

Fostervattenemboli

SFOAI vårmöte 2023

Ove Karlsson, MD PhD

Sahlgrenska, Sverige och världen

36-årig kvinna

Vecka 39+2

- BMI 27
- Viktuppgång 25 kg
- Astma
- Singelnjure
- Rökare
- IV-gravida, III-para (PN)

- PROM
- Ultraljud
 - Placenta framvägg
 - Ej föreliggande
- Hem

36-årig kvinna

- 8.15 värkar inlagd
- 8.35 kramp
 - Susp eklampsi
 - Nål svårt, stesolid rektalt
- 8.42 magnesium bolus + inf
- 8.51 Bltr 192/106 p 115
 - Trandate iv
 - Fortsatt medvetslös
- 8.53 fosterbradykardi
 - Larmsnitt (Op sal på avd)

Vecka 39+3

- 8.54 flytt till operationsbord
 - Andningsstopp
 - Intubation
 - Första andetaget imma och CO2 retur
 - Fjärde andetaget ingen CO2 retur
- 8.57 cirkulationskollaps
 - HR 33 – atropin (pvk subkutan)
 - Hjärtstopp – HLR, adrenalin
 - Hjärtlarm och perimortem snitt
 - Barn utskaffas lätt Apgar 1-4-4
 - Ingen ablatio eller blod i buk

36-årig kvinna

- HLR
 - Artärnål (bra pulsationer)
 - Utbyte CO2
 - Mätbara blodtryck
- Perimortem snitt
 - Inget blod eller ablatio
 - Peanger över kärl och packar buk
- Adrenalin 1 mg x 8
- Calcium
- Tribonat
- Blodgas
- Svårt med nålar
- Tilltagande stor blödning uterus
 - 2 st 0 neg blod

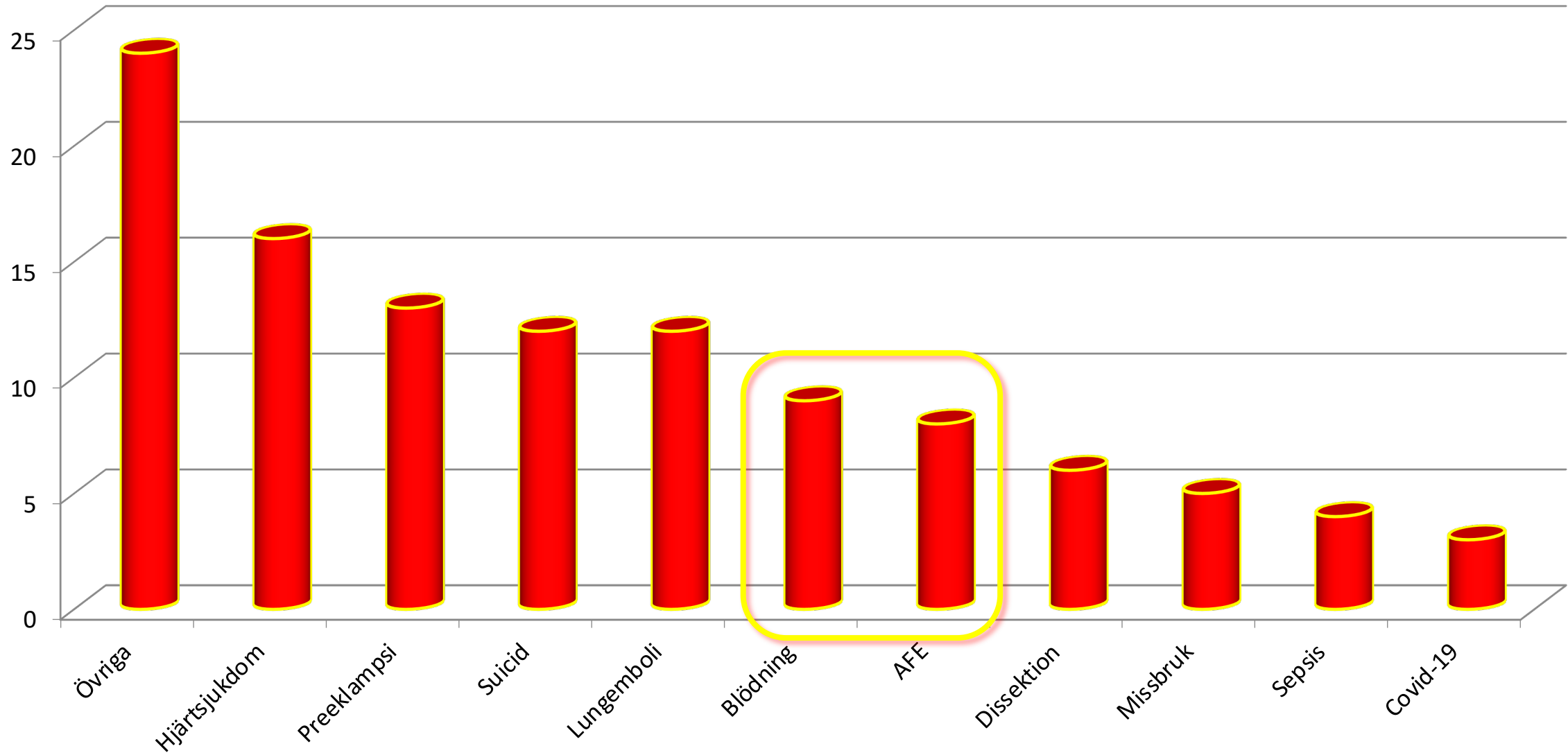
Vecka 39+3

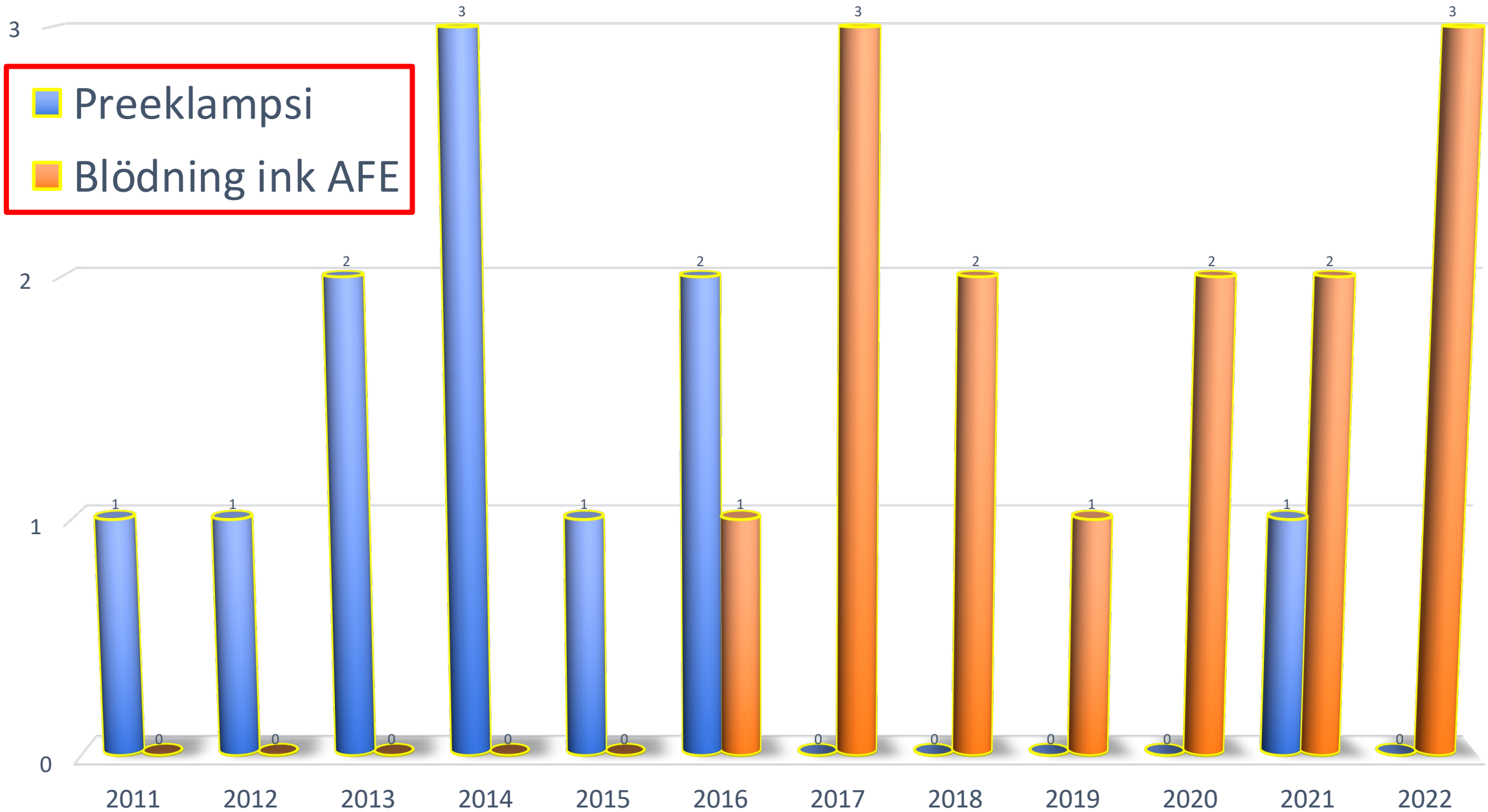
- UCG (ej hjärtprob)
 - Ev vätske spatium perikard
 - Upprepade gånger
 - Total akinesi
- Perikardtappning x 2
 - Indikation: ev behandlingsbar åtgärd
 - 30-40 ml + 20 ml
- HLR lugn och systematisk
- Avbryter HLR efter 40 min
- Postop summerad blödning 4120 ml

Obduktion

- Lungkärl: skivepitel färgat pos för CK 5/6 AE1/3, inga tromber
- Fostervattenembolisering

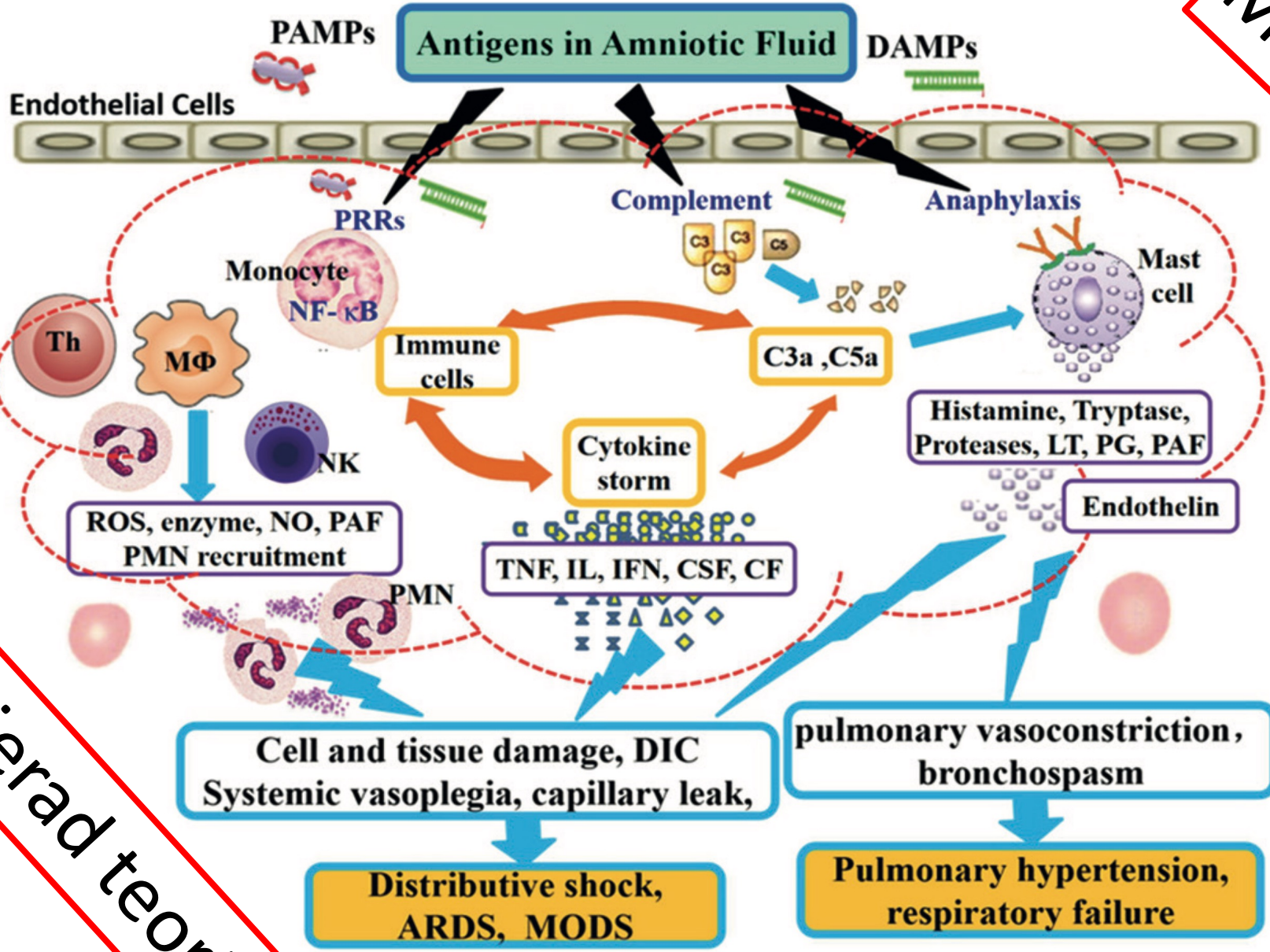
Mödradödlighet Sverige 2007-2022





Lönar det sig att arbeta med det?

Mekanisk teori



Immun medierad teori

Amniotic fluid embolism: Pathophysiology from the perspective of pathology

Naoaki Tamura , Mustari Farhana , Tomoaki Oda, Hiroaki Itoh and Naohiro Kanayama

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- 21-100 % fetala celler i moders cirkulation utan AFE
- AFE
 - Fetala celler in i moders cirkulation
 - Signifikant lungemboli 10-15 %, alt
 - Immunologisk/anafylaktoid reaktion
- Olika färgningar för att hitta fetala celler
- Patologiska förändringar i lungor och uterus
 - Lungor: pulmonell vasospasm, ödem och aktivering av trombocyter, vita blodkroppar och komplement
 - Uterus: komplement aktivering och inflammation vilket leder till DIC, atoni och blödning

type of syndrome characterized by the abrupt onset of hypoxia, pulmonary coagulopathy (DIC), occurring during labor, delivery, or the presence of amniotic components into the maternal circulation. AFE is a rare but fatal obstetrical complications, resulting in a high mortality rate among patients requiring intensive critical management, we often encounter patients with the presentation of AFE-related conditions. A major concern is that there is a lack of consensus on the pathophysiology of AFE, because its pathophysiology is still not well understood. This article reviews the pathophysiology of AFE and currently proposed pathophysiology.

It is characterized by an acute allergic-like reaction, disseminated intravascular coagulopathy, postpartum hemorrhage, and uterine atony.

Klinik

- Hypotension 100
- Fetal distress 100
- Lungödem 93
- Cirkulationsstopp 87
- Cyanos 83
- Koagulopati 83
- Dyspné 49
- Kramp 48
- Atoni 23
- Bronkospasm, transient hypertension
hosta, huvudvärk mm



BJA Education, 18(8): 234–238 (2018)

doi: 10.1016/j.bjae.2018.05.002

Advance Access Publication Date: 28 June 2018

Amniotic fluid embolism

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Learning objectives

By reading this article, you should be able to:

- Identify the risk factors for amniotic fluid embolism (AFE).
- Discuss the pathophysiological processes underlying the clinical features of AFE.
- Differentiate between more common causes of maternal collapse and AFE.
- Interpret different diagnostic tests in the context of AFE, including new markers and specialised investigations.
- Implement practical management strategies for a patient suspected to have AFE.

Key points



- Amniotic fluid embolism is still a leading cause of maternal mortality.
- There is a temporal relationship between the two proposed pathophysiological theories: mechanical and immunological.
- The clinical picture is non-specific, but hypotension and fetal distress have been identified in all cases.
- Many laboratory tests are helpful in guiding diagnosis.



Risikfaktorer

- Ålder > 35 år
- Manligt foster
- Multipla graviditeter
- Etnisk minoritet
- Polyhydramnios
- Placenta previa
- Eklampsi

- Induktion
- Hyperstimulering
- Instrumentell förlossning
- Kejsarsnitt
- Uterusruptur
- Cervixtrauma
- Ablatio



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- Discuss the pathophysiological processes underlying the clinical features of AFE.
- Differentiate between more common causes of maternal collapse and AFE.
- Interpret different diagnostic tests in the context of AFE, including new markers and specialised investigations.
- Implement prompt management of a woman suspected to have AFE.

Key points

- Amniotic fluid embolism is still a leading cause of maternal mortality.
- There is a temporal relationship between the two proposed pathophysiological theories: mechanical and immunological.
- The clinical picture is non-specific, but hypertension and fetal distress have been identified in all cases.
- Many laboratory tests are helpful in guiding management, but none is proved to aid diagnosis at the time.
- Management should not be delayed where the diagnosis is difficult or by exclusion.

Diagnos

- Klinisk diagnos
- Uteslutningsdiagnos

UKOSS

You are here: NPEU Home / UKOSS

UK Obstetric Surveillance System (UKOSS)

UKOSS: A national system to study rare disorders of pregnancy

1. Maternell kollaps + en/flera:

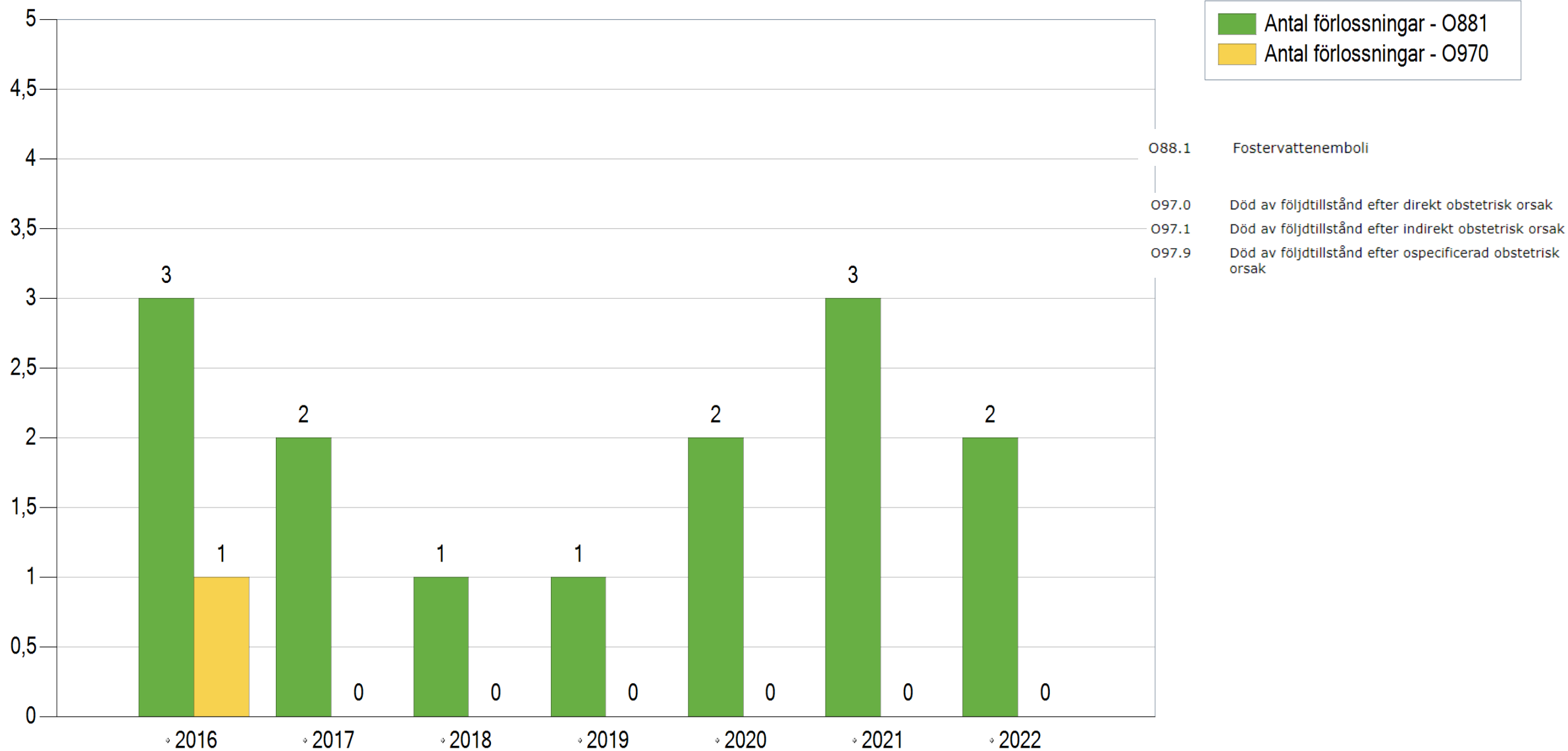
- Fetal distress
- Hjärtstopp
- Arytmi
- Koagulopati
- Hypotension
- Blödning
- Kramp
- Dyspné
- Prodromal symtom (upp till 4 t)

2. Postmortem diagnostik

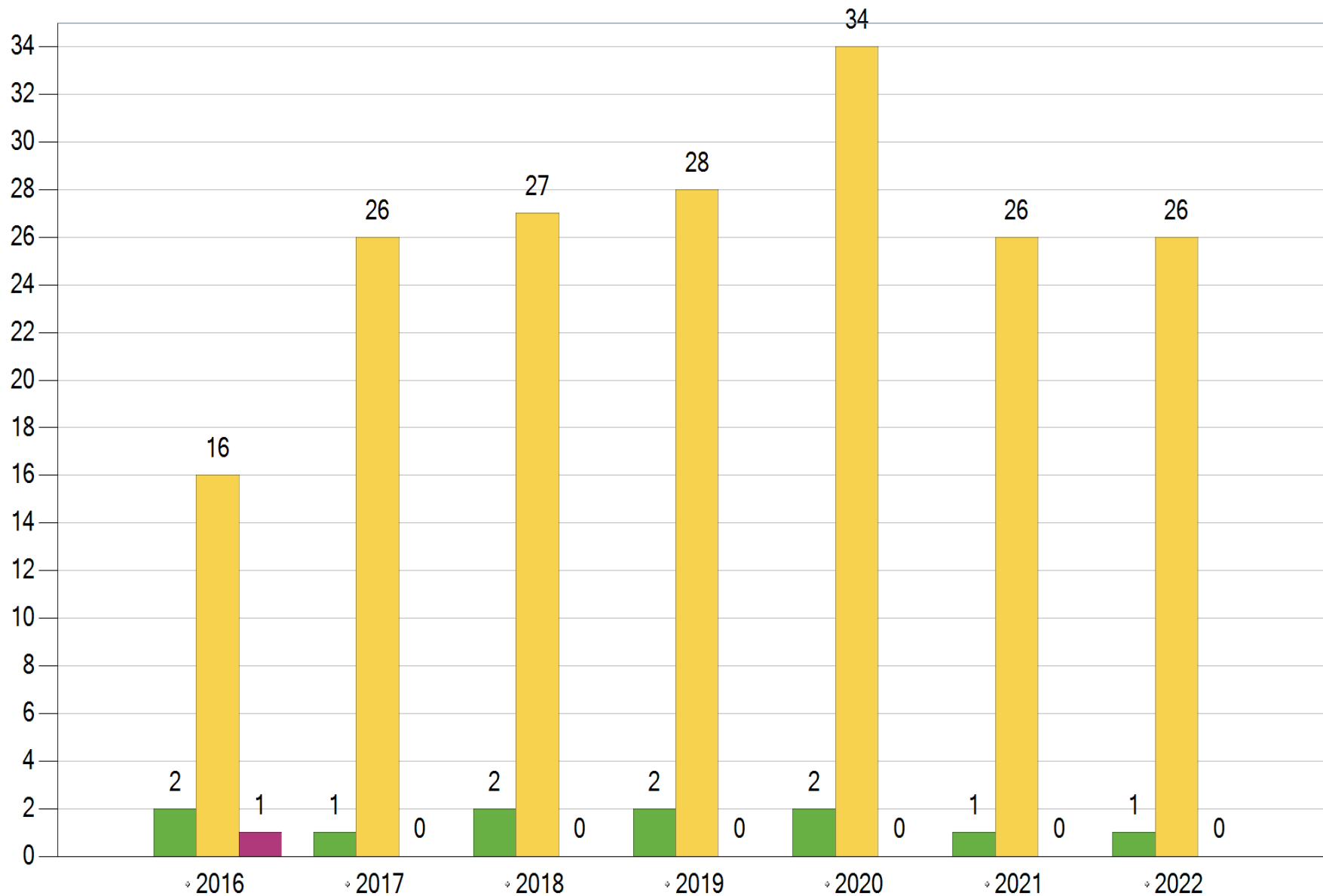
Differential diagnoser

- Lungemboli
- Peripartum kardiomyopati
- Sepsis
- Hjärtinfarkt
- Luftemboli
- Eklampsi
- Anafylaxi
- Total spinal

Antal kvinnor med fostervattenemboli samt dödsfall i Sverige 2016-2022



Antal kvinnor med lungemboli i Sverige 2016-2022



- Antal förlossningar - I260
- Antal förlossningar - I269
- Antal förlossningar - O970

I26.9 [Lungemboli utan uppgift om akut cor pulmonale Internetmedicin \(3\) • 1177](#)

I26.0 [Lungemboli med uppgift om akut cor pulmonale Internetmedicin \(2\) • 1177](#)

O97.0 Död av följdtilstånd efter direkt obstetrisk orsak

O97.1 Död av följdtilstånd efter indirekt obstetrisk orsak

O97.9 Död av följdtilstånd efter ospecificerad obstetrisk orsak

DOI: 10.1111/1471-0528.16625

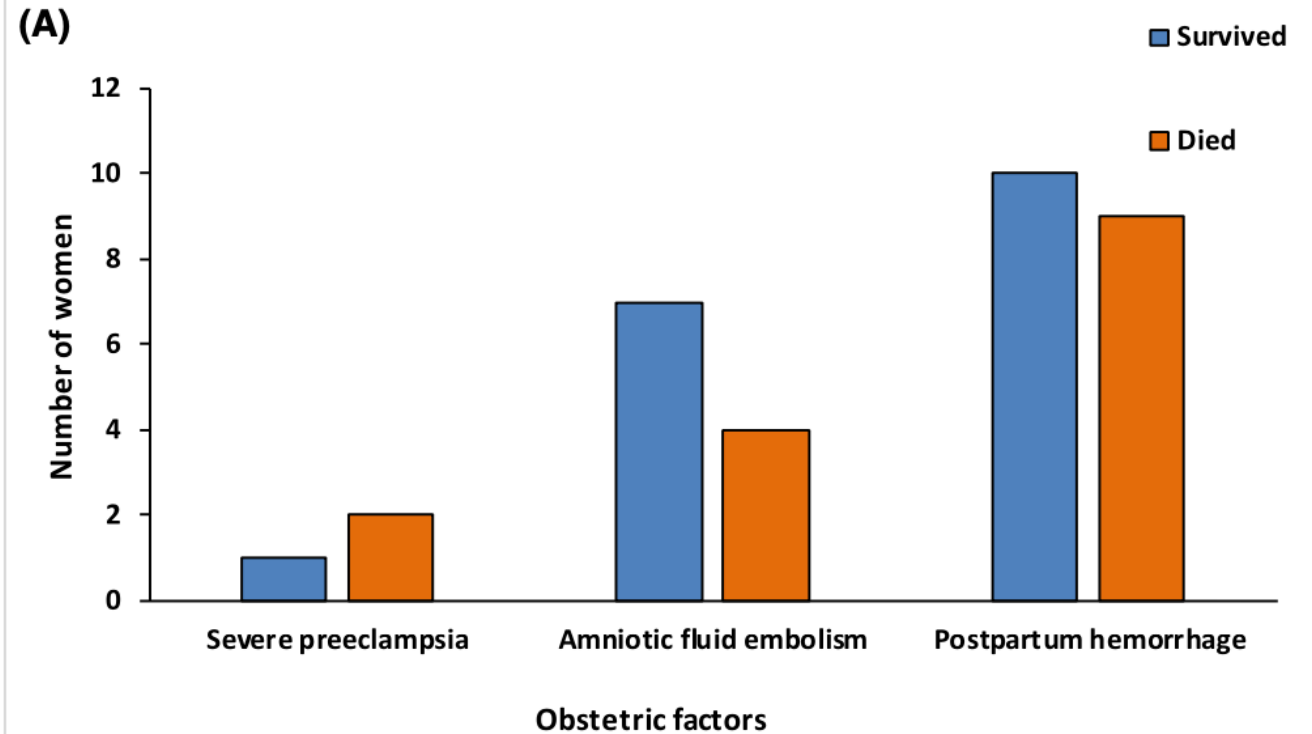
www.bjog.org

Maternal cardiac arrest

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Vad göra?

ABCDE ink
Perimortem snitt

Intensivvård

- Respiration
- Hjärta/cirkulation
- Hemostas

Patientnära instrument

- Ultraljud
- TEG/ROTEM
- Blodgas



Echocardiography findings in amniotic fluid embolism: a systematic review of the literature

Observations échocardiographiques lors d'une embolie de liquide amniotique : une revue systématique de la littérature

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Received: 17 May 2022 / Revised: 1 August 2022 / Accepted: 1 August 2022
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Abstract

Purpose Amniotic fluid embolism (AFE) is a leading cause of obstetrical cardiac arrest and maternal morbidity.

Clinical Opinion

ajog.org

Amniotic fluid embolism: principles of early clinical management



Luis D. Pacheco, MD; Steven L. Clark, MD; Miranda Klassen, BS; Gary D. V. Hankins, MD

Amniotic fluid embolism is an uncommon, but potentially lethal, complication of pregnancy. Because amniotic fluid embolism usually is seen with cardiac arrest, the initial immediate response should be to provide high-quality cardiopulmonary resuscitation. We describe key features of initial treatment of patients with amniotic fluid embolism. Where available, we recommend performing transthoracic or transesophageal echocardiography as soon as possible because this is an easy and reliable method of identifying a failing right ventricle. If such failure is identified, treatment that is tailored at improving right ventricular performance should be initiated with the use of inotropic agents and pulmonary vasodilators. Blood pressure support with vasopressors is preferred over fluid infusion in the setting of severe right ventricular compromise. Amniotic fluid embolism–related coagulopathy should be managed with hemostatic resuscitation with the use of a 1:1:1 ratio of packed red cells, fresh frozen plasma, and platelets (with cryoprecipitate as needed to maintain a serum fibrinogen of >150–200 mg/dL). In cases that require prolonged cardiopulmonary resuscitation or, after arrest, severe ventricular dysfunction refractory to medical management, consideration for venoarterial extracorporeal membrane oxygenation should be given.

Key words: blood product, cardiac arrest, cryoprecipitate, coagulation, dobutamine, norepinephrine, platelet, right ventricular failure

described in cases of AFE may provide the best chance at improved maternal and fetal outcomes.⁴ Although no data exist to document improved survival of such women with any specific treatment regimen, we describe here 1 organized, logical approach to the initial acute management of AFE that is recommended by the authors who have extensive experience in critical care obstetrics.

Cardiac arrest

Because AFE often presents with cardiac arrest, the initial immediate response should be to provide high-quality cardiopulmonary resuscitation (CPR). Without delay, chest compressions should be started, with the heel of the hand placed in the lower half of the sternum and a compression depth of

- Pulmonell hypertension
- Hö kammar svikt
- Biventrikulär svikt
- HK-svikt risk hjärtstopp

Diagnostik peripartal maternell kollaps

Ultraljud hjärta

- ***Högerkammarsvikt?***
- Ja → Fostervattenemboli
alt lungemboli
- Nej → Diff diagnoser

TEG/Rotem

- ***Koagulationspåverkan?***
- Ja → Fostervattenemboli
- Nej → Lungemboli

FIGURE 2
Immediate acute management of amniotic fluid embolism

Cardiorespiratory collapse from suspected amniotic fluid embolism

- Start immediate high-quality CPR.
- Chest compressions 100–120/min, and avoid hyperventilation.
- Defibrillate as indicated.
- Prepare for operative vaginal delivery (if indicated) and early perimortem cesarean delivery if ≥ 23 weeks.

- Early TTE, usually after return of spontaneous circulation.
- TTE may be used during CPR administration using the subxiphoid view or during the short pauses (< 10 seconds) used to check for a pulse (importantly chest compressions should NEVER be interrupted to obtain a TTE).

- If evidence of acute cor pulmonale, start treatment of right ventricular failure with vasopressors, inotropes, and pulmonary vasodilators.
- Avoid fluid boluses.

- In cases of coagulopathy and significant bleeding, early activation of massive transfusion protocols with the use of hemostatic resuscitation is fundamental.
- Use uterotonics and clinical criteria as indicated for the need of operative intervention to control bleeding (eg, uterine balloon tamponade or packing, repair of genital lacerations, or even laparotomy for hemostatic sutures or hysterectomy).

Persistent hemodynamic instability despite medical management or need for prolonged CPR may require consideration for VA ECMO.

Amniotic fluid embolism: principles of early



TABLE
Pharmacologic agents used to treat acute right ventricular failure

Pharmacologic agent	Dosage
Norepinephrine (vasopressor)	0.05–3.3 $\mu\text{g}/\text{kg}/\text{min}$
Dobutamine (inotrope)	2.5–5 $\mu\text{g}/\text{kg}/\text{min}$ (usually avoid doses $> 5 \mu\text{g}/\text{kg}/\text{min}$ because tachycardia at higher doses may limit right ventricular filling and consequently cardiac output)
Milrinone (inotrope)	0.25–0.75 $\mu\text{g}/\text{kg}/\text{min}$
Sildenafil (pulmonary vasodilator)	20 mg orally every 8 hrs
Inhaled nitric oxide (pulmonary vasodilator)	5–40 parts per million
Inhaled prostacyclin (epoprostenol, pulmonary vasodilator)	10–50 $\text{ng}/\text{kg}/\text{min}$
Intravenous prostacyclin (epoprostenol, pulmonary vasodilator)	Start at 1–2 $\text{ng}/\text{kg}/\text{min}$, titrate to desired effect

Pacheco. Immediate management of amniotic fluid embolism. Am J Obstet Gynecol 2020.

30-årig kvinna

- Frisk, BMI 29
- II-gravida, I-para
- 2019 grad IV bristning
- Graviditetsvecka 38+3
- Plan: elektivt kejsarsnitt

Elektivt kejsarsnitt

- Spinal ua
- Fenylefrin pga blodtrycksfall
- Barnet utskaffas med lätthet, placenta framvägg och exprimeras lätt.
- 5-10 minuter efter avnavling, plötsligt händer det något:
 - Pat känner sig konstig och illamående
 - Blodtryck bra
 - Okontaktbar och får sträckkramp

30-årig kvinna

Okontaktbar och kramp, vad göra?

- Ringer efter hjälp
- Söver och intuberar ua
- Lättventilerad.
- Bradykardi
- Inget koldioxid utbyte
- Huuuuu, asystoli...

1. HLR och adrenalin
2. ROSC inom 2 minuter
3. Artärnål, blodgas
4. UCG
5. ROTEM

30-årig kvinna

Maternell k

• Diagnos?

- Hjärtstopp
- Lungemboli
- Cirkulations kollaps
- Blödningschock
- Allergisk chock
- Fostervattenemboli

Vad göra?

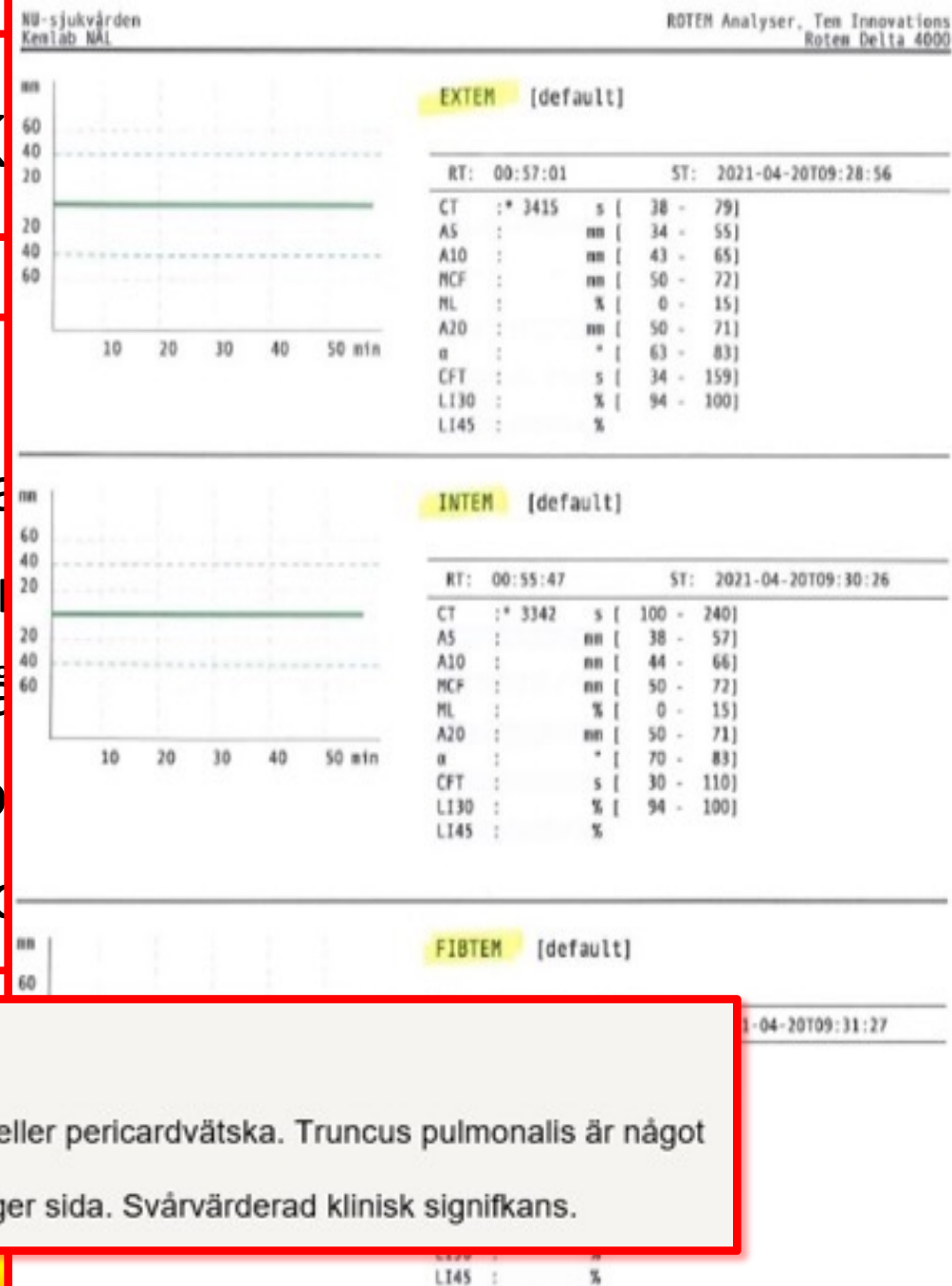
- Obstetriker avsluta
- Viss atoni, oxytocin
- Viss blödning, 2 på
- Överväger trombo
- CT hjärna, thorax o

2021-04-20 DT lungartär, angio med kontrast

Det finns lungemboli i högersidig ovanlobartär.

Dekliva atelektaser bilateralt. Inga övertygande ground glass för tätningar. Ingen pleuravätska eller pericardvätska. Truncus pulmonalis är något vid, mäter 34 mm i tvärmått. Ingen dilatation av höger kammare.

Det finns en gasbubbla i truncus pulmonalis. Gas ses också i v subclavia och v mammae höger sida. Svårvärderad klinisk signifikans.

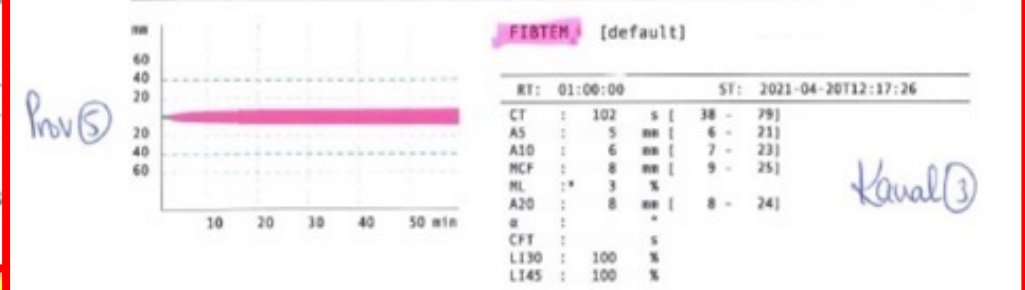
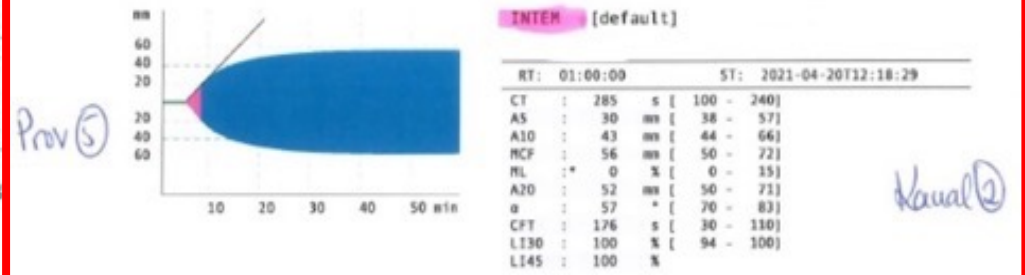
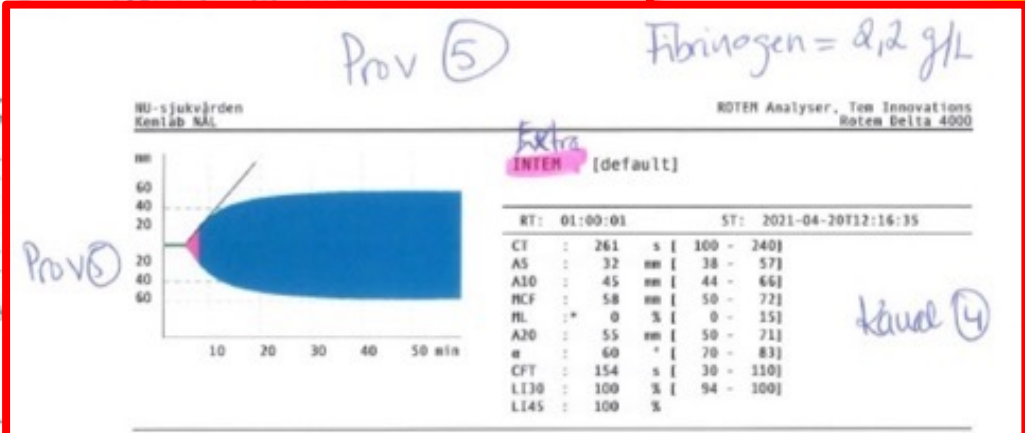
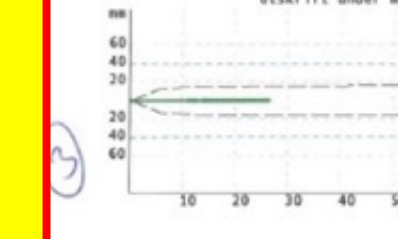
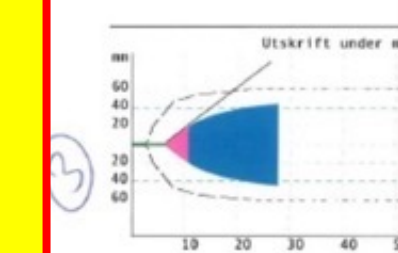
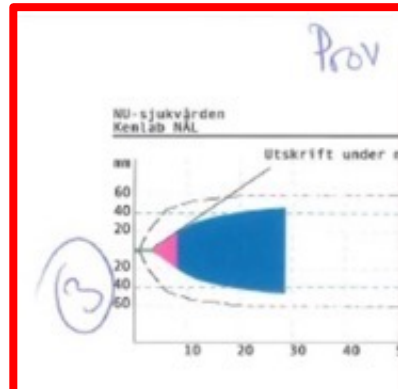
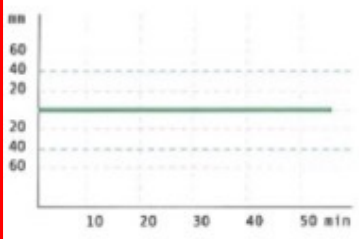
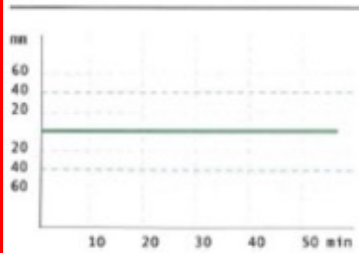
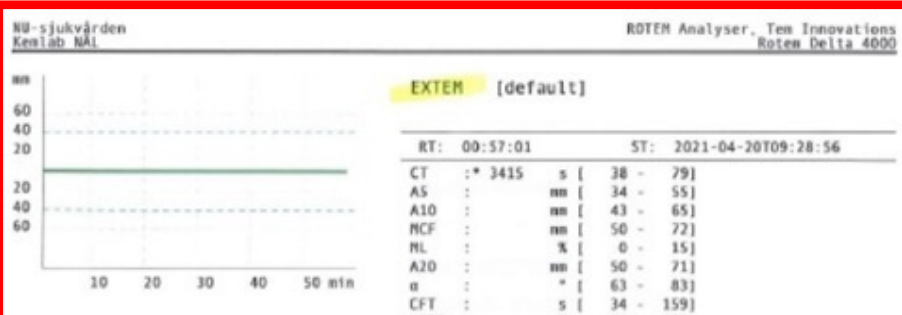


30-årig kvinna

Maternell kollaps, Rotem

Vad gjorde de?

- Fibrinogen 6 g
- Efter mer behandling:
- Tranexamsyra 1 g
- Fibrinogen 12 g
- Erytrorycer 7 st
- FFP 7 st
- Trombocyter 2 st



30-årig kvinna

Maternell kollaps, IVA

- Lättventilerad
- Noradrenalin 0,4 mg/kg/min
- Fortsatt blödning
- Försök uterusballong
- Transfusion
 - Erythrocyter
 - FFP
 - Trombocyter

- Dag 1
 - Extuberad och till avd
- Dag 2
 - UCG ua
- Dag 5
 - Hem

Diagnos: Fostervattenemboli

- Maternell kollaps
- DIC

- ABCDE
- Perimortem snitt

Fostervattenemboli

- Ultraljud
- TEG/ROTEM

- Milrinon
- Noradrenalin

- Tranexamsyra
- 4:4:1
- Fibrinogen

- Prostac.
- NO
- ECMO

