

Amniotic fluid embolism General view, multicenter survey and practical approach

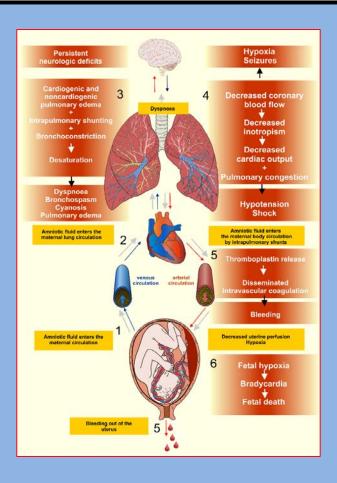
Alexander Ioscovich MD

Director of Gynecological and High Risk Obstetric Anesthesia Unit, Shaare Zedek Medical Center, Hebrew University, Jerusalem Chairman, Israel Association of Obstetric Anesthesia





Incidence of Amniotic Fluid Embolism: 2 - 8 cases / 100 000 births



Did you have patients with AFE?

Table 2 Signs and symptoms of amniotic fluid embolism

	Frequenc
Hypotension	100%
Fetal distress	100%
Pulmonary oedema or ARDS	93%
Cardiopulmonary arrest	87%
Cyanosis	83%
Coagulopathy	83%
Dyspnoea	49%
Seizure	48%
Uterine atony	23%
Bronchospasm	15%
Transient hypertension	11%
Cough	7%
Headache	7%
Chest pain	2%

Adapted from Clark SL. 20; ARDS: adult respiratory

syndrome.

Signs and symptoms of AFE

TABLE 3

Differential diagnoses by clinical symptoms

Clinical manifesta- tion/symptoms	Amniotic fluid embolism	Pulmonary embolism	Myocardial infarction	Peripartal cardiomyopathy
Manifestation	During labor/birth → hours postpartum	2 to 15 times more common during labor than pregnancy	21% peripartally 34% postpartally	Third trimester: approx. 9% to 80% up to 4 months postpartum
Risk factors	+/nonspecific	+++/specific	+++/specific	+/nonspecific
Cardiac arrest	++	+ ++	+	+
Chest pain	-	++ → +++	+++	++
Cardiac arrhythmia	+ ++	++ → +++	+++	++
Dyspnea	+++	+	+ ++	++
Hypotension	+++	+ ++	+ ++	+/-
Neurological symptoms	++	+ secondary	(+) secondary	(+) secondary
Coagulopathy	++	-	-	-
Acute fetal distress	+ ++	(+) secondary	(+) secondary	No data

^{-:} None or rare; +: Occasional; ++: Common; +++: Very common; Table from Rath W.: Fruchtwasserembolie, Lungenembolie (Amniotic Fluid Embolism, Pulmonary Embolism). In: Feige A., Rath W., Schmidt S (eds.): Kreißsaal-Kompendium, Stuttgart, New York, Thieme 2013; 142–9 (e17). Reproduced with the kind permission of Thieme Publishers

Amniotic Fluid Embolism (AFE)

Diagnostic criteria and Differential diagnosis

TABLE 2

Amniotic fluid embolism diagnosis criteria

UK Obstetric Surveillance System (UKOSS) 2010 (3)

No other clear cause: acute cardiovascular collapse with one or more of the following signs:

- Acute fetal compromise
- Cardiac arrest
- Cardiac arrhythmia
- Coagulopathy
- Hypotension
- Maternal hemorrhage*
- Premonitory symptoms, (e.g. restlessness, anxiety, agitation)
- Seizures
- (Sudden onset) shortness of breath

Benson M. et al. 2007 (18)

Pregnant women up to 48 hours after birth with one or more of the following symptoms and requiring treatment:

- Hypotension (and/or cardiac arrest)
- Respiratory distress
- Disseminated intravascular coagulation
- Coma and/or seizures
- No other medical explanation for clinical course

Modified according to (3) and (18)

Elective CS

1 min after delivery:

► Hypotension (80/40 mmHg)

≻Bigemini

≻Agitation

Table 2. Differential Diagnosis of Amniotic Fluid Embolism

Obstetric causes

Acute hemorrhage

Placental abruption

Uterine rupture

Uterine atony

Eclampsia

Peripartum cardiomyopathy

Anesthetic causes

High spinal anesthesia

Aspiration

Local anesthetic toxicity

Nonobstetric causes

Pulmonary embolism

Air embolism

Anaphylaxis

Sepsis/septic shock

^{*} Excluding women with maternal hemmorrhage as the first symptom with no evidence of early coagulopathy or cardiorespiratory compromise or in cases of postnatal evidence of fetal squames or hairs in the lung

TABLE 1

Incidence of amniotic fluid embolism

Country	Period	Incidence (n/100 000 births)	Case-related mortality	Perinatal mortality
Australia*1	2001 to 2007	3.3	35%	32%
USA*1	1999 to 2003	7.7	21.6%	No data
Canada*1	1991 to 2002	6.0	13%	No data
U.K. *2	2005 to 2009	2.0	20%	13.5%
The Nether- lands*2	2004 to 2006	2.5	11%	38.1%

^{*1} Retrospective population-based studies

Modified according to (12), Knight M. et al.: BMC Pregnancy & Childbirth 2012; 12: 7

The condition is rare – around one in 50 000 births in the UK.

In the UK, AFE is researched through the UK Obstetric Surveillance System (UKOSS)

Knight M, Tuffnell D, Brocklehurst P, Spark P, Kurinczuk JJ, on behalf of the UK Obstetric Surveillance System. Incidence and risk factors for amniotic fluid embolism. *Obstetrics and Gynecology* 2010; 115: 910–7.

Table 1 Incidence of amniotic fluid embolism and case fatality rates in published series

	Year published	Incidence (per 100 000 maternities)	Case fatality rate (%)
Knight ⁷	2012	1.9-6.1	11–43
Kramer ²¹	2012	2.5	27
Knight ³	2010	2.0	20
Oi ⁴⁴	2010	Not reported	48
Roberts ⁷²	2010	3.3	35
Abenhaim ⁸	2008	7.7	21.6
Samuelsson ⁷³	2007	1.9	44
Kramer ⁷⁴	2006	6.1	13
Tuffnell ³⁶	2005	Not reported	29.5
Yang ⁷⁵	2000	Not reported	89
Gilbert ⁷⁶	1999	4.8	26.4
Clark ²⁰	1996	Not reported	61
Burrows ⁷⁷	1995	3.4	22
Hogberg ⁷⁸	1985	Not reported	67
Morgan ⁶⁴	1979	Not reported	86

Incidence of Amniotic Fluid Embolism (AFE) is estimated to occur between 1 in 8000 and 1 in 80,000 deliveries.

The true incidence is unknown because of inaccurate diagnosis and inconsistent reporting of nonfatal cases.

^{*2} Case-related validation from prospective studies

Squamous and/or trophoblastic cells in pulmonary blood vessels as a indicator of AFE

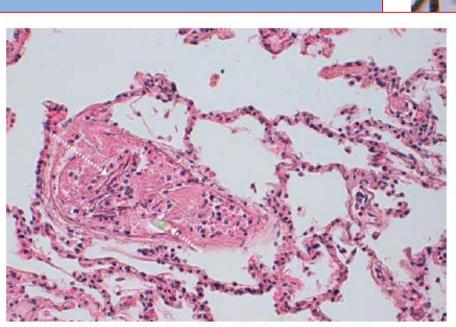
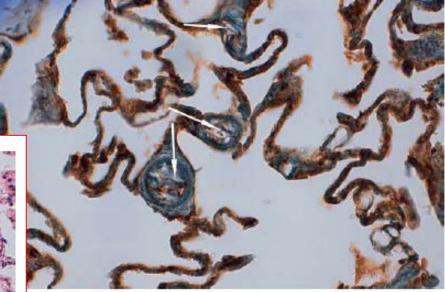
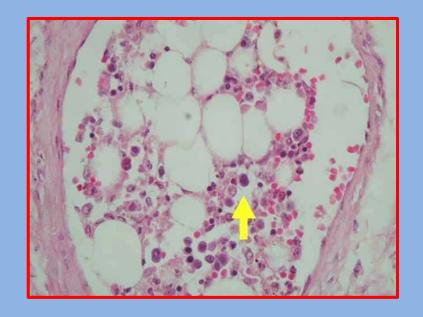


Figure 2: A blood vessel enclosed by lamellar epithelial squames (long dotted arrow) embedded in a fibrin thrombus (two transparent arrows). The lower part of the picture shows a transparent, cylindrical structure corresponding to a lanugo hair (short dotted arrow). Hematoxylin eosin staining: 200x. Survival time: 8 hours



: Immunohistochemically marked epithelial squames in pulmonary arterioles Cytokeratin, 200x





Pulmonary artery blood specimens from 14 term pregnant women with severe pregnancy-induced hypertension suggest that squamous or trophoblastic cells may be normally present in the maternal pulmonary circulation during the peripartum period and that their presence is not pathognomonic of clinically significant amniotic fluid embolism. Squamous and trophoblastic cells in the maternal pulmonary circulation identified by invasive

hemodynamic monitoring during the peripartum period Wesley Lee AJOG Volume 155, Issue 5, Pages 999–1001

Clark, SL, Pavlova, Z, Greenspoon, J, Horenstein, J, Phelan, JP. **Squamous cells in the maternal pulmonary circulation.** *Am J Obstet Gynecol.* 1986;154:104.

Covone, AE, Johnson, PM, Mutton, E et al, Trophoblastic cells in peripheral blood from pregnant women. Lancet. 1984;2:841.

"The pathogenesis of AFE is not yet fully clear."

- Mechanical obstruction of the pulmonary vessels by amniotic fluids component
- Anaphylactoid reaction hypothesis

AFE is "Anaphylactoid syndrome of pregnancy"





Lee at al, and other investigators could not reproduce the AFE-syndrome by injecting AF directly into mini-pig and monkey model circulation.

Clark SL, Hankins GD, Dudley DA, Dildy GA, Porter TF: Amniotic fluid embolism: analysis of the national registry. AJOG 1995; 172: 1158–67

Coagulopathy

"The cause of DIC in AFE is as yet unclear..."

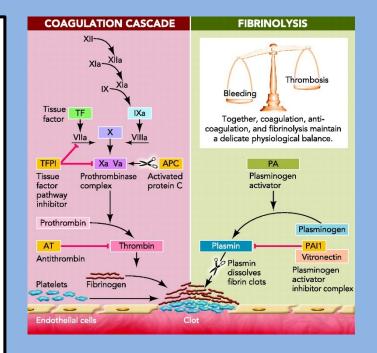
•1-st - Procoagulant theory

AF contains many procoagulant substances (tissue factor and phosphatidylcerine) which can lead directly or indirectly (via cytokines or complement activation) to DIC with consumptive coagulopathy.

•2 –nd - Fibrinolytic theory

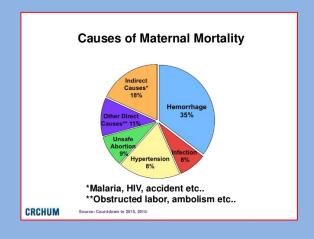
DIS as a result of massive fibrinolysis, as AF also contains increased concentration of urokinaselike plasminogen activators.

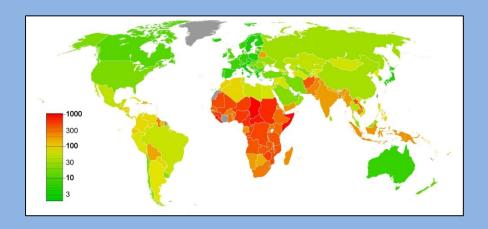
Prosper SC, Goudge CS, Lupo VR. Recombinant factor VIIa to successfully manage disseminated intravascular coagulation from amniotic fluid embolism. Obstet Gynecol 2007;109:524–5



Unfortunately, not a great deal has changed in our understanding of the pathophysiology in the last 10 years.







According to statistics, it is the **AFE**

- most common cause of maternal death in Australia and Germany (in 2011 8 out of 12 cases)
- > second-most common in the USA and the U.K.

The identified risk factors for AFE are:

- •Maternal age 35 and above (odds ratio [OR] 1.86)
- Cesarean section (OR 12.4)
- •Placenta previa (OR 10.5)
- •Multiple pregnancy (OR 8.5) (In Vitro Fertilisation)

PubMed search 2000-2003

Amniotic Fluid Embolism: an Interdisciplinary Challenge Epidemiology, Diagnosis and Treatment Werner H.

Dtsch Arztebl Int 2014; 111(8): 126-32

International Journal of Obstetric Anesthesia (2013) 22, 329–336 0959-289X/S - see front matter Crown Copyright © 2013 Published by Elsevier Ltd. All rights reserved. http://dx.doi.org/10.1016/j.jioa.2013.08.004

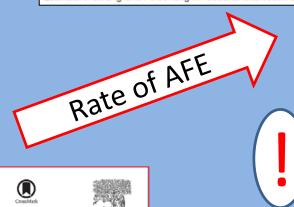
REVIEW ARTICLE

Amniotic fluid embolism: a leading cause of maternal death yet still a medical conundrum

Clark SL, Belfort MA, Dildy GA: Maternal death in the 21st century prevention and relationship to caesarean delivery, AJOG 2008; 199; 36.

The Confidential Enquiry into Maternal and Child Health (CEMACH): Saving Mother's lives reviewing maternal deaths to make childhood safer 2003–2006. London: CEMACH 2007.

AG "Mütterliche Todesfälle" 2011: AQUA-Institut für angewandte Qualitätsförderung und Forschung im Gesundheitswesen 2012.





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International Journal of Obstetric Anesthesia (2013) 22, 329–33 0959-289X/S - see front matter Crown Copyright © 2013 Published by Elsevier Ltd. All rights reserved. http://dx.doi.org/10.1016/j.ijoa.2013.08.004





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TABLE 1 Incidence of amniotic fluid embolism Period Incidence Case-related Perinatal (n/100 000 mortality mortality births) Australia*1 2001 to 2007 3.3 35% 32% USA*1 1999 to 2003 7.7 21.6% No data Canada*1 1991 to 2002 6.0 13% No data U.K. *2 13.5% 2005 to 2009 2.0 20% 38.1% 2004 to 2006 2.5 11% The Netherlands*2

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Case related maternal mortality from AFE 37% in the UK registry 2005 and 20% in the UK in 2009

Tuffnell DJ. United Kingdom amniotic fluid embolism register. BJOG 2005;112:1625–9

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The true incidence is unknown because of inaccurate diagnosis and inconsistent reporting of nonfatal cases.

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I Survive AFE!!!

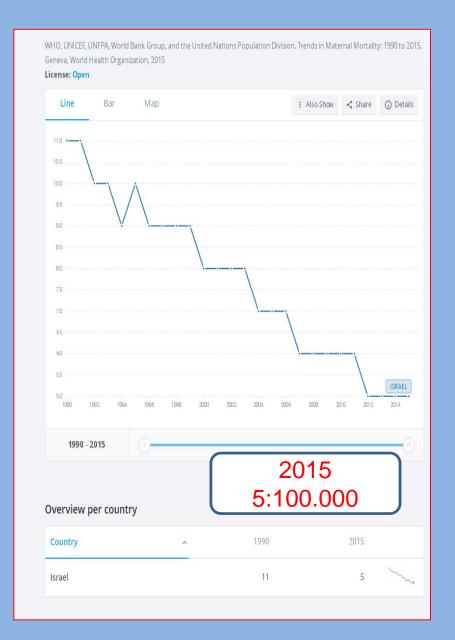
"Maternal morbidity is also high (up to 60%) and only 15% of survivors may be neurologically intact."

Clark SL Amniotic fluid embolism: analysis of the national registry. Am J Obstet Gynecol 1995;172:1158–67



"One of the most devastating long-term consequences of AFE is neurologic dysfunction. As many as 85% of survivors demonstrate residual neurologic deficits".

Moore J, Baldisseri MR. Amniotic fluid embolism. Crit Care Med. 2005;33(Suppl 10):S279–85.



In Israel

6-8 annually maternal death

2/3 as a result of AFE



Our data

- Retrospective chart review 2005-2015
- 5 Hospitals with total 50.000 labors annually
 - Hospitals with 6-16.000 labors annually
- •20 cases of AFE
- Maternal age -34.4 ± 4.9 (25-40)
 - •In Israel mean maternal age in 2014 was 30.5
 - •OECD Family Database http://www.oecd.org/els/family/database.htm
- Gravity 4.1 (1-9)
- Parity 2.4 (0-5)
- 38-41w (out of 2 cases 21 and 22w)
- No: HTN, DM, Hypo/Hypercoagulation
- Toxemia
- No allergies No placenta previa
- 2 (10%) cases after IVF All cases singleton, 1 post CS
- 2 (10%) macrosomia and 3 (15%) polyhydramnios

The main risk factors for AFE are as follows (3): maternal age 35 years or above (odds ratio [OR]: 1.86; 95% confidence interval [95% CI]: 0.99 to 3.48), Cesarean delivery (OR: 12.4; 95% CI: 6.5 to 23.6), placenta previa (OR: 10.5; 95% CI: 0.94 to 117.2), and multiple pregnancy (OR: 8.5; 95% CI: 2.92 to 24.6). Despite discrepancies between studies (4, 5, 22), a recent prospective study proposed that induction of labor increases the risk of AFE by 35% (3). The increasing

Dtsch Arztebl Int 2014; 111(8): 126-32

2 (10%) – no labor 13 (65%) – active labor 1 (5%) – postpartum 3 (15%) – emergency CS 1 (5%) – elective CS (CS – 4 cases 20%)

No cases with induction of labor

The main risk factors for AFE are as follows (3): maternal age 35 years or above (odds ratio [OR]: 1.86; 95% confidence interval [95% CI]: 0.99 to 3.48), Cesarean delivery (OR: 12.4; 95% CI: 6.5 to 23.6), placenta previa (OR: 10.5; 95% CI: 0.94 to 117.2), and multiple pregnancy (OR: 8.5; 95% CI: 2.92 to 24.6). Despite discrepancies between studies (4, 5, 22), a recent prospective study proposed that induction of labor increases the risk of AFE by 35% (3). The increasing

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Analgesia/Anesthesia before AFE

11 (55%) Epidural analgesia in labor

2 (10%) Conversion epidural for CS1 (5%) General anesthesia for Em CS1 (5%) Spinal for elective CS

Preceding event to AFE

3 (15%) – rupture of membrane 1 (5%) – tetania of uterus 3(15%) – "baby out"



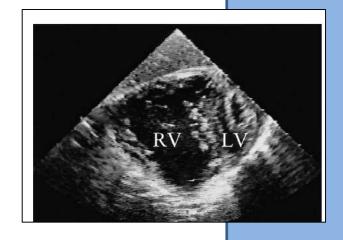
Resuscitation

<u>Intubation</u> – 18 (90%) 0-90 min <u>Chest compression -</u> 12 (60%) 0-12 min ; max 40 min <u>Defibrilation</u> – 7 (35%) 0-15 min; max 6 time

<u>CS + Perimortem CS</u> – 11 (55%) time "baby out" – 0 -20 min (10.3min) <u>Neonatal Outcome</u>?

 $\frac{\text{TEE}}{\text{TTE}} - 10 (50\%)$ $\frac{\text{TTE}}{\text{TTE}} - 2 (10\%)$ Signs of Acute R V failure – 11 (55%)

<u>Arterial line</u> - 14 (70%) <u>Central line</u> - 9 (45%) <u>"High flow" line</u> - 9 (45%)



ECMO – 4 (20%); time to connection- 90 – 300 min

Early maternal complication

Seizures – 5 (25%)

Uterine atonia – 17 (85%)

Coagulopathy – 19 (95%)

Reoperation (for hysterectomy) -5 (25%)

RF -1 (5%) **TRALI** -1 (5%) **MOF** -2 (10%)







<u>Die</u> – 3 (15%)

Severe neurologic injury – 3 (15%)
Minor neurologic injury – 5 (25%)

<u>ICU 0-30 dais</u> <u>Hospital – up to 80 dais</u>

9 (45%) – no residual complication

Neonatal Outcome



Perinatal mortality – 20-25%

With only 50% of surviving neonates being neurologically intact.

Table 3. Fetal/infant outcomes among confirmed AFE cases $(n = 120 \text{ for stillbirths}, n = 54 \text{ for neonatal outcomes}^*)$

Outcome	n (%)	Adjusted OR (95% CI)
Stillbirth	5 (4.2)	5.9 (2.0–17.4)
Asphyxia	15 (27.8)	36.0 (18.6–69.7)
Mechanical ventilation	17 (31.5)	11.8 (6.0-23.4)
Bacterial sepsis	5 (9.3)	5.0 (1.8-14.0)
Seizure	8 (14.8)	22.8 (9.7-53.3)
Jaundice (nonimmune haemolytic/traumatic)	9 (16.7)	1.4 (0.6–3.2)
Length of stay >7 days	18 (33.3)	18.5 (8.5-40.2)

^{*}Based on linked maternal and infant file available since 2001/02.

Gist RS, Stafford IP, Leibowitz AB, Beilin Y. Amniotic fluid embolism. Anesth Analg. 2009;108:1599–602.

Neonatal outcome in Peri mortem CS for viable baby (> 25 w)

Place of peri mortem CS	Time of Baby Out	Apgar 1 min	Apgar 5 min	Sex	Die
Delivery Suite	13	0	1	М	
Operation Room	14	2	9	F	
Operation Room	17	2	4	М	
Operation Room	10	5	9	М	
Operation Room	12	1	3	NI	
Operation Room	20	8	10	М	
Operation Room	10	1	3	М	
Delivery Suite	5	3	10	F	
Operation Room	4	6	10	NI	

Consider performing PMCD within 4 min of cardiac arrest if there is no ROSC in pregnant patients beyond 20 wk of gestation.



Our data

2 y a population –based descriptive study	10 y five hospitals survey
Estimated rate 5.4 cases /100.000	Estimated rate 4 cases /100.000
46% in DS and 46% in OR	55% in DS and 35% in OR
42% induction or augmentation of labor	No cases of induction or augmentation
67% underwent of CS	55% underwent of CS
24% after IVF	No cases after IVF
42% had full resuscitation	50% had full resuscitation
18% had hysterectomy	25% Hysterectomy
ECMO – no cases	20% ECMO
85% - Blood or blood products / Recombinant Factor VIIa -24%	85% - Blood or blood products/ Recombinant Factor VIIa – 5%
15% woman died	15% woman died
7% neonatal mortality	No final data about neonatal mortality

Monitoring and Resuscitation in case of AFE

AHA Scientific Statement

Cardiac Arrest in Pregnancy
A Scientific Statement From the American Heart Association

(Circulation. 2015;132:1747-1773.

Recommendations

- Chest compressions should be performed at a rate of at least 100 per minute at a depth of at least 2 in (5 cm), allowing full recoil before the next compression, with minimal interruptions, and at a compression-ventilation ratio of 30:2⁵¹ (Class IIa; Level of Evidence C).
- Interruptions should be minimized and limited to 10 seconds except for specific interventions such as insertion of an advanced airway or use of a defibrillator⁵¹ (Class IIa; Level of Evidence C).
- 3. The patient should be placed supine for chest compressions (*Class I; Level of Evidence C*).
- There is no literature examining the use of mechanical chest compressions in pregnancy, and this is not advised at this time.

- 1 Chest compression 100/min5 cm with minimal interruptionCompress/Ventilation ratio 30:2
- 2 interruption just for 10 sec for intubation or defibrillation
 No problem with defibrillation, take out the internal (scalp) monitor
- **3** Supine position !!!
- 4 Just manual chest compression

- Continuous manual LUD should be performed on all pregnant women who are in cardiac arrest in which the uterus is palpated at or above the umbilicus to relieve aortocaval compression during resuscitation (Class I; Level of Evidence C).
- If the uterus is difficult to assess (eg, in the morbidly obese), attempts should be made to perform manual LUD if technically feasible (Class IIb; Level of Evidence C).

Left Uterine Displacement



Figure 4. Manual left uterine displacement by the 2-handed technique from the left of the patient.



Figure 3. Manual left uterine displacement by the 1-handed technique from the right of the patient during adult resuscitation.

Perimortem cesarean delivery (PMCD)

Both resuscitation and obstetric guidelines suggest that:

Perimortem cesarean delivery be considered within 4 minutes of maternal collapse if there is no return of spontaneous circulation (ROSC) with the delivery of the fetus within 5 minutes in women beyond 20 weeks of gestation.

Optimal "Baby out time" < 5 min

<5 min from arrest to delivery – 70% perinatal survival 6-15 min from arrest to delivery – 12% perinatal survival

Vanden Hoek TL, Morrison LJ, Shuster M, Donnino M, Sinz E, Lavonas EJ, et al. Part 12: cardiac arrest in special situations: 2010 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care. Circulation 2010;122:S829–61.

Soar J, Perkins GD, Abbas G, Alfonzo A, Barelli A, Bierens JJ, et al. European Resuscitation Council Guidelines for Resuscitation 2010 Section 8. Cardiac arrest in special circumstances: Electrolyte abnormalities, poisoning, drowning, accidental hypothermia, hyperthermia, asthma, anaphylaxis, cardiac surgery, trauma, pregnancy, electrocution. Resuscitation 2010;81:1400–33.

PMCD Technique

The procedure should be performed at the site of the maternal resuscitation. Time should not be wasted moving the patient.

Table 2. Recommended Equipment for Perimortem Cesarean Section*

Equipment Contents of the Emergency Cesarean Delivery Tray

Scalpel with No. 10 blade

Lower end of a Balfour retractor

Pack of sponges

2 Kelly clamps

Needle driver

Russian forceps

Sutures and suture scissors

*The items listed in this table represent suggestions. The contents should be selected to meet the specific needs, preferences, and skills of the practitioner and healthcare facility.

Perimortem Cesarean Kit

- Knife handle with #10 blade
- Kelly clamos
- Mayo scissors
- Bandage scissors
- Tooth forceps
- Needle holders
- Sutures

- Laparotomy sponges
- Clear plastic abdominal drape
- IV pitocin
- Normal saline vials
- Syringes with needle

Echocardiography as a part of management of AFE

From 1999

...Transesophageal echocardiography revealed normal left ventricular contractility and gross enlargement of the right ventricle and main pulmonary trunk, consistent with acute right ventricular pressure overload and underloading of the left ventricle. Despite resuscitative efforts, the patient died three hours postoperatively

Amniotic fluid embolism: early findings of transesophageal echocardiography.

Anesth Analg. 1999 Dec;89(6):1456-8

Shechtman M¹, Ziser A, Markovits R, Rozenberg B

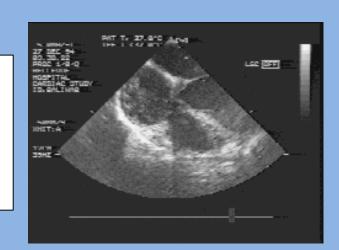
Department of Anesthesiology, Rambam Medical Center, Haifa, Israel.

Our expiriens

TEE – 10 (50%)

TTE - 2 (10%) → 12 case

Signs of Acute R V failure – 11



From 1999 to 2016

"Any dyspnea, chest pain, syncope or collapses must alert women health care providers to engage rapidly the diagnosis process and emergent treatment of the cardio-circulatory failure. The larger competence of the obstetric anesthetists to perform echocardiographic diagnosis at the parturient bedside helps to a more rapid management than previously."

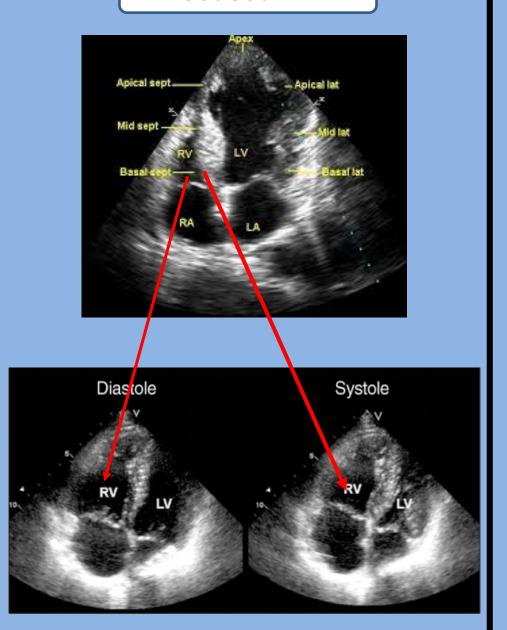
Anaesth Crit Care Pain Med. 2016 Jul 5.

Cardiovascular emergencies and cardiac arrest in a pregnant woman.

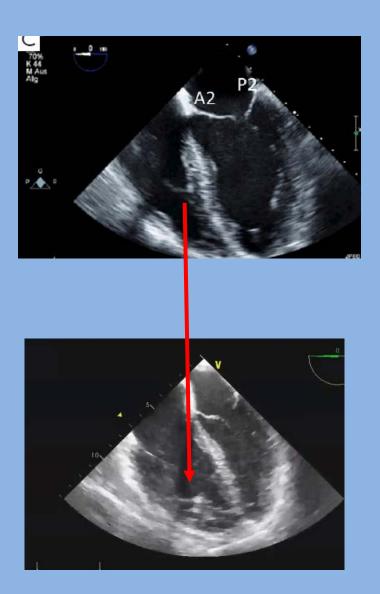
<u>Ducloy-Bouthors AS</u>

Transesophageal or Focused transthoracic echography

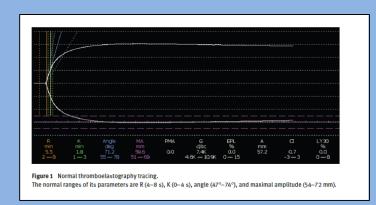
Focused TTE



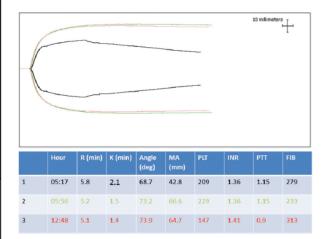
TEE



Thromboelastography for management of AFE







16u PLT 20u Cryo

8u FFP

2u FFP 5u PLT 10u Cryo

Figure 2 Thromboelastography (TEG) of the first case.

Trace 1 (black) was measured during administrating 8 units of plasma, 16 units of platelets, and 20 units of cryoprecipitate to the patient. It shows a decrease in the strength of the clot (low maximal amplitude). The second trace 2 (green) was recorded about 40 min later and it shows TEG parameters within normal limits. The third trace (red) was taken after the patients received 2 units of plasma, 5 units of platelets, and 10 units of cryoprecipitate. It is similar to the second trace.

DE GRUYTER Case Rep. Perinat. Med. 2014; aop

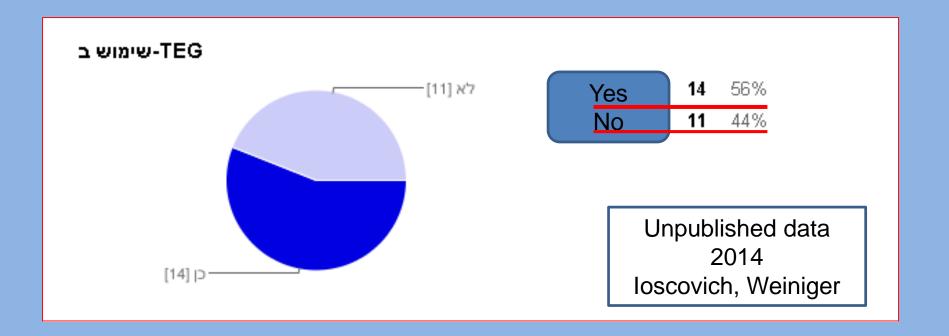
Yigal Helviza, Sharon Einava, Moshe Hersch, Henry Shapiro and Alexander Ioscovich*

Thromboelastography as a part of management of amniotic fluid embolism

Figure 3 Thromboelastography (TEG) of the second case.

The first trace (black) was taken before any blood product was administered to the patient. It shows that no coagulation was detected by the fact that it is a straight line and the international normalized ratio, partial thromboplastin time, and fibrinogen could not be measured. Trace number 2 (green) was taken about 3.5 h later, after administrating to the patient 2 units of plasma and 10 units of cryoprecipitate. Here there is a prolongation of R and K and decrease of the angle. These correspond to a delay in the initiation of the clot and of its propagation. Furthermore, the maximal amplitude severely decreased, aiming towards a weaker clot. The last trace (red) was taken after the patient received 10 units of cryoprecipitate. Although the R normalized, and there is improvement in the other parameters, there is still coagulooathy.

TEG for Massive bleeding in CS in Israel



From 0% in 2006 to 56% from all Obstetric Units in 2014

Cardio Pulmonary Bypass or Extra Corporal Membrane Oxygenation as a part of a management of AFE

ECMO and Intraaortic contrapulsation

Hsieh YY, Chang CC, Li PC, Tsai HD, Tsai CH. Successful application of extracorporeal membrane oxygenation and intraaortic counterpulsation as lifesaving therapy for a patient with amniotic fluid embolism. Am J Obstet Gynecol **2000**;183:496–7

Amniotic Fluid Embolism
Causing Catastrophic Pulmonary
Vasoconstriction: Diagnosis
by Transesophageal
Echocardiogram and Treatment
by Cardiopulmonary Bypass

Russell D. Stanten, MD, Leigh I. G. Iverson, MD, Terrance M. Daugharty, MD, Stuart M. Lovett, MD, Crystal Terry, MD, and Edward Blumenstock, MD 2003

Summit Medical Center, Oakland, California

Cardiopulmonary Bypass for treatment of AFE

53 min after AFE she was placed on CPB

After 83 min - disconnection from CPB

Cardio Pulmonary Bypass or Extra Corporal Membrane Oxygenation as a part of a management of AFE

. The majority of deaths now are in women who do not survive the acute phase: only three of the 12 women who died in the UK series reached the intensive care unit Incidence and risk factors for

Incidence and risk factors for amniotic fluid embolism. Obstetrics and Gynecology 2010; 115: 910–7.

Amniotic fluid embolism: incidence, risk factors, and impact on perinatal outcome

MS Kramer,^a J Rouleau,^b S Liu,^b S Bartholomew,^b KS Joseph^c for the Maternal Health Study Group of the Canadian Perinatal Surveillance System

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Canada 2012 120 cases No data about ECMO Germany 2015

Australia 2015

No data about ECMO

Accepted 25 February 2012. Published Online 24 April 2012.

Early Application of Extracorporeal Membrane Oxygenation in a Patient with Amniotic Fluid Embolism

Chang-Hsun Ho, Kuen-Bao Chen, Shih-Kai Liu, Yu-Fang Liu, Hung-Chun Cheng, Rick Sai-Chuen Wu*

Department of Anesthesia, Pain Management and Critical Care Medicine, China Medical University Hospital, Taichung, Taiwan, R.O.C.

Neurologic sequel



In our case series 4 patients were connected to ECMO:

2 patients die

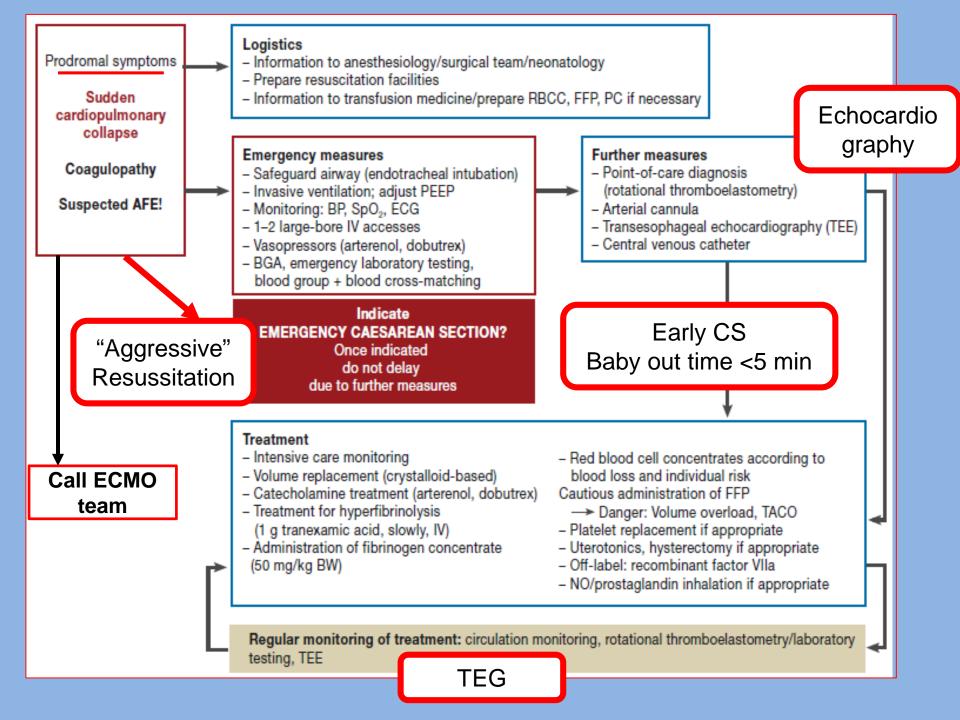
1 had major neurologic sequel

1 was intact, but she was connected to ECMO 5h after acute stage of AFE, secondary to heard failure

Inhaled nitric oxide has been used

in the treatment of right-sided heart failure and pulmonary hypertension.

McDonnell NJ, Chan BO, Frengley RW. Rapid reversal of critical haemodynamic compromise with nitric oxide in a patient with amniotic fluid embolism. Int J Obstet Anesth 2007;16:269–73













Welcome to Israel!









