical Criteria for Septic Shock: For the Third International Consensus Definitions for Sepsis and Septic Shock (Sepsis-3). JAMA 2016; 315: 775–787. DOI: 10.1001/jama.2016.0289

[11] Antonucci E, Fiaccadori E, Donadello K et al. Myocardial depression in sepsis: from pathogenesis to clinical manifestations and treatment. J Crit Care 2014; 29: 500–511. DOI: 10.1016/j.jcrc.2014.03.028

[12] Evans L, Rhodes A, Alhazzani W et al. Surviving sepsis campaign: international guidelines for management of sepsis and septic shock 2021. Intensive Care Med 2021; 47: 1181–1247. DOI: 10.1007/s00134-021-06506-y

[13] Carle C, Alexander P, Columb M et al. Design and internal validation of an obstetric early warning score: secondary analysis of the Intensive Care National Audit and Research Centre Case Mix Programme database. Anaesthesia 2013; 68: 354–367. DOI: 10.1111/anae.12180

[14] Louie JK, Salibay CJ, Kang M et al. Pregnancy and severe influenza infection in the 2013–2014 influenza season. Obstet Gynecol 2015; 125: 184–192. DOI: 10.1097/AOG.0000000000000593

[15] Bowyer L, Robinson HL, Barrett H et al. SOMANZ guidelines for the investigation and management sepsis in pregnancy. Aust N Z J Obstet Gynaecol 2017; 57: 540–551. DOI: 10.1111/ajo.12646

[16] Oseghale O, Vlahos R, O'Leary JJ et al. Influenza Virus Infection during Pregnancy as a Trigger of Acute and Chronic Complications. Viruses 2022; 14: 2729. DOI: 10.3390/v14122729

[17] Allotey J, Stallings E, Bonet M et al. Clinical manifestations, risk factors, and maternal and perinatal outcomes of coronavirus disease 2019 in pregnancy: living systematic review and meta-analysis. BMJ 2020; 370: m3320. DOI: 10.1136/bmj.m3320

[18] Vasquez DN, Giannoni R, Salvatierra A et al. Ventilatory Parameters in Obstetric Patients With COVID-19 and Impact of Delivery: A Multicenter Prospective Cohort Study. Chest 2023; 163: 554–566. DOI: 10.1016/j.chest.2022.10.010

[19] Kalafat E, Prasad S, Birol P et al. An internally validated prediction model for critical COVID-19 infection and intensive care unit admission in symptomatic pregnant women. Am J Obstet Gynecol 2022; 226: 403.e1–403.e13. DOI: 10.1016/j.ajog.2021.09.024

[20] Gotts JE, Matthay MA. Sepsis: pathophysiology and clinical management. BMJ 2016; 353: i1585. DOI: 10.1136/bmj.i1585

[21] Brunkhorst FM, Weigand MA, Pletz M et al. [S3 Guideline Sepsis-prevention, diagnosis, therapy, and aftercare : Long version]. Med Klin Intensivmed Notfmed 2020; 115: 37–109. DOI: 10.1007/s00063-020-00685-0

[22] Neuhaus S, Neuhaus C, Weigand MA et al. [Principles of intensive medical care in pregnant patients]. Anaesthesist 2021; 70: 621–630. DOI: 10.1007/s00101-021-00947-2

[23] Berlit P. [Neurological Disorders and Pregnancy]. Fortschr Neurol Psychiatr 2016; 84: 103–111; quiz 112–113. DOI: 10.1055/s-0041-108911

[24] Bateman BT, Schumacher HC, Bushnell CD et al. Intracerebral hemorrhage in pregnancy: frequency, risk factors, and outcome. Neurology 2006; 67: 424–429. DOI: 10.1212/01.wnl.0000228277.84760.a2

[25] Kowalski A, Brandis D. Shock Resuscitation. In: StatPearls. Treasure Island (FL): StatPearls Publishing; 2024

[26] Stein DM, Knight WA. Emergency Neurological Life Support: Traumatic Spine Injury. Neurocrit Care 2017; 27: 170–180. DOI: 10.1007/s12028-017-0462-z

[27] Briggs GG, Freeman RK, Tower CV et al. Brigg's Drugs in Pregnancy and Lactation: A Reference Guide to Fetal and Neonatal Risk. Online (Stand: 27.11.2024): https://www.wolterskluwer.com/en/solutions/ovid/briggs-drugs-in-pregnancy-and-lactation-a-reference-guide-to-fetal-and-neonatal-risk-730

[28] Abboud T, Raya J, Sadri S et al. Fetal and maternal cardiovascular effects of atropine and glycopyrrolate. Anesth Analg 1983; 62: 426–430

[29] Banayan J, Rana S, Mueller A et al. Cardiogenic shock in pregnancy: Analysis from the National Inpatient Sample. Hypertens Pregnancy 2017; 36: 117–123. DOI: 10.1080/10641955.2016.1242606

[30] Chung E, Leinwand LA. Pregnancy as a cardiac stress model. Cardiovasc Res 2014; 101: 561–570. DOI: 10.1093/cvr/cvu013

[31] Regitz-Zagrosek V, Roos-Hesselink JW, Bauersachs J et al. 2018 ESC Guidelines for the management of cardiovascular diseases during pregnancy. Eur Heart J 2018; 39: 3165–3241. DOI: 10.1093/eurheartj/ehy340

[32] Davis MB, Arany Z, McNamara DM et al. Peripartum Cardiomyopathy: JACC State-of-the-Art Review. J Am Coll Cardiol 2020; 75: 207–221. DOI: 10.1016/j.jacc.2019.11.014

[33] James AH, Jamison MG, Biswas MS et al. Acute myocardial infarction in pregnancy: a United States population-based study. Circulation 2006; 113: 1564–1571. DOI: 10.1161/CIRCULATIONAHA.105.576751

[34] Prakash S, Pramanik V, Chellani H et al. Maternal and neonatal effects of bolus administration of ephedrine and phenylephrine during spinal anaesthesia for caesarean delivery: a randomised study. Int J Obstet Anesth 2010; 19: 24–30. DOI: 10.1016/j.ijoa.2009.02.007

[35] Furer A, Wessler J, Burkhoff D. Hemodynamics of Cardiogenic Shock. Interv Cardiol Clin 2017; 6: 359–371. DOI: 10.1016/j.iccl.2017.03.006

[36] Pich H, Heller AR. [Obstructive shock]. Anaesthesist 2015; 64: 403–419. DOI: 10.1007/s00101-015-0031-9

[37] Pacheco LD, Clark SL, Klassen M et al. Amniotic fluid embolism: principles of early clinical management. Am J Obstet Gynecol 2020; 222: 48–52. DOI: 10.1016/j.ajog.2019.07.036

[38] Tschöp J, Lier H, Annecke T. Anästhesiologisches Management der Fruchtwasserembolie. Anasthesiol Intensivmed Notfallmed Schmerzther 2020; 55: 674–685. DOI: 10.1055/a-1070-6843

[39] Kaur K, Bhardwaj M, Kumar P et al. Amniotic fluid embolism. J Anaesthesiol Clin Pharmacol 2016; 32: 153–159. DOI: 10.4103/0970-9185.173356